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DETECTION OF BIOFILM FORMATION AND SOME VIRULENCE FACTORS IN *PSEUDOMONAS AERUGINOSA*, AND THE EFFECT OF SOME ANTIBIOTICS

Objective. *Pseudomonas aeruginosa (P. aeruginosa) is a present everywhere and opportunistic bacterium pathogen. The existence of numerous virulence factors i.e. exo-toxin, exo-enzyme genes, and biofilm may be contributed in the pathogenesis and pathogenicity of the bacterium. The* ***goals*** *of the present work were to detect biofilm formation, some biofilm genes, and the effect of antibiotics against P. aeruginosa.* ***Methods.*** *All isolates were identified using API 20E and 16S rRNA techniques. The microtiter plate method (MTPM) was used to detect biofilm formation. The polymerase chain reaction (PCR) was used to find some virulence genes e.g. pelA, pslA.* ***Result****s. A total of 64 P. aeruginosa isolates were identified as P. aeruginosa. The majority of infection belonged to burn infections — 27 (42.2%), followed by ear — 17 (26.5%), and urine — 20 (31.3%). The results of biofilm detection using MTPM showed that all P. aeruginosa isolates were able to produce biofilm but at different levels. PCR technique was used to detect biofilm genes. Studies showed that 61 (95.30%) and 63 (99.32%) isolates carried pelA and pslA genes, respectively. Moreover, a susceptibility test was used to select 10 antibiotics. P. aeruginosa isolates were resistant to cefotaxime — 61 (95.3%), carbenicillin — 59 (92.2%), ampicillin — 38 (59.4%), piperacilin/tazobactam — 29 (45.3%), streptomycin — 28 (43.8%), moxifloxacin — 27 (42.4%), ticarcilin — 26 (40.6%), ciprofloxacin — 24 (37.5%), gentamicin — 20 (31.3%), and neomycin — 13 (20.3%).* ***Conclusions.*** *Biofilm is produced by P. aeruginosa at different levels. The molecular technique showed that the pelA and pslA genes are associated with the form of biofilm in P. aeruginosa isolates. The susceptibility tests showed that the most active antibiotics against P. aeruginosa were neomycin, gentamycin, and ciprofloxacin, respectively.*

Keywords: *Pseudomonas aeruginosa, biofilm, virulence factors, antibiotics.*

Pseudomonas aeruginosa plays an important role in hospital-acquired and life-threatening infections. Furthermore, it is one of the leading hospital- acquired infection pathogens affecting humans hospitalized while being essentially resistant to a wide range of anti-microbial agents, i.e. antibiotics [1].

The ability of *P. aeruginosa* to form biofilms is considered the main reason for the pathogenesis of chronic *P. aeruginosa* infections, which form

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communities of the causative agents in a self-producing polymeric matrix during different stages of the disease [2]. There is a relationship between *P. aeruginosa* and susceptibility to antibiotics, which demonstrates real resistance to numerous anti-microbial agents, such as antibiotics [3]. In addition, Schulzeet al. [4] clarified that biofilm is a microorganism population of microbes, which are enclosed by the self-produced slime or matrix of extracellular polysaccharide (EPS). EPS is considered a major matrix component of biofilm and can be significant in the development of biofilm. Motta, et al. [5] stated that the form of biofilm represents a complex group of sessile cells, structured organized bacterial cells, which are attached to surfaces that grow and interact as a community. Also, Colvin, et al. [6] mentioned that several isolates *P. aeruginosa* are proficient at forming biofilms, have the capability to produce many kinds of sugars, and produce at least 3 types of EPS, alginate (*alg*). EPS contains glucose, and rich polysaccharide, which different cellulose, as known psl, and exopolysaccharides containing D-mannose rhamnosus, and D-glucose known as pel. Moreover, in mucoid strains of *P. aeruginosa*, *alg* is the prevalent EPS of the matrix. In addition, non-mucoid strains of *P. aeruginosa* produce mostly *pel* and *psl* polysaccharides to form vital membranes [7]. There are different stages of biofilm formation by *P. aeruginosa* mediated by EPS i.e. pili and flagella. These structures play an important role in adherence [8]. Strateva and Yordanov [9] and Breidenstein, et al. [10] observed the resistance for many anti-microbial agents via permeability of the outer membrane.

