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# Detection of Virulence Factors and Extensive Drug Resistance in Staphylococcus Aureus Isolated from Clinical Specimen and Nasal Carriage in Erbil City

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***Abstract*--***Staphylococcus aureus (S. aureus) is the causative agent of a variety of infections with generally significant morbidity and mortality. S. aureus colonize healthy people, nasal colonization can cause opportunistic and sometimes life-threating infections. Nasal carriage with virulent strains of methicillin resistant S. aureus (MRSA) poses a high risk for infection. This study was undertaken to assess the S. aureus nasal carriage rate. Also to isolate S. aureus strains from patients, including MRSA strains. The clinical samples include sputum, urine, wound, burn, and vaginal swabs. Phenotypic tests were used to detect virulence factors, and to detect the incidence of multidrug- resistance (MDR), extensive drug- resistance (XDR), and pan drug-resistance (PDR). The positivity of clinical samples was (52.26%), and percentage of S. aureus in positive cultures was (18.45%), so it's the predominant pathogen. In healthy participant's (20.33%) were nasal carriers of S. aureus. In S. aureus isolated from clinical samples biofilm formation (72%), hemolysin production (100%), penicillinase production (90%), multidrug- resistance (52%), MRSA (28%), extensive drug- resistance (16%), and no pan drug-resistance was isolated. In S. aureus isolated from nasal carrier biofilm formation (62%), hemolysin production (100%), penicillinase production (84%), multidrug- resistance (18%), MRSA (4%), extensive drug- resistance (2%), and no pan drug-resistance was isolated. The prevalence of virulence factors among S. aureus isolated from nasal of healthy individuals could indicate predisposition to infections. Eradication of MRSA from nasal carrier will prevent infections in specific patient categories.*

***Keywords*--***Extensive drug resistance, nasal carriage, and methicillin resistance S. aureus.*

## I. INTRODUCTION

*Staphylococcus aureus* (*S. aureus*) is an epidemiologically important pathogen that known for its nasal colonization. *S. aureus* is usually present in 20-40% of the population (Al-haj *et al.*,2018). Nasal carriage of *S. aureus* is the source for staphylococcal infection (Ayepola *et al.*,2015). *S. aureus* carriers among food handlers can cause food contamination resulting food poisoning (Udo *et al.*,2009;Alhashimi*et al.*,2017). An MRSA outbreak involving (18.6%) 8 of 43 patients occurred in surgical ICU of a university hospital in the United States in 1996. Investigation of the cause concluded that a single physician was the source of this outbreak; he was a nasal carrier of MRSA and suffered an upper respiratory infection. The spreading of endogenous *S. aureus* in the air increasing by 40-fold during viral upper respiratory infections and infection outbreaks may occur (Sherertz *et al.*,1996;Sakr*et al.*,2018).

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Bacterial infections can cause disease in 2 million people each year in the United States, and as a result of antibiotic resistant infection 23,000 people die. Estimated deaths with MRSA infection is about 11,000 (Frieden, 2013).

*S. aureus* is a frequent cause of infection in both community and hospital (Wertheim *et al.,* 2005). Vast array of virulence factors such as adhesive surface protein, toxins and enzymes can cause infection. Biofilms are complex communities of bacteria encased in an extracellular polymeric matrix. Biofilm formation is contributing to bacterial virulence reduced clearance by immune system also reduced susceptibility to antibiotics (den Reijer*et al.,* 2016).Biofilm protect specialized dormant cells called persister cells that are tolerant to antibiotics which cause difficult-to-treat recalcitrant infections (Lewis, 2007;Mulani*et al.,* 2019).

