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# TO STUDY ON BIOFILM FORMATION AMONG NASAL ISOLATES AND ITS ROLE IN ANTIBIOTIC RESISTANCE

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## ABSTRACT

Biofilm have demonstrated unfathomable effectiveness in environmental adaptation. By recycling different organic and inorganic elements of the earth, they have a significant impact on the environment. Currently, biofilms are used to purify sewage water, hazardous waste, and solid waste in addition to producing biofuels, enzymes, and medicinal pharmaceuticals. Aside from these advantages, biofilms have demonstrated their detrimental effects on people, plants, and animals. Patients who have persistent infections are affected by biofilms. Biofilms in plants produce a number of illnesses that can lead to significant economic and vegetative losses. A significant issue in many sectors is the corrosion of metals caused by biofouling. Additionally, treated stored water that has been contaminated by biofilms can become unusable. The combination of biofilms' benefits and drawbacks has prompted a lot of scientific research. Agricultural, bioengineering, biomimetic, drug development, waste management, bioremediation, clinical medicine, diagnostics, and other fields of basic and applied sciences have all been included in the scope of biofilm research. Among these, biological research concentrating on microbial pathogenesis and infection prevention has seen an increase in biofilm investigations during the past two decades. Resistance develops in bacteria by "natural selection" or "genetic mutation." The varied forms of drug resistance that exist today may result from mutations occurring in different segments of the genome. For instance, a mutation in a chromosomal gene that transports a specific antibiotic into the bacterial cell decreases the pathogen's capacity to transport that antibiotic, while a mutation in a different gene changes the intracellular target protein for a different antibiotic, reducing the antibiotics' inhibitory effects. The antibiotic-resistant gene is passed down from altered parental cells to its offspring by natural selection. Everyeach generation has the potential to strengthen any existing resistance characteristics. However, bacterial genetics alone cannot explain the rise in resistance; human behavior, such as the careless use of antibiotics in the cattle industry, also plays a crucial role. Avoiding these behaviors could help the community's resistance from spreading.

**KEY WORDS:** Biofilm, Formation, Nasal, Isolates, Antibiotic, Resistance, Bacterial Genetics.

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## INTRODUCTION

When Antony Van Leuwenhoek detected tiny organisms termed "animilicules" on the encrustation of human teeth in the 16th century, the term "biofilm" was first used. Leeuwenhoek could recognize the complexity and compactness of these animilicules on the tooth surface despite his ignorance of microbes. Later, in the 1940s, Heukelekian and Heller provided an example of how they perceived microbial formation and growth on surfaces. In their work that was printed in the Journal of Bacteriology, they provided the first description of "bacterial slime" German researchers like Claude Zobell studied the microbial communities' surface attachment phenomena. He was able to count more bacterial cells on a surface than were present in the sea water that was nearby Scientists were able to distinguish between the nature of microorganisms in planktonic condition (free living condition) and microbial communities at the end of the 20th century thanks to a solid understanding of microbial communities. Characklis extended the microbial communities developed on industrial water systems, referring to these communities as microbial slime. However, none of these researchers utilized the phrase "biofilm." The term "biofilm" was originally used in a description published in Microbial Ecology in 1975 by Mack WN, Mack JP, and Ackerson AO. To explain its development on a trickling waste water filter, they came up with the term "biofilm" By tracing extracellular polymeric substances (EPS) with ruthenium red and osmium tetroxide, they also helped to pioneer the field of biofilm research. J. William Costerton, popularly known as "Bill Costerton," has recently made significant contributions to the field of biofilm biology and is now regarded as the "Father of Biofilm Biology" .

## STRUCTURE AND DEVELOPMENT OF BIOFILMS

Due to the release of EPS, bacteria clump together to form biofilms on surfaces. One or more types of bacteria can create them. The structural and functional unit of biofilms is represented by microcolonies, and because these microcolonies are closely related, they may withstand severe environments better. Because of their unparalleled durability, these microcolonies also favor the exchange of resources and nutrients between cells. Additionally, the biofilm's cells exchange extrachromosomal DNA, which leads to treatment resistance. When compared to their counterparts in the planktonic realm, these qualities have given them a distinctive identity. Many scientists have been motivated to learn the hidden secrets of this

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complex creature by the multifaceted character of biofilms. According to studies, biofilms have a higher cell density (about 108 to 1011 cells per gram of wet weight) and are composed of several species in their natural habitat. In addition to their complexity, biofilms alter structurally and functionally at various times throughout their development. At various stages of biofilm growth, it primarily involves the timely expression and secretion of a variety of proteins and signaling molecules. The next five stages broadly correspond to biofilm development .

