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*Original Article*

# Detection of ExoS and Las B virulence genes in *Pseudomonas aeruginosa* isolates from burn patients by PCR in Duhok, Iraq

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## Abstract

Introduction: *Pseudomonas aeruginosa* is a common cause of nosocomial infection in both immunocompetent and immunocompromised individuals. It exhibits innate resistance to a wide range of antibiotics and threatens global public health as a silent pandemic.

Methodology: From September 2024 to March 2025, 120 clinical specimens were collected from patients with infected burns of both genders and various ages at the Burn hospital in Duhok city, Iraq. The obtained *Pseudomonas aeruginosa* isolates were used for the molecular characterization of the virulence genes, the *ExoS* and *Las B,* by Polymerase Chain Reaction (PCR).

Results: The overall prevalence of *Pseudomonas aeruginosa* was 37.5% (45/120), with a significantly higher rate among females than males (45.7% vs 26%). Furthermore, the highest rate of infection (55%) was among ages of > 20-30 years, while the lowest rate, 12.5% was among ages of < 1-10, with a highly significant difference ($p = 0.001$). All of the examined isolates were 100% (22/22) positive for *Exo S* and 95.5% (21/22) for *Las B* genes.

Conclusions: This study underscores the high prevalence of these virulence genes among the *Pseudomonas aeruginosa* isolates from infected burns, indicating their clinical and epidemiological significance. Additionally, their phylogenetic relatedness highlights their pathogenic potential and role in the international dissemination of virulent lineages.

**Key words:** *Pseudomonas aeruginosa*, PCR, Burn, *Exo S*, *Las B*.

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## Introduction

Burns can result from a variety of sources, including heat, radiation, electricity, friction, and chemical contact. Based on estimates, over 180,000 individuals die from burns yearly, the majority of them living in low- and middle-income countries [1]. According to the CDC (2023) [2], burn wounds create the perfect conditions for pathogenic bacteria of both endogenous and foreign origin that thrive in polluted areas. Furthermore, burns can be at high risk of infection with nosocomial and multidrug-resistant bacteria like *P. aeruginosa* or other resistant bacteria while the patient is residing in the hospital [3]. A number of factors, such as immunocompromised individuals, exposed skin surfaces, invasive hospital surgeries, and long stays in the hospital, may result in infections in burn patients [4]. Invasive infection can be affected by the patient's age, body surface area, burn wound depth, and the amount and type of microorganisms, enzymes, or toxins secreted [5]. Furthermore, 75% of all deaths occur in patients with severe burns that cover more than 40 per cent of their surface area due to sepsis and other complications [6].

These bacteria can often overcome the human defenses through the production of a number of virulence agents, like exoenzyme S *Exo S*, that cause infection and tissue damage [7]. The damage can be caused by the production of elastin, which can damage the human lung tissue, *Las B*, a metallo-proteinase enzyme, promotes *P. aeruginosa* infections in burns, pneumonia, and eye lens infections [8]. In medical environments, *P. aeruginosa* can colonize ventilators and other equipment in intensive care units by producing biofilm [9,10]. The current study was adopted to investigate the distribution and the phylogenetic relationship of the virulence genes, the *Exo S* and *Las B* of *P. aeruginosa,* using PCR amplification by species-specific primers among infected burn patients residing in the Burn Hospital in Duhok City/Iraq.

## Methodology

*Sample Collection and Study Design*

In the current study, 120 clinical samples were collected from burn-injured inpatients of both genders who attended the Burn Hospital in Duhok City, Iraq, over a period from September 2024 to March 2025. Patients were excluded if they had received pre-antimicrobial therapy or did not comply with the study's requirements. From each patient, a single swab was

taken before wound dressing from the deep area of the wound containing pus discharge and placed in a tube containing 5mL of brain heart infusion broth. Each tube was labeled with the patient's complete information. Prior to the study an ethical approval was obtained from the authorities of Duhok General Health Directorate for taking patients samples, consent, and data. The collected specimens were transported to the microbiology laboratory of Polytechnic University for culture, identification, and other analysis, such as oxidase and Catalase tests, pigments production (pyoverdine and pyocyanin), as well as Triple Sugar Iron (TSI).