*S. aureus* produce coagulase enzyme, which is capable of coagulating blood plasma. *S. aureus* also produce toxins such as hemolysins, alpha α, beta β, gamma ɤ, and delta δ hemolysin. Hemolysin cause cytolysis of erythrocytes (Turk Dagi*et al.,* 2015). Beta-hemolysin was enhanced by incubation below 10°c after treatment at 37°c, and is thus known as "hot-cold" hemolysin. Delta toxin is characterized by synergism effect with beta toxin. Gamma hemolysin is not identifiable on blood agar plates due to inhibitory effect of agar on toxin activity (Cunha and Calsolari, 2008).

Abuse of antibiotics provoked the appearance of multidrug resistant (MDR), also extensively drug resistant (XDR) *S. aureus*, which render the most effective drug ineffective (Mulani*et al.,* 2019). Infections caused by resistant bacteria increase the cost of treatment, extend staying at the hospital, and most importantly cause a significant increase in both morbidity and mortality rates (Yilmaz and Aslantas, 2017). Two mechanisms contribute to penicillin G (Pc) resistance in *S. aureus;* first involving the production of penicillinase encoded by blaZ, which can inactivate Pc by hydrolyzing the B-lactam ring, and second, involving an altered Pc-binding protein, PBP2a, encoded by mecA (Takayama *et al.*,2018). Oxacillin resistant *S. aureus* are resistant to all most recently available beta-lactam antibiotics, except for new cephalosporins with anti-MRSA activity like ceftaroline and ceftobiprole (Ferreira *et al.,* 2017).

Glycopeptides like vancomycin are still common mainstay of therapy for MRSA infections (Sancak, 2011). The prevalence of MRSA carriage increase in a healthy community and, therefore, the surveillance of MRSA in the nasal carriage has been investigated in healthy individuals.

## II. MATERIALS AND METHODS

A total of 519 samples were taken from patients clinically suspected of having wound infection, burn infection, urinary tract infection, vaginitis, and pulmonary infection, admitted to Erbil Hospitals, and 246 nasal swab samples were obtained from apparently healthy individuals (children, students, and workers). Sampling were involved over time period from January to June 2019. The protocol for nasal sampling was consists of rubbing the commercial swab in the anterior nares of each nostril nearly four rotary movements (Bode *et al.,* 2010;Sakr *et al.,* 2018). Standard microbiological methods were used to isolate *S. aureus* including, culturing on selective media (mannitol salt agar), catalase test, API Staph, and tube coagulase test (Alhashimi *et al.*,2017;Abimana*et al.,* 2019).

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### 2.1 Assay for hemolytic activity

The hemolytic activity was evaluated by plating Staphylococci strains on 5% bovine blood for hemolysin production. The criteria for alpha-hemolysin identification were complete (transparent) lytic zone with blurred edges, and for beta-hemolysin identification were incomplete lytic zone (non-transparent), which turn into complete with sharp edges after overnight incubation at 4°c. But for delta-hemolysin identification synergistic hemolysis method described by Hebert and Hancock was used. Beta-hemolytic *S. aureus* vertically seeded and the samples to be tested for delta-hemolysin streaked in sides, as shown in Figure-1 (Ebrahimi *et al.,* 2014).

**Figure 1:** Beta-hemolytic S. aureus verticallyseeded. Samples on two sides are positive for delta-hemolysin.

### 2.2 Penicillin disk diffusion zone edge method for B-lactamase (penicillinase) production

The inhibition halo diameter of the penicillin can be used with zone edge test because the same disk is used in the two tests. This is a low-cost test and comfortably can be used in small and medium-sized laboratories where susceptibility testing is commonly performed by the disk diffusion method (Ferreira *et al.*, 2017).

**Disk diffusion test**: were performed on Muller Hinton Agar(MHA) by applying P10. P10 zone diameters were interpreted according to the CLSI criteria (isolates with a P10 diameter of ≤ 28mm were considered resistant) (Takayama *et al.*, 2018).

**P10 disk zone edge test: -** TheKirby-BauerPc disk diffusion zone of inhibition was virtually assessed as "sharp" or "fuzzy". A "sharp zone/cliff edge " at the inhibition zone around the disk suggested penicillinase production, were as a "fuzzy zone/beach edge " suggested no penicillinase production (Ferreira *et al.*, 2017; Takayama *et al.*, 2018).