1. The initial unfastened attachment Cells will only hazily cling to the surface at this stage. The Brownian movement, hydrodynamics, and gravity all favor this stage. In addition to this, the attachment is also influenced by the surface's temperature, pH, ions, and nutrient availability.

2. Secondary firm attachment - At this stage, cells begin secreting biomolecules, which helps them adhere to surfaces irreversibly. EPS is the main biomolecule in charge of this connection.

3. Functional maturation: Microcolonies enter a state of quiescence and exhibit decreased metabolic activity at this stage. The expression of genes and proteins is active in certain of the active cells. Additionally, they secrete chemicals that trigger quorum sensing.

4. Structural maturation - This step is accomplished by the secretion of extracellular DNA (eDNA) and other sticky proteins besides EPS. The mature biofilm is greatly stabilized and structurally supported by these biomolecules.

5. Dispersion - A mature biofilm is made up of an active microbial community that keeps sharing metabolites with the deeper biofilm cells from the nearby area. Most microbial biofilms have also evolved to disperse at a specific time based on the surrounding environmental circumstances when these active cells become dormant, which makes dispersion unavoidable for a mature biofilm. These elements cause the biofilm to lose its structural stability and the cells to disseminate throughout the media. Over time, these cells disperse and plant biofilm on additional surfaces, and the biofilm cycle continues.

## ANTIBIOTIC RESISTANCE

Antibiotic resistance has been identified as a potential risk factor for biofilm development, which is also a major cause of nosocomial and community-acquired infections. Both host immune system and

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antibacterial activities are hampered by biofilm. S. aureus produces biofilms, which is a key component of its virulence. A phenotypic shift in S. aureus to adapt to its surroundings in the face of environmental stressors is the production of biofilm and is a documented technique used by some organisms to initiate and maintain specific infections, as well as a means of enhancing their virulence and antibiotic tolerance. S. aureus biofilm is the source of illnesses like endocarditis, osteomyelitis, and infections from medical devices, which are difficult to treat with antibiotics. Data on the incidence of biofilm development among staphylococcal nasal carriage in India are scarce. Similar to this, U Nagaraju et al. reported one of the earliest cases of nasal carriage in India, where he discovered 10.9% carriage, while reported Nasal Carriage of Staphylococcus aureus in HIV-Infected Individuals. The community is concerned about the screening for MRSA nasal carriers in especially given the pathogen's propensity for multidrug resistance, which leads to numerous infections as well as biofilm development that makes them resistant to treatments and causes chronic infection. There are several techniques for detecting the development of biofilms, including Congo red agar plates, tubes, micro titre plates, and molecular techniques. The most well-liked of them is the micro titre plate/Tissue culture plate approach.

## RESEARCH METHODOLOGY

The purpose of the study was to determine the prevalence of S. aureus and MRSA nasal carriage among HIV-positive individuals as the case group and healthy individuals as the control group in Agra and the surrounding areas, as well as to determine the likely risk factors and the impact of biofilm on antibiotic resistance.

## SCREENING AND SELECTION OF STUDY POPULATION:

**Case Group:** Individuals who were HIV-positive after being tested at the ICTC, microbiology department, S. N. Medical College, Agra, and who wanted to contribute their samples and take part in the study. All patients' acquaintances, family members, and other participants who volunteered to participate in the study and who were tested as HIV-seronegative served.

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## PATIENT S DEMOGRAPHY:

Patients' demographic data, including age, sex, marital status, level of education, occupation, socioeconomic standing, close contact with someone who has been diagnosed with staphylococcal infection, prior staphylococcal infection, use of antistaphylococcal antibiotics, history of hospitalization, recent surgery, fever, skin lesions/SSTI, smoking, alcohol use, HIV status, antiretroviral therapy, and CD4 count, are collected during the sample collection process.