*Isolation and identification of Pseudomonas aeruginosa*

Identification and isolation of *Pseudomonas aeruginosa*. The collected specimens were incubated for 24 hours at 37 °C, followed by culturing on MacConkey (Neogene Company, USA), Nutrient (Neogene Company, USA), Blood (Neogene Company, USA), and Cetrimide agars (Neogene Company, USA) using the streak method. The first diagnosis of isolates was made using Gram's staining of the culture, hemolysis on blood agar, pigment development, and the smell in cultures. The second test relied on the physical and biochemical characteristics of pure colonies [11]. For a long time, all confirmed *P. aeruginosa* isolates were stored at -20 °C in 50% glycerol broth. To do this, a small part of a pure isolated colony was added to 3 milliliters of sterile nutritional broth, and the tube was then incubated at 37 °C.

The isolates were confirmed as *P. aeruginosa* based on positive oxidase and catalase reactions, and the growth characteristics on TSI agar showed an alkaline/alkaline (K/K) reaction without the production of gas or $H_2S$. Additional confirmation was performed by testing their ability to grow at 42 ºC. Furthermore, the production of distinctive pigments (pyocyanin and pyoverdine), the growth on cetrimide agar, positive motility, and a grape-like odor, all of these features support my findings of the isolates as *P. aeruginosa.*

Subsequently, 0.5 mL of the bacterial culture was homogenized with 0.5 mL of sterile glycerol in a sterile tube, thoroughly mixed, and stored at –20 °C. Under these conditions, the isolates remain stable and suitable for molecular analysis for up to six months without undergoing any detectable changes [12].

*PCR Technique used for DNA Extraction and amplification.*

*Pseudomonas aeruginosa* bacterial DNA extraction was performed as directed by the manufacturer; genomic DNA was extracted from bacterial isolates using the High-yield DNA Purification Kit. Prior to storing the yielded DNA, its purity and concentration were estimated using a Nano-Drop TM One UV-Vis Spectrophotometer (Thermo Fisher Scientific, Waltham, MA, USA). The produced DNA was stored in a deep freezer at -20 °C for PCR amplification. The primers used to amplify these *P. aeruginosa* genes are shown in Table 1. The final volume of each PCR reaction was 20 µL, as shown in Table 2. For amplification, the prepared reaction tubes were loaded into the thermal cycler; Table 3 lists the amplification settings for each gene. A 1.5% agarose gel stained with RedSafe dye in Tris-Borate EDTA buffer (10x) was used to electrophorese the PCR products. After operating at 45 volts for 15 minutes, the voltage of the immersed gel was increased to 85 volts for an hour. The

**Table 1.** Primers used for identification and detection of virulence genes.

| Name of primers | Sequence of primers (5'—3') | Molecular weight | References |
|---|---|---|---|
| *Exo-S*-F | CTTGAAGGGACTCGACAAGG | 504 | [13] |
| *Exo-S*-R | TTCAGGTCCGCGTAGTGAAT | | |
| *Las B*-F | GGAATGAACGAAGCGTTCTC | 300 | [14] |
| *Las B*-R | GGTCCAGTAGTAGCGGTTGG | | |

**Table 2.** Protocol of PCR reaction mixture volumes.

| Gene | Volume of reaction | DNA | Primer F&R | Master Mix | D.W |
|---|---|---|---|---|---|
| *Exo S, Las B* | 20 µL (10 pmol) | 3 µL | 2 µL | 10 µL | 5 µL |