### 2.3 Phenotypic MRSA screening

Methicillin resistant in S. aureus was detected by mecA-mediated resistance by using cefoxitin disk (30µg) on Mueller Hinton Agar plate inoculated with test strain as per standard disk diffusion recommendation and incubated at 33-35°c for 16-18 hours. Inhibition zone ≤ 21mm with cefoxitin disk was interpreted as mecA positive according to CLSI guidelines cefoxitin is used as a surrogate marker for mec-A mediated oxacillin resistance (CLSI,2012; Basak *et al.*, 2016).

Also MRSA isolates were identified by the oxacillin disk diffusion method. Cultures with turbidity of a 0.5 McFarland standard inoculated on Mueller Hinton agar, and oxacillin disk (1µg) was placed in the inoculum.

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Inhibition zone diameter were measured and recorded after 24 h of incubation at 35°c (zones were interpreted as follows susceptible ≥ 13mm, intermediate 11-12mm, and resistant ≤ 10mm) (Gorwitz *et al.,* 2008).

**2.4 Biofilm formation**

Polystyrene surface was used for biofilm formation by growing bacterial isolates on plates with 96-flat well. Urine samples were collected from healthy volunteers who didn't use antibiotic in the prior 2 months and had no history of urinary tract infection. Aseptically, urinary samples were collected in sterile containers. After centrifugation at 3000 rpm/5min, 200µl centrifuged urine was added to all 96 wells. Bacterial suspension was prepared by transferring single colonies from each bacterial isolates to the 5ml sterile suspension media (bioMerieux, France). The turbidity of each bacterial suspension was adjusted to 0.50 McFarland Turbidity Range. After that, 5µl from bacterial suspension was transferred to all wells except the two wells were used as control negative, the plates were incubated at 37°C for 24 hrs.

After incubation all plates were washed by phosphate buffer saline (PBS) pH 7.2, for three times to remove unbound bacterial cells, then the plates exposed to air-dryandstained with crystal violet (0.1%).After incubation at room temperature for 30min, the plates were washed off using distilled water and kept for air-dried. The bound bacteria were quantified by addition of ethanol 70% and measurement of the dissolved crystal violet at 600nm using 96-flat wells microtiter plate ELISA reader. The biofilm degree based on the absorbance values obtained for individual isolates as described by Mathur *et al.,* (2006) and Toma, (2010) as shown in Table (1).

**Table 1:** Biofilm degree

| **Biofilm Formation (Absorbance at 600nm)** | **Biofilm degree** |
|---|---|
| 0.04 - 0.059 | No biofilm |
| 0.06 - 0.099 | Weak |
| 0.1 - 0.2 | Moderate |
| > 0.2 | Strong |

**2.5 Criteria for finding multidrug resistance, extensively drug resistance, and pan drug resistance in *S. aureus***

Colonies of *S. aureus* were inoculated in 5ml of 0.85% saline, and the turbidity was adjusted to match 0.5McFarland standard. Then sterile cotton swabs were dipped into the inoculums and spread evenly on to Mueller Hinton agar (Abimana *et al.,* 2019). The following antimicrobial discs were applied aseptically to the MHA plates and incubated overnight at 37°c: vancomycin (VA,30µg), penicillin(P,10U), amoxicillin (AX,25µg), erythromycin (E, 15µg), tetracycline (TE,30µg), amoxicillin-clavulanic acid (AMC,20µg/10µg), methicillin (MET,10µg ),

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cefoxitin (30µg), ciprofloxacin (CIP,5µg), trimethoprim-sulfamethoxazole (SXT,1.25µg/23.75µg), oxacillin (OXA, 1µg), fusidic acid (FA, 10µg), rifampicin (RA, 5µg), clindamycin, (DA, 2µg), and gentamycin (CN, 10µg).