Table 1. **Primers tested for *P. aeruginosa* and selected primer to biofilm genes**

Gene		Primer sequence	Size of product (bp.)
16S rRNA	F	TCACGCTATCAGATGAGCC	
	R	TAACTTACTGCCCTTCCTCCC	250
PelA	F	CAGCAAGAAAGGAATCGCC	
	R	TGCTCTTTCAGATACCCGAC	258
PslA	F	GGTCCCGGAGAACTACAACT	
	R	GGCATAAGATCGCATCCAGG	159

The **goals** of the present work were to detect biofilm and some biofilm genes, and to investigate the effect of antibiotics on *P. aeruginosa*.

Materials and methods. A total of 250 clinical samples were collected from different sites of infection as swabs from different regions i.e. burn, ear, and urine from patients hospitalized in four different health centers: Al-Zahraa and Al-Karama teaching hospitals, Al-Kut hospital for gynecology, public health laboratory in Al-Kut city/ Wasit province in Iraq. The samples were grown on several traditional and rich media. Initially, bacteria were cultured on the blood agar, then on a selective media such as *Pseudomonas* agar, which was used to detect *P. aeruginosa* isolates, and bacteria cultures were incubated at 37 °C for 18–20 hr. conventional and molecular techniques were used to diagnose several isolates of *P. aeruginosa*. Several microbiological techniques were used for the detection of *P. aeruginosa* isolates, such as the Analytical Profile Index (API) and polymerase chain reaction (PCR) as a molecular technique Mueller Hinton's agar was used to assess the sensitivity of different *P. aeruginosa* isolates to antibiotics.

DNA in several isolates of *P. aeruginosa* was diagnosed using the Geneaid Genomic DNA extraction Kit (USA). DNA was extracted according to the instructions of the manufacturer . The specified primers, e.g. 16S rRNA, were used to detect *P. aeruginosa* isolates, *pelA*, and *pslA* synthesized using Eurofins MWG Operon (MWG, Germany) as follows: 16S rRNA (FJ972538.1), *PelA* (CBYA010000077.1), *PslA* (JQ794873.1), as an accessing number (Table 1).

Preparation of PCR master mix. PCR master mix was prepared using GoTag Green Master Mix Kit (Promega, USA), according to the instructions of the manufacturer, as outlined in Table 2.

Thermocycler program for PCR PCR thermocycler conditions for *P. aeruginosa* were achieved by PCR thermocycler system and the same for each gene except for annealing temperature (At), as summarized in Table 3.

Also, susceptibility tests were conducted by the disc technique for isolates *P. aeruginosa* using various antimicrobial agents. The following antibiotics were used: moxifloxacin (MFX) 10μg, ciprofloxacin (CIP) 5μg, cefotaxime (CTX) 30μg, piperacilin/tazobactam (PTZ) 100μg, ticarcilin (TC) 75μg, ampicillin (AMP) 30μg, carbenicillin (PY) 100μg, gentamicin (GMN) 15μg, streptomycin (RX) 15μg, and neomycin (NMN) 10μg.

The analysis of data and variance was used for all tests with $P < 0.01$.

Results. A total of 250 different clinical samples were collected, which include various sites of human, i.e. burns, urine, and ear (Table 4).

A total of 64 (25.6%) *P. aeruginosa* isolates were obtained from different clinical samples. They were identified according to the conventional and molecular techniques including the culture and microscopic examination, biochemical tests, API 20E kit, Vitek2 system, and PCR. Then all the results were confirmed by molecular techniques, such as 16S rRNA.

The biofilm produced in isolates of *P. aeruginosa* was estimated as complex communities of bacterial cells with adhesion capability to different surfaces i.e. plastics, metals, medical implant materials, and tissues. Biofilm formation enhances the survival of microorganisms such as bacteria, making all of them difficult to damage [11]. In the current study, in total 64 isolates of *P. aeruginosa* were evaluated by the microtitre plate method (MTPM).

The biofilm production was detected using MTPM, and the results demonstrated that all *P. aeruginosa* isolates produce biofilms of different quality as follows: 10 (15.60%), 13 (20.30%), and 41 (64.10%) isolates produce weak, moderate, and strong biofilms, respectively (Fig. 1).