**Table 3.** PCR setting for detection of virulence genes present in *Pseudomonas aeruginosa* based on analysis.

| Gene name | Temperature °C /Time | | | | |
|---|---|---|---|---|---|
| | Initial Denaturation | Cycling condition | | | Final Extension |
| | | Denaturation | Annealing | Extension | |
| *Exo S* | 94 ºC, For 5 minutes | 94 ºC, For 30 seconds | 57 ºC, For 30 seconds 35 cycles | 72 ºC, For 30 seconds | 72 ºC for 5 minutes, 1 cycle |
| *Las B* | 94 ºC, For 3 minutes; 1 cycle | 94 ºC; 30 seconds | 60 ºC; 1minute 30 cycles | 72 ºC; 90 seconds | 72 ºC for 5 minutes, 1 cycle |

**Table 4.** The Distribution of *Pseudomonas aeruginosa* in clinical burn specimen.

| Clinical specimen | Number of examine specimen | Positive specimen (%) |
|---|---|---|
| Burn swab | 120 | 45 (37.5) |

**Table 5.** The correlation between *P. aeruginosa* isolation rates and numbers among varying ages and genders.

| Variables | No. Examined | *P. aeruginosa* No % | |
|---|---|---|---|
| **Gender** | | | |
| Females | 70 | 32 (45.7%) | $p = 0.028$; $\chi^2 = 4.837$ |
| Males | 50 | 13 (26%) | |
| **Age (years)** | | No examined from each age group | |
| < 1-10 | 16 | 2/16 (12.5) | |
| > 10-20 | 30 | 15 / 30 (50) | $p = 0.001$, $\chi^2 = 17.211$ |
| > 20-30 | 40 | 22 / 40 (55) | |
| > 30-40 | 34 | 6 / 34 (17.6) | |
| Total | 120 | 45 (37.5) | |

molecular weights of the PCR products were estimated using reference DNA ladders with a range of 100 to 1000. The PCR amplification was verified by viewing the gel under a UV trans illuminator [13].

*Sequence analysis and phylogenetic tree*

For DNA sequencing, the obtained PCR products were submitted to Macrogen company (South Korea) using the Sanger sequenced method, and the obtained sequences were cleaned and trimmed using BioEdit software. Then these sequences were submitted to NCBI- GenBank for registration. The phylogenetic tree was constructed using MEGA software using the Maximum-Likelihood Method and the Tamura 3-parameter model for comparing the obtained sequences with the available corresponding nucleotide sequences.

*Statistical analysis*

SPSS version 25 software was used for statistical analysis of the collected data. The Chi-square ($X^2$) test was used to assess how the qualitative variables differed from one another. A $p < 0.05$ is considered significant, whereas values over that are considered non-significant.

## Results

*Distribution of Pseudomonas aeruginosa in clinical burn specimens*

The distribution of *P. aeruginosa* in burn clinical specimens is shown in Table 4. According to the study results, *P. aeruginosa* was found to be detected in 37.5% (45/120) of the patient specimens. As regards to gender, the rate of isolates from females was significantly higher ($p = 0.028$) in comparison to males (45.7% vs 26%), as shown in Table 5. Ages between 20-30 years showed the highest rate of *P. aeruginosa* isolates, accounting for 55%, while the lowest rate (12.5%) was among ages above 1-10 years, with highly significant differences ($p = 0.001$) among them (Table 5).

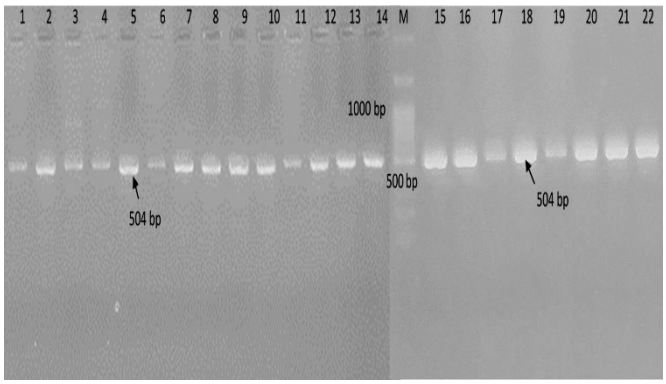
*Molecular detection of Pseudomonas aeruginosa virulence genes by using species-specific primers*