## SAMPLES PROCESSING:

Nasal swabs from every patient and the control group were taken, and they were subsequently seeded into the blood agar (BA) and mannitol-salt agar (MSA) media. Overnight, seeded Petri plates were incubated aerobically at 370°C in the incubator. While colonies from MSA-medium were yellow/pink/colorless, those from BA-medium were white/yellow/creamy in color. The acquired colonies were tested for DNase, coagulase, catalase, and Gram's staining. The isolates that tested positive for the enzymes catalase, coagulase, DNase, and mannitol fermentation were identified as SA. Colonies of SA were then put into MHA medium with 6 g/ml of Oxacillin and 4% sodium chloride, and any growth identified in this medium was determined to be MRSA. The 30-gram dose of cefoxitin was also evaluated for susceptibility during the antibiotic susceptibility testing. MRSA is defined as the antibiotic inhibition zone with a diameter of 21 mm.

## RESULTS AND DISCUSSION

In our investigation, MRSA nasal carriage was more common in HIV-positive individuals (18.75%) than in the control group (6.25%), and 78% of MRSA isolates were able to form biofilms, compared to 33% in the control group. According to the TCP Method, 22% of MRSA were powerful biofilm makers, compared to 0% in the control group. By using the TCP approach, MRSA was a Moderate biofilm producer in 56% of the cases and 33% of the controls, respectively.

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In the tube method, 67% of the MRSA isolates produced biofilm, of which 17% and 50% were strong and moderate producers. In the control group, however, none of the MRSA isolates produced biofilm. In the CRA technique, 28% of MRSA produced biofilms, but none did so in the control group. On a Congo Red Agar plate, black colonies were marked as biofilm producers, whereas red colonies were marked as non-biofilm producers.

As a result, the CRA approach indicated that roughly 55% of S. aureus isolates that had been identified as biofilm producers by micro titre plate assay were not biofilm producers. However, the crystal violet micro titre approach similarly reported 100% of the S. aureus isolates that had been identified as biofilm producers by the CRA method.

Although their findings (76.6%) of MRSA that produced slime layers were higher than ours (28%) by the CRA method, Nahla A. Melake et alinvestigation .'s on Methicillin-Resistant Staphylococcus aureus strains isolated among Nasal Carriers reported that 83% of the MRSA were biofilm producing.

Similar findings were made in a Bangalore study by Saroj Golia et al., where it was discovered that 49.45% of S. aureus isolates had biofilm development, with 75.32% of those being methicillin resistant and 30.47% being methicillin sensitive.

Similar to our work, discovered that 78.78% of MRSA produced biofilms. But contrary to our findings, which showed that 22% of MRSA isolates were strong, 56% were moderate, and 22% were non/weak biofilm producers, strong biofilm formation was seen in 52.38%, weak biofilm formation in 26.40%, and biofilm non-producer in 21.21%.

In a similar vein, discovered 15.4% Strong, 19.2% Medium, and 65.4% Weak biofilm producer MRSA when researching Biofilm Formation among Nasal Carriage Methicillin Resistance Staphylococcus aureus. In a manner comparable to our findings, observed 23.5% Strong & 29.4% Moderate Biofilm.

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## Table- 1: Detection of Biofilm by three different methods among cases & Controls

| Bio Film Production | | Tissue culture Plate method | | | | | | | | Tube method | | | | | | | | CRA Method | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Nasal Isolates | No | Total Producer | | Strong Producer | | Moderate Producer | | Weak /Non Producer | | Total Producer | | Strong Producer | | Moderate Producer | | Weak /Non Producer | | Total Producer | | Non Producer | |
| CASE | No | No | % | No | % | No | % | No | % | No | % | No | % | No | % | No | % | No | % | No | % |
| Cases S.aureus (96) | 96 | 38 | 40 | 12 | 13 | 26 | 27 | 58 | 60 | 36 | 38 | 8 | 8 | 28 | 29 | 60 | 63 | 17 | 18 | 79 | 82 |
| (Cases) MRSA (18) | 18 | 14 | 78 | 4 | 22 | 10 | 56 | 4 | 22 | 12 | 67 | 3 | 17 | 9 | 50 | 6 | 33 | 5 | 28 | 13 | 72 |
| (Cases) MSSA (78) | 78 | 24 | 31 | 8 | 10 | 16 | 21 | 54 | 69 | 24 | 31 | 5 | 6 | 19 | 24 | 54 | 69 | 12 | 15 | 66 | 85 |
| CONTROL | | | | | | | | | | | | | | | | | | | | | |
| (Control) S. aureus (48) | 48 | 5 | 10 | 0 | 0 | 5 | 10 | 43 | 90 | 4 | 8 | 0 | 0 | 4 | 8 | 44 | 92 | 0 | 0 | 48 | 100 |
| (Control) MRSA (03) | 3 | 1 | 33 | 0 | 0 | 1 | 33 | 2 | 67 | 0 | 0 | 0 | 0 | 0 | 0 | 3 | 100 | 0 | 0 | 3 | 100 |
| (Control) MSSA (45) | 45 | 4 | 9 | 0 | 0 | 4 | 9 | 41 | 91 | 4 | 9 | 0 | 0 | 4 | 9 | 41 | 91 | 0 | 0 | 45 | 100 |
| overall (144) S. aureus | 144 | 43 | 30 | 12 | 8 | 31 | 22 | 101 | 70 | 40 | 28 | 8 | 6 | 32 | 22 | 104 | 72 | 17 | 12 | 127 | 88 |
| overall (296) CONS | 296 | 73 | 25 | 25 | 8 | 48 | 16 | 223 | 75 | 57 | 19 | 17 | 6 | 40 | 14 | 239 | 81 | 16 | 5 | 280 | 95 |