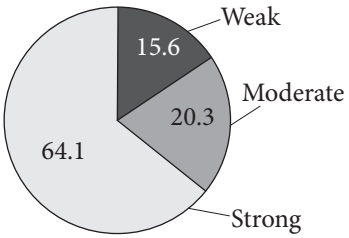

Fig. 1. Different results of using MTP method to detect the production of biofilm in *P. aeruginosa*

Table 2. **Master mix of PCR**

Master mix of PCR	Volume (μL)
Master mix	12.5
Primer forward (10p/mol)	1
Primer reverse (10p/mol)	1
Nuclease free water	8.5
Template of DNA (20–40 ng/μL)	2
Total	25

Table 3. **PCR thermocycler system of 16S rRNA, *pelA*, *pslA* for *P. aeruginosa***

PCR Stage	Temperature (°C)	Time (min)	Repeat (cycles)
Initial Denaturation	95	5	1
Denaturation	95	0.5	
Annealing	53[1], 52[2], 58[3]	0.5	
Extension	72	0.5	30
Final extension	72	7	1
Hold	10	10	

Note: For 16S rRNA[1], *pelA*[2], *pslA*[3]

Table 4. **Number and percentage (No. (%)) of *P. aeruginosa* isolates from different sites of human**

Source of isolate	No. (%)
Burn	27 (42.2)
Ear	17 (26.5)
Urine	20 (31.3)
Total	64 (100)

Note: $P < 0.05$

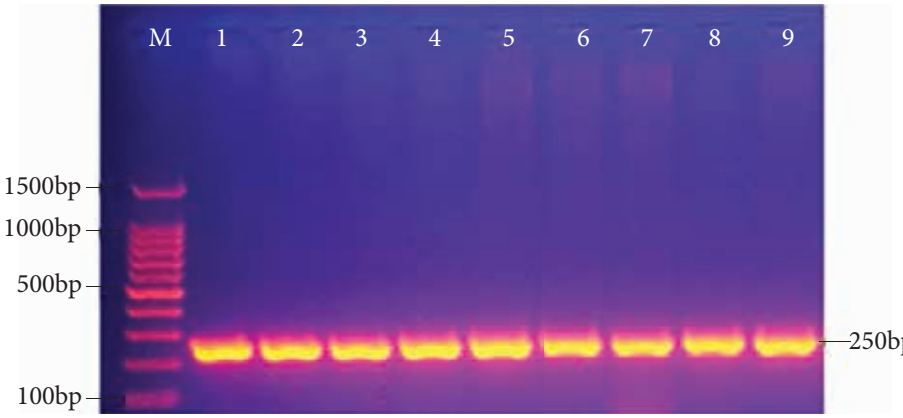

Fig. 2. Agarose gel electrophoresis results for different genes

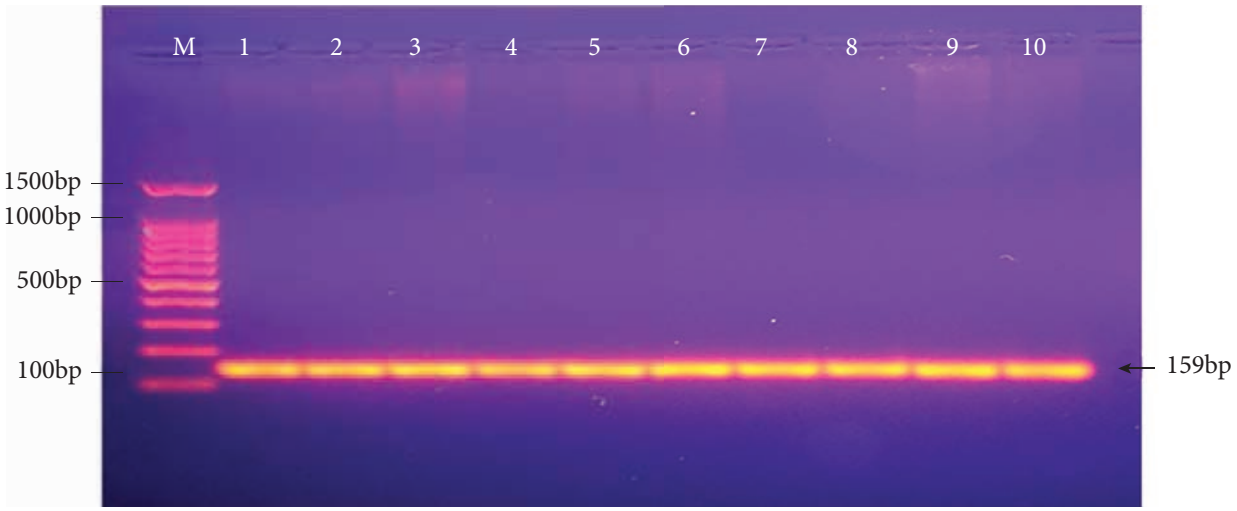

Fig. 3. Agarose gel electrophoresis and genomic DNA isolated from *P. aeruginosa* observed by PCR product analysis for the *pslA* gene in different isolates of *P. aeruginosa* by specified primer, with M: Marker (100—1500bp). All lines were (1—10) positive PCR amplification for *P. aeruginosa* isolates at the 159bp PCR size of the product.