Criteria for finding multidrug resistance, extensively drug resistance, and pan drug resistance in *S. aureus* depictedinworksheet. Multidrug resistance (MDR) one or more of these have to apply: (i) an MRSA is always considered MDR by virtue of being an MRSA (ii) non susceptible to at least one agent in three or more antimicrobial categories. Extensively drug resistance (XDR): non-susceptible to at least one agent in all but two or fewer antimicrobial categories. Pan drug resistance (PDR) non-susceptible to all antimicrobial agents in all antimicrobial categories. Thus, bacteria isolate that is characterized as XDR will also be characterized as MDR. (Magiorakos *et al.,* 2012, Basak *et al.,* 2016).

**Table:2** Worksheet for antimicrobial categories and agents used to define multidrug resistance, extensively drug resistance, and pandrug resistance in S. aureus

| **Antimicrobial categories** | | **Antimicrobial agents** | **Pandrug resistance (PDR)** | **Extensive drug resistance (XDR)** | **Multidrug resistance (MDR)** |
|---|---|---|---|---|---|
| Aminoglycoside | | Gentamicin | x | x | |
| Ansamycin | | Rifampicin | x | x | |
| Beta-lactams | Penicillins | Penicillin | x | x | x |
| | Aminopenicillin | Amoxicillin | x | x | |
| | Penicillinase-stable penicillins | Oxacillin, Methicillin | x<br>x | x<br>x | |
| | Beta-lactam/beta-lactamase inhibitor | Amoxicillin-clavulanic acid | x<br>x | x<br>x | |
| | cephamycins | Cefoxitin | | | |
| Fluoroquinolones | | Ciprofloxacin | x | x | |
| Folate pathway inhibitors | | Trimethoprim-sulphamethoxazole | x | x | |
| Fucidanes | | Fusidic acid | x | x | |
| Glycopeptides | | Vancomycin | x | | |
| Lincosamides | | Clidamycin | x | | |
| Macrolides | | Erythromycin | x | x | x |
| Tetracycline | | Tetracycline | x | x | x |

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## III. RESULTS

In total of 519 clinical samples 271 (52.26%) were positive. 50/271 (18.45%) of positive samples was *S. aureus* as depictedin Table 2,nearly one-fifth of infections caused by *S. aureus.* Results showed that the highest percentage of *S. aureus* were isolated from wound swabs representing about (24.32%). The *S. aureus* nasal carriage rate was 50/246 (20.33%) as depicted in Table 3.

A total of 100 *S. aureus* isolated, 50 isolates were obtained from clinical samples and 50 isolates were obtained from healthy nasal carrier. All cultures investigated by API Staph were Gram positive cocci, positive for catalas, positive for coagulase, and fermented of Manitol salt agar.

In *S. aureus* isolated fromclinical samples beta-hemolysin production were (48%), alpha hemolysin production were (46%), and delta hemolysin production were (6%), in total hemolysin production were (100%). Biofilm formation was (72%), penicillinase production was (92%).

In *S. aureus* isolated fromnasal carrier beta-hemolysin production were (56%), alpha-hemolysin production were (44%), in total hemolysin production were (100%). Biofilm formation (62%), penicillinase production (84%), as shown in Figures (2,3,4,5 and 6) and depictedin Table (4).

These isolates showed different susceptibility towards antibiotics. All isolates of *S. aureus* isolated from clinical samples and *S. aureus* isolated from nasal carrier were sensitive to vancomycin. The effective antibiotic against isolates were ciprofloxacin, fusidic acid, clindamycin, gentamycin, and trimethoprim-sulfamethoxazole. Adversely, the less effective antibiotics were penicillin, and amoxicillin. However, the effect of other antibiotics was variable among the isolates.