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While from Pune, Maharashtra, identified 26.07% of MRSA isolates as Strong biofilm-producers, which is slightly larger than our findings, study conducted in Tamilnadu revealed 16.76% of the MRSA isolates to be Strong biofilm Producers using TCP technique.

found that among burn patients in an Iranian study similar to this one, 97.5% of MRSA were biofilm producers, 17.5% were strong, 62.5% were moderate, and 20% were weak/non biofilm, compared to 60% of MSSA who were biofilm producers, 8% were strong, 24% were moderate, and 68% were weak/non biofilm. By using the Tissue Culture Plate method, observed that 16.76% of the MRSA isolates were powerful biofilm makers. This entire discovery supports the outcome we observed. In their investigation into the development of biofilms among clinical isolates of MRSA from HIV-positive individuals in Limpopo Province, South Africa, found that 10% Strong, 38% Moderate, and 52% Weak biofilm producers of MRSA. This finding was less significant than ours.

The TCP approach, which has been referenced and reported by numerous authors before, was regarded as the gold standard in this study when comparing alternative methods for detecting the formation of biofilms.

TCP approach was shown to be the best way in our study when comparing several methods for detecting the formation of biofilm. TCP technique had a sensitivity of 100.00% (95% CI: 95.89% to 100.00%), tube method had a sensitivity of 79.55% (95% CI: 69.61% to 87.40%), and CRA method had a sensitivity of 31.82% (95% CI: 22.29% to 42.61%). Similar to this, the specificity for the TCP, TM, and CRA methods was observed to be 100.00% (95%CI-97.24% to 100.00%), 98.48% (95%CI- 94.63% to 99.82%), and 99.24% (95%CI-95.85% to 99.98%), respectively. According to statistical data, the accuracy of the TCP technique is 100.00% (95% CI- 98.34% to 100.00%), the accuracy of the Tube method is 90.91% (95% CI- 86.31% to 94.36%), and the accuracy of the CRA method is 72.27% (95% CI-65.86% to 78.08%).

the TCP method identified 22.7% Strong, 41% Moderate, and 36.3% Weak/Non-Biofilm Producers, while the Tube method identified 21% Strong, 30% Moderate, and 51% Weak/Non-Biofilm Producers, and the CRA method identified 3.6% Strong, 6.3% Moderate, and 90% Weak/Non-Biofilm Producers.

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They discovered that the Tube Method had 73% sensitivity, 92.5% specificity, 94% PPV, 66% NPV, and 80% accuracy while the Congo Red Agar Method had 11% sensitivity, 92% specificity, 73% PPV, 37% NPV, and 41% accuracy when they assumed the Microtitre plate method as the gold standard. This discovery agreed with our findings.

Several publications have concluded that the Tissue Culture Plate method is the most sensitive, accurate, and repeatable screening approach for the detection of biofilm formation.