The virulence genes *Exo S* and *Las B*, which cause tissue damage, are found in the genome of *P. aeruginosa*. A standard PCR method and Agarose gel electrophoresis were used to assess the frequency of these two virulence genes among *P. aeruginosa* isolates. The PCR results of the *Exo S* gene showed a band size of 504 bp, which was detected in all samples, as demonstrated in Figure 1. The produced *Exo S* gene segments were sent to South Korea for sequencing; the

**Table 6.** Accession numbers of *Pseudomonas aeruginosa* isolates carrying *Exo S* genes and rate of similarity with other references.

| Species | Accession number | Strain | Region | % Similarities |
|---|---|---|---|---|
| *Pseudomonas aeruginosa* | PV600284 | SAMS Kurd | Iraq | |
| *Pseudomonas aeruginosa* | PV600285 | SAMS Kurd | Iraq | |
| *Pseudomonas aeruginosa* | CP106743 | PALA15 | United Kingdom | 100 |
| *Pseudomonas aeruginosa* | CP176783 | J215 | USA | 100 |
| *Pseudomonas aeruginosa* | CP115257 | F029 | Denmark | 100 |
| *Pseudomonas aeruginosa* | CP147542 | GN06816 | USA | 100 |
| *Pseudomonas aeruginosa* | CP104783 | AUS-2 | Australia | 100 |
| *Pseudomonas aeruginosa* | CP050322 | DVT729 | USA | 100 |
| *Pseudomonas aeruginosa* | CP143899 | PA26 | United Kingdom | 100 |
| *Pseudomonas aeruginosa* | LR130530 | paerg003 | Switzerland | 100 |
| *Pseudomonas aeruginosa* | CP115232 | F050 | Denmark | 100 |
| *Pseudomonas aeruginosa* | LR134309 | NCTC12903 | United Kingdom | 100 |

**Figure 1.** The Polymerase chain reaction amplification of the *Exo S* gene with a molecular weight of (504 bps) for detection of *P. aeruginosa* by using 1.5 % agarose gel electrophoresis with (6 µl) of Prime Red-Safe dye. Lane (M) marker of DNA with a molecular weight of (100 bps), lanes (1-22) showed the specimen's bands.




obtained sequences were submitted to GenBank and given accession codes (PV600284, PV600285). Table 6 shows how these sequences displayed similar homologies with other sequences that have been recorded in the GenBank. Phylogenetic investigation based on *Exo S* gene showed that *P. aeruginosa* isolates from human infected burns (PV600284 and PV600285) form clade with other sequences from Denmark (CP115232), United Kingdom (LR134309), Switzerland (LR130530), and USA (CP050322), with a bootstrap 63% as shown in Figure 2.

*Las B,* the second virulence gene, this gene was amplified in 95.5% of the tested specimens using species-specific primer and produced band sizes of 300 bps as shown in Figure 3. The produced segments were sent for sequencing, and the received sequences were recorded in GenBank under accession numbers (PQ963870, PQ963871). As seen in Table 7 and Figure 4. These sequences displayed comparable homologies

**Figure 3.** The Polymerase chain reaction amplification of the *Las B* gene with a molecular weight of (300 bps) for detection of *P. aeruginosa* by using 1.5 % agarose gel electrophoresis with (6 µl) of Prime in Red-Safe dye. Lane (M) marker of DNA with a molecular weight of (100 bps), lanes (1-22 except 19) showed the specimen's bands.

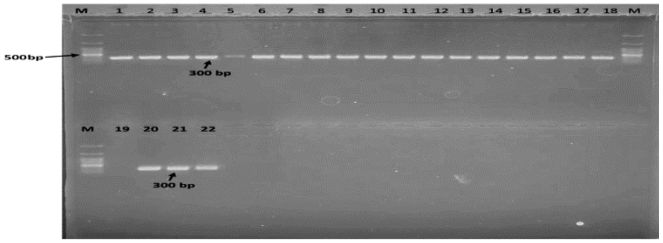