In *S. aureus* isolated from clinical samples multidrug- resistance were (52%), MRSA (28%), extensive drug- resistance (16%), and no pan drug-resistance was isolated. But in *S. aureus* isolated from nasal carrier multidrug- resistance (18%), MRSA (4%), extensive drug- resistance (2%), and no pan drug-resistance was isolated as depictedin Tables (5and 6).

**Table 3:** Percentage of isolated Staphylococcus aureus from different clinical samples.

| **Clinical samples** | **No. of samples** | **No. of positive cultures** | **Percentage of *S. aureus* in positive cultures** |
|---|---|---|---|
| Wound swab | 88 | 74/88 (84.09%) | 18/74 (24.32%) |
| Burn swab | 86 | 66/86 (76.74%) | 15/66 (22.73%) |
| Urine | 148 | 51/148 (34.46%) | 8/51 (15.69%) |
| Vaginal swab | 132 | 42/132 (31.82%) | 5/42 (11.90%) |

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| | | | |
|---|---|---|---|
| Sputum | 65 | 38/65 (58.46%) | 4/38 (10.53%) |
| Total | 519 | 271/519 (52.26%) | 50/271 (18.45%) |

**Table 4:** Prevalence of nasal colonization with Staphylococcus aureus

| **Sex** | **Age in year** | **No. of samples** | **Percentage of nasal carrier S. aureus** |
|---|---|---|---|
| Male | 1-20 | 32 | 10/32 (31.25%) |
| | 21-40 | 33 | 7/33 (21.21%) |
| | 41-60 | 31 | 5/31 (16.12%) |
| | ≥ 61 | 32 | 5/32 (15.63%) |
| Sub-total-1 | | 128 | 27/128 (21.09%) |
| Female | 1-20 | 31 | 9/31 (29.03%) |
| | 21-40 | 31 | 6/31 (19.35%) |
| | 41-60 | 29 | 5/29 (17.24%) |
| | ≥ 61 | 27 | 3/27 (11.11%) |
| Sub-total-2 | | 118 | 23/118 (19.49%) |
| Total | | 246 | 50/246 (20.33%) |

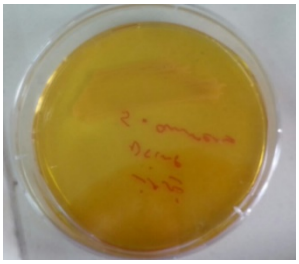

**Figure 2:** Staphylococcus aureuson mannitol salt agar

**Figure 3:** Hemolysin productionby Staphylococcus aureus

**DOI: 10.37200/IJPR/V24SP1/PR201163**
**Received: 16 Jan 2020 | Revised: 06 Feb 2020 | Accepted: 25 Feb 2020**

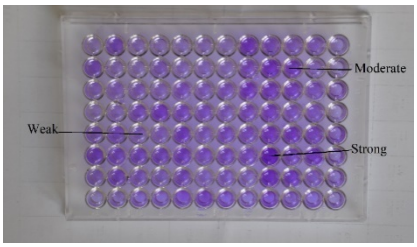


**Figure 4:** Biofilm formationby Staphylococcus aureus in different degree; weak, moderate and strong.

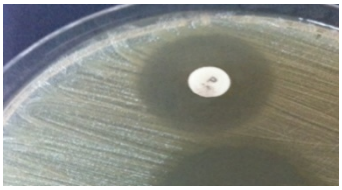

**Figure 5:A**-positive zone edge test (sharp zone/cliff edge)penicillinase production.

**Figure 5: B**- negative zone edge test (fuzzy zone/ beach edge) no penicillinase production.

**Table 5:** Comparison of hemolysin production between Staphylococcus aureusisolated from clinical samples and nasal carrier

| **hemolysin production** | **Type** | **clinical sample *S. aureus*** | **nasal carrier *S. aureus*** |
|---|---|---|---|
| Hemolysin | Beta-hemolysin | 24/50 (48%) | 28/50 (56%) |
| | Alpha-hemolysin | 23/50 (46%) | 22/50 (44%) |
| | Delta-hemolysin | 3/50 (6%) | 0/50 (0%) |
| | Gamma-hemolysin | 0/50 (0%) | 0/50 (0%) |
| Totalhemolysin production | | 50/50(100%) | 50/50(100%) |