Although the majority of authors cited the Tissue Culture Plate/Microtitre Plate Method as the most effective and trustworthy method with the highest sensitivity to identify the formation of biofilms in vitro, numerous other researchers recommended the Tube Method as a more effective tool for this while others recommended the CRA Method as a more effective tool and may be used as an alternative phenotypic test for the TCP Method. Through the use of the tissue culture/microtitre plate method (TCP) and the tube method (TM), it was simple to distinguish between microorganisms that produced strong biofilms, moderate biofilms, and weak or non-biofilms, whereas the Congo red agar (CRA) based method could only distinguish between microorganisms that produced slime layers and biofilms and those that did not. The TCP technique was simpler to read and more sensitive. For significantly biofilm-producing bacteria, the tube approach correlates well with the TCP test, but because various observers saw different findings, it was challenging to distinguish between weak and biofilm-negative isolates. High variability was consequently seen, making the tube method's classification of biofilms as positive or negative challenging. In line with earlier results, the tube test cannot be suggested as a universal screening test to find isolates that produce biofilm.

All three approaches are useful for identifying S. aureus strains that produce biofilms, however given our observations, we are unable to endorse the CRA method for identifying S. aureus/MRSA isolates that produce biofilms. Our results show that the TCP approach is a precise and repeatable screening method, and this method may be used as a trustworthy quantitative instrument to assess biofilm development by nasal isolates of staphylococci. We therefore advise using the TCP technique to identify biofilm development in S. aureus isolates.

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In our investigation, the resistance pattern between case and control demonstrated a statistically significant difference (P=0.05). Similarly, a statistically significant difference was seen in the resistance pattern of S. aureus in biofilm producers and non-producers (see tables – 15 to 20).

Teicoplanin, Linezolid, and Vancomycin resistance among Biofilm makers & Non Producer MRSA in Case Group (Table-19) is nonexistent. The statistical data for resistance reveals a significant difference against amikacin (P= 0.0021), azithromycin (P= 0.0026), chloramphenicol (P=0.0003), cotrimoxazole (P=0.0001), gentamycin (P=0.0005), levofloxacin (P=0.0463), and tetracycline (P= 0.0021), but not against Clindamycin (P= 0.5019), ciproflox Similar to this, a statistical examination of MRSA biofilm producers in patients and controls (Table 20) reveals a significant difference against all non-beta lactam antibiotics (P= 0.05). There was no evidence of Teicoplanin, Linezolid, or Vancomycin resistance. According to standards, 83.33% of MRSA isolates had multidrug resistance (MDR), of which 22.22% exhibited resistance to six different classes of antibiotics. 70.6% of the MRSA biofilm producers were multi-drug resistant.

**Table- 2: Comparison of different methods for detection of Biofilm formation (considering TCP method as Gold standard)**

| | **TCP Method** | | **TM Method** | | **CRA Method** | |
|---|---|---|---|---|---|---|
| **STATISTICS** | **Value** | **95% CI** | **Value** | **95% CI** | **Value** | **95% CI** |
| **Sensitivity** | 100.00% | 95.88% to 100.00% | 79.54% | 69.60% to 87.30% | 31.82% | 22.28% to 42.61% |
| **Specificity** | 100.00% | 97.23% to 100.00% | 98.47% | 94.62% to 99.82% | 99.24% | 95.84% to 99.98% |
| **Positive Likelihood Ratio** | - | - | 52.5 | 13.20 to 208.56 | 42 | 5.81 to 303.08 |
| **Negative Likelihood Ratio** | 0 | - | 0.21 | 0.14 to 0.30 | 0.69 | 0.60 to 0.68 |

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| | | | | | | |
|---|---|---|---|---|---|---|
| **Disease prevalence** | 40.00% | 33.36% to 46.81% | 40.00% | 33.36% to 46.81% | 40.00% | 33.46% to 46.81% |
| **Positive Predictive Value** | 100.00% | - | 97.22% | 89.81% to 99.29% | 96.55% | 79.50% to 99.50% |
| **Negative Predictive Value** | 100.00% | - | 87.74% | 82.60% to 91.50% | 68.48% | 65.31% to 71.59% |
| **Accuracy** | 100.00% | 98.24% to 100.00% | 90.81% | 86.31% to 94.36% | 72.37% | 65.75% to 78.08% |

observed 100% resistance to ofloxacin, tetracycline, ciprofloxacin, and cotrimoxazole as well as 100% sensitivity to vancomycin in their investigation conducted in Tamilnadu. This observation fits with what we discovered. In this investigation, ciprofloxacin, cotrimoxazole, and clindamycin showed the highest levels of resistance; however, tetracycline shown good efficacy. Vancomycin sensitivity was also 100%. Similar to our observation, discovered a statistically significant difference in the antibiogram between biofilm producer & Non Producer isolates.