As for agarose gel electrophoresis results for different genes, they are presented in the following figure.

As summarized in Fig. 2, identification of *P. aeruginosa* and biofilm-producing genes by PCR technique, various *P. aeruginosa* isolates were identified by 16S rRNA as PCR+ve.

Likewise, PCR was used to detect the existence of different genes that form biofilm, such as *pelA* and *pslA*, in various isolates of *P. aeruginosa*.

The *pelA* gene was detected in 57 (89.10%) isolates from all isolates of *P. aeruginosa* as PCR+ve, and 7 (10.90%) isolates as PCR-ve. In addition, the *pslA* gene was detected in 61 (95.30%) isolates as PCR+ve, and 3(4.70%) isolates for each as PCR-ve, as shown in Fig. 3.

Also, susceptibility and resistivity *in vitro* to different antibiotics against *P. aeruginosa* isolates were studied (Table 4). All isolates of *P. aeruginosa* were resistant to cefotaxime, carbenicillin, and ampicillin: 61 (95.3%), 59 (92.2%), and 39 (59.4%) respectively. The isolates of *P. aeruginosa* demonstrated a different degree of resistance to each antibiotic, and their number and percentage were: piperacilin/tazobactam 29 (45.3), streptomycin 28 (43.8), moxifloxacin

27 (42.4%), ticarcilin 26 (40.6%), ciprofloxacin 24 (37.5%), gentamicin 20 (31.3%), and neomycin 13 (20.3), as listed in Table 5.

Discussion. The production of biofilm/alginate has been evaluated as a remarkable determinant of the pathogenicity of *P. aeruginosa* infections [12]. Also, Nasirmoghadas et al. [13] mentioned that several mucoid *P. aeruginosa* isolates produce vast levels of alginate, and this was considered an essential component of biofilm.

Moreover, there are two different studies by Prince et al. [14] and Oncel et al. [15] who have observed the ability to form biofilm in 28.6% and 65.2% of different clinical isolates of chronic illness in the upper respiratory tract patients, respectively. On the other hand, Nasirmoghadas et al. [13] have found that *P. aeruginosa* isolates (23.3%) from clinical samples were positive for biofilm formation. In the present study, the predominance of strong biofilm formation was found in 41 (64.10%) *P. aeruginosa* clinical isolates. The difference in the demonstration of biofilm formation between the present and previous studies may be due to the variation in the sites of clinical isolates and conditions of the studies.

As mentioned above, biofilms are complex microbial communities characterized by adhesion capability for different surfaces such as metals, plastics, medical implant materials, and various tissues. The important role in biofilm formation of such factors as alginate, *pel*A, *psl*A in the establishment and persistence of infections has highlighted in the majority of the prior studies conducted by Patankar et al. [16]. These factors play a significant role in the surface attachment for more isolates of *P. aeruginosa* in spite of different contribution of *PelA* and *PslA* to the structure of biofilm. In addition, they are three EPS that form essential structures of the biofilm matrix and numerous biological functions, particularly concerning protection of the bacteria cells from anti-microbial factors and responses of the immune system [17, 18]. Franklin et al. [19] mentioned that the biofilm components in *P. aeruginosa* include at least three specific EPS, namely *Pel*, *Psl*, and alginate.

The current results are close to the ones obtained by Hisham, et al. [20] who found that 16 (80%) isolates produced a strong biofilm, 2 (10%) isolates — a moderate biofilm, and 2 (10%) isolates — a weak biofilm. Also, Abd El-Galil et al. [21] demonstrated that 42 (84%) isolates produced a strong biofilm, 4 (8%) isolates — a moderate biofilm, and 4 (8%) isolates — a weak biofilm .