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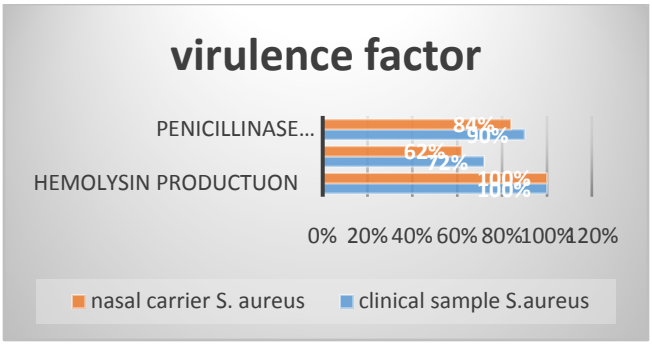


**Figure 6:**Comparison of prevalence of virulence factors between Staphylococcus aureusisolated from clinical samples and nasal carrier

**Table 6**: percentage of sensitivity of clinical sample and nasal carrier *S. aureus*

| **Antimicrobial agent** | **Sensitivity of clinical sample *S. aureus*** | **Sensitivity of nasal carrier *S. aureus*** |
|---|---|---|
| Amoxicillin | 14% | 20% |
| Amoxicillin/Clavulanic acid | 48% | 92% |
| Penicillin | 8% | 16% |
| Oxacillin | 72% | 96% |
| Cefoxitin | 72% | 96% |
| Methicillin | 72% | 96% |
| Ciprofloxacin | 90% | 98% |
| Fusidic acid | 84% | 98% |
| Vancomycin | 100% | 100% |
| Clidamycin | 84% | 94% |
| Erythromycin | 76% | 82% |
| Tetracycline | 74% | 82% |
| Trimethoprium-sulphamethoxazole | 84% | 96% |
| Rifampicin | 78% | 96% |
| Gentamicin | 84% | 98% |

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**Table 7:** Percentage of multidrug-resistance, methicillin resistance S. aureus, extensive drug-resistance, and Pan drug-resistance in clinical sample and nasal carrier Staphylococcus aureus

| **Antimicrobial resistance** | **Clinical sample *S. aureus*** | **Nasal carrier *S. aureus*** |
|---|---|---|
| Multidrug-resistance (MDR) | 52% | 18% |
| Methicillin resistance *S. aureus* (MRSA) | 28% | 4% |
| Extensive drug-resistance (XDR) | 16% | 2% |
| Pan drug-resistance (PDR) | 0% | 0% |

## IV. DISCUSSION

Inour result*S. aureus* is a common cause of infection, and the highest percentage were isolated from wound swab (24.32%). Nearly one-fifth (18.45%) of infections caused by *S. aureus.* This result is similar to fagbomedo and femi-Ola (2017) they reported that (19.32) of wound cultures yield no bacterial growth and the most common bacterial species detected *S. aureus* (25.32%).

The severity of *S. aureus* infections may be related to the biofilm formation and hemolysins production. Ebrahimi *et al.,* (2014) isolated *S. aureus* from wound and skin infection andthey reported that (40.8%) α-hemolytic and (40.8%) β-hemolytic, (2.25%) of *S. aureus* isolates were positive for α and β, and (12.24%) were δ hemolysin producer. (83.7%) of *S. aureus* isolates were biofilm producers.