The highest rate of multiple drug resistance was found among clinical isolates, 18.7%, according to research of Staphylococcus aureus isolated from HIV patients in the Limpopo Province of South Africa. Resistance to isolates of S. aureus that create biofilms and isolates that don't produce biofilms didn't show any discernible differences. This outcome was at odds with what we discovered. Vancomycin (10.3%) & Teicoplanin (17.9%) resistance was shown to be more prevalent in Nasal MRSA isolates that create biofilms, according to Nahla Erythromycin (100%), clindamycin (75%), ciprofloxacin (75%), SXT (75%), gentamycin (50%), tetracycline (0%) & amikacin (0%) were all observed to be resistant . For biofilm producer MRSA, which is essentially identical to our observation, erythromycin (64%) and the antibiotics tetracycline and amikacin (0% vs. 29%) are the only differences.

Similar to our study results, found no statistically significant difference in ciprofloxacin and vancomycin resistance between biofilm producers and non-producers.

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Similar to our findings, found considerable resistance to erythromycin, clindamycin, tetracycline, fluoroquinolones, and cotrimoxazole against clinical isolates of MRSA that build biofilms.

Among others: All MRSA and MSSA isolates are shown to be sensitive to linezolid, teicoplanin, and vancomycin,. The percentage of resistance to ciprofloxacin was constant (51.28%), but resistance to gentamycin (71.8%), amikacin (64.1%), tetracycline (89.74%), and erythromycin (87.18%) increased, while mupirocin (10.26%) decreased. Amikacin (31.6%), Ciprofloxacin (57.9%), Vancomycin (0%) and Linezolid (0%) showed very identical patterns of antibiotic resistance, but erythromycin (89.5%) and gentamycin (79%) showed enhanced resistance.

## CONCLUSION

In response to environmental difficulties, S. aureus exhibits phenotypic changes, such as the creation of biofilms, which help it adapt to its environment, maintain particular infections, and increase its levels of antimicrobial resistance. A low concentration of antibiotic residues, , can cause biofilm formation and boost the spread of antibiotic resistance genes., once the biofilm has formed, antibiotic efficiency is drastically decreased, and the growth of the biofilm is stimulated by several antibiotics at sub-inhibitory concentrations, such as linezolid and clarithromycin. In some strains of shown that sub minimum inhibitory doses of -lactam antibiotics dramatically stimulate extracellular DNA release that is reliant on autolysin and biofilm formation. The quantity of biofilm induction was up to ten times more and inversely related to how much biofilm the strain naturally formed when not exposed to antibiotics.

The most Methicillin-induced biofilm induction was seen in MRSA strains of lineages USA, USA, and USA. In contrast to MSSA strains, which often exhibit significant levels of biofilm formation in the absence of antibiotics, found that MRSA strains showed greater biofilm induction. 2In their investigation, they found that sub-MIC concentrations of -lactam antibiotics cause the MRSA strain LAC/USA to develop biofilms, but they did not find the same effect with non—lactam antibiotics. It is well known that the bacteria in biofilm are substantially less vulnerable to antibiotics due to the extracellular matrix's inactivation of the antimicrobials and their low penetration through the surface layer covering the microbial community.

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Extracellular DNA cleavage results in the development of a modified biofilm, which enables greater antibiotic penetration. As a result, DNase increases the effectiveness of antibiotics, reducing biofilm biomass and CFU counts.

MRSA that produces biofilm and is isolated from a nasal carrier can easily spread to hospital patients and other members of the public. Therefore, it is concerning for community health, particularly for those who are immunocompromised like HIV-positive individuals who are susceptible to such type of illnesses. Concern should be expressed over research showing an increase in antibiotic resistance among biofilm producers. Therefore, routine screenings for MRSA, biofilm formation, and the carrier status of S. aureus strains, HIV patients, and HIV patients should be conducted. Consecutive detection of potential relationships between biofilm formation and pathogenesis as well as profiles of S. aureus/MRSA drug resistance will undoubtedly result in better control strategies, particularly for patients who are immunocompromised.

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