As for our antibiotic tests, *P. aeruginosa* isolates were resistant to cefotaxime (95.3%) and carbencilin (92.2%), although, the majority of them were sensitive to many antibiotics, such as neomycin (79.7%), gentamicin (69.7%), and ciprofloxacin (62.5%) (Table 5). The resistance may be attributed to gene cassettes, plasmids, and transposons as well as to the widespread use of antibiotics and increased resistance in different pathogenic bacteria [22]. Furthermore, Toukam et al. [23] stated the improper use of antibiotics by professionals of health care and/or non-skilled practitioners as well as by common people, along with adequate surveillance due to the lack of information outgoing from routine antimicrobial

Table 5. **Antibiotic susceptibility and resistivity *for different isolates of P. aeruginosa***

Antibiotics	Resistant		Sensitive		Intermediate	
	No.	(%)	No.	(%)	No.	(%)
Moxifloxacin	27	42.2	36	56.3	1	1.5
Ciprofloxacin	24	37.5	40	62.5	0	0.0
Ticarcilin	26	40.6	37	57.8	1	1.6
Piperacilin/Tazobactam	29	45.3	31	48.4	3	6.3
Cefotaxime	61	95.3	3	4.7	0	0.0
Ampicillin	38	59.4	24	37.5	1	3.1
Carbenicillin	59	92.2	66	7.8	0	0.0
Gentamicin	20	31.3	44	69.7	0	0.0
Streptomycin	28	43.8	31	48.4	5	7.8
Neomycin	13	20.3	51	79.7	0	0.0

Note: $P < 0.01$

susceptibility and resistivity tests, including rotating reports from developing countries. Our results for the sensitivity and resistivity profile are compatible with those obtained by Al-Zaidi [24], Hwang and Yoon [3], and Kamali et al. [25].

Conclusions. In the current study, production of strong biofilms was detected in *P. aeruginosa* isolates, and a high number of data on their formation and susceptibility to antibiotics were obtained and compared with the literature. In particular, the molecular technique showed that the *pelA* and *pslA* genes are associated with the form of biofilm in different isolates of *P. aeruginosa*. The results of the susceptibility and resistivity tests of antibiotics demonstrate that the most active compounds against *P. aeruginosa* are neomycin, gentamycin, and ciprofloxacin.

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ОЦІНКА ЗДАТНОСТІ ДО ФОРМУВАННЯ БІОПЛІВКИ ТА ДЕЯКИХ ФАКТОРІВ ВІРУЛЕНТНОСТІ *PSEUDOMONAS AERUGINOSA* ТА ДІЯ ДЕЯКИХ АНТИБІОТИКІВ

Синьогнійна паличка (*Pseudomonas aeruginosa*) — повсемісно поширена умовно-патогенна бактерія. Існування численних факторів вірулентності, таких як екзотоксин, гени екзоферментів, а також здатність до утворення біоплівки, може сприяти підвищенню патогенності цієї бактерії. **Мета** даної роботи полягала у виявленні здатності до утворення біоплівки та деяких генів, які відповідають за формування біоплівки, а також у визначенні впливу та дії антибіотиків на ізоляти *P. aeruginosa*. **Методи.** Усі ізоляти були ідентифіковані методами API 20E, Vitek2 та 16S рРНК. Для виявлення здатності до формування біоплівки використовували метод мікротитраційних планшетів (МТП). Для виявлення генів вірулентності *pelA* та *pslA* використовували полімеразну ланцюгову реакцію (ПЛР). **Результати**. Всього 64 ізоляти були ідентифіковані як *P. aeruginosa.* Найчастіше цей мікроорганізм виявляли у пацієнтів з післяопіковими інфекціями — 27 (42,2%), на другому місці були інфекції сечового міхура 20 (31,3%) і на третьому — інфекції вуха — 17 (26,5%). За допомогою МТРМ встановлено здатність утворювати біоплівку у всіх ізолятів *P. aeruginosa*, але інтенсивність до утворення біоплівки була різною. Для виявлення генів біоплівки була використана методика ПЛР, яка показала, що 61 (95,30%) і 63 (99,32%) ізолятів несуть відповідно гени *pelA* та *pslA*. Крім того, визначено чутливості ізолятів до 10 антибіотиків. Показано, що ізоляти *P. aeruginosa* резистентні до цефотаксиму — 61 (95,3%), карбеніциліну — 59 (92,2%), ампіциліну — 38 (59,4%), піперациліну/тазобактаму — 29 (45,3%), стрептоміцину — 28 (43,8%), моксифлоксацину — 27 (42,4%), тикарциліну — 26 (40,6%), ципрофлоксацину — 24 (37,5%), гентаміцину — 20 (31,3%) та неоміцину — 13 (20,3%). **Висновки**. Молекулярні методи дослідження показали, що гени *pelA* та *pslA* пов›язані з процесом формування біоплівки ізолятами *P. aeruginosa*. За результатами тесту на чутливість встановлено, що найактивнішими речовинами проти *P. aeruginosa* є неоміцин, гентаміцин та ципрофлоксацин.

***Ключові слова*:** *Pseudomonas aeruginosa*, біоплівка, фактори вірулентності, антибіотики.