The penicillin disc zone edge, which appears sharp or fuzzy, has been known to correlate with the production of penicillinase for many years. Recent studies demonstrated that chromogenic cephalosporin-based B-lactamase tests are less sensitive than disc zone edge *interpretation* when they are compared with detection of *blaZ* by PCR (Papanicolas*et al.,* 2014). In 2012, CLSI started to recommend the zone edge test. This test was found to be more sensitive than the nitrocefin method in detecting beta-lactamase production in *S. aureus* (Ferreira *et al.,* 2017). The *S. aureus* nasal carriage rate was (20.33%). The results show the prevalence of virulence factor among nasal carrier *S. aureus.* The results also show that the carriage of MRSA (4%) exists among healthy persons. Turk Dagi*et al.,* (2015) reported that *S. aureus* was isolated in (17.3%) nasal samples. (97.1%/) isolates were MSSA and (2.9%) were MRSA. But Lo *et al.,* (2007) from Taiwan, found that (25%) of children had *S. aureus* carriage, and (13.2%) of children had MRSA.

The prevalence of MRSA nasal colonization in the healthy Maltese population was found to be (8.81%), United Kingdom (1.5%), Greece (0.94%) and Taiwan (3.5%) (Scerri *et al.,* 2013). MRSA nasal colonization has been reported in different African countries at different prevalence, for instance (12.7%) in Ethiopia, (35.8%) in Botswana, and (46%) in Uganda (Abimana*et al.,* 2019).

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The prevalence of colonization with MRSA in the United State population in 2001-2002 was (0.8%) and it was (1.5%) in 2003-2004. This represented an increase in the prevalence of colonization with MRSA. An increase in colonization with MRSA was anticipated, one potential explanation is an increase in population-level exposure to antimicrobial agents that may suppress MSSA and promote colonization with MRSA (Gorwitz*et al.,* 2008).

Alhashimi*et al.,* 2017, reported that the prevalence of nasal carriage *S. aureus* is high (30.1%) among food handlers in Karbala city. Therefore, strict measure is necessary to prevent food contamination with *S. aureus* during food handling.

Many persons are colonized with *S. aureus*, MRSA colonization prevalence may vary with organism characteristics and demographic (Kuehner*et al.,* 2006).

*S. aureus* isolateswhether MSSA or MRSA showed a high resistance to amoxicillin and amoxicillin-clavulanic acid.In 2009 on the antimicrobial susceptibility pattern of *S. aureus*, cultured from clinical samples in 97 surgical wards located in Ukraine, the prevalence of MRSA ranged from (10.8%) to (84.1%) depending on the location (Netsvyetayeva*et al.,* 2014).Some *S. aureus* strains remains susceptible to penicillin. Thus, this antimicrobial agent remains the treatment of choice for patients infected with penicillin susceptible isolated (Takayama *et al.,* 2018).

Fusidic acid is topical drug which is used for the treatment of Staphylococcal skin infection. Clindamycin is an alternative drug for the treatment of skin and soft-tissue infections caused by both MSSA and MRSA. Also, this antibiotic is an alternative drug to be used in penicillin-allergic patients. On the other hand, resistance to this antibiotic reduce the efficacy of the drug (Yilmaz and Aslantas, 2017). Oguzkaya-Artan*et al.,* (2018) determined fusidic acid resistance in (5.6%) of *S. aureus* strains isolated from the nasal cavities of healthy preschool children.
Eradication of MRSA nasal colonization is widely inferred to prevent infections in high risk subjects e.g. surgical patients (Critchley, 2006).

The prevalence of virulence factors, MRSA and extensive drug resistance among *S. aureus* isolated from nasal of healthy individuals could indicate predisposition to infections or contamination of food with *S. aureus* duringfood handling. Efforts to prevent such threat is by reducing person to person spread through screening, treatment and eradication of *S. aureus.*

## V. CONCLUSION

The results pinpointed on the prevalence of MRSA and extensive drug resistance *S. aureus* (EDRSA) among healthy individuals. This may indicate the accelerated spread of MRSA in the community. Therefore, prophylaxis must be taken from MRSA endogenous flora, and continuing surveillance is needed to develop strategies that control the spread of MRSA and extensive drug resistance *S. aureus* (EDRSA) among patients and healthy individuals.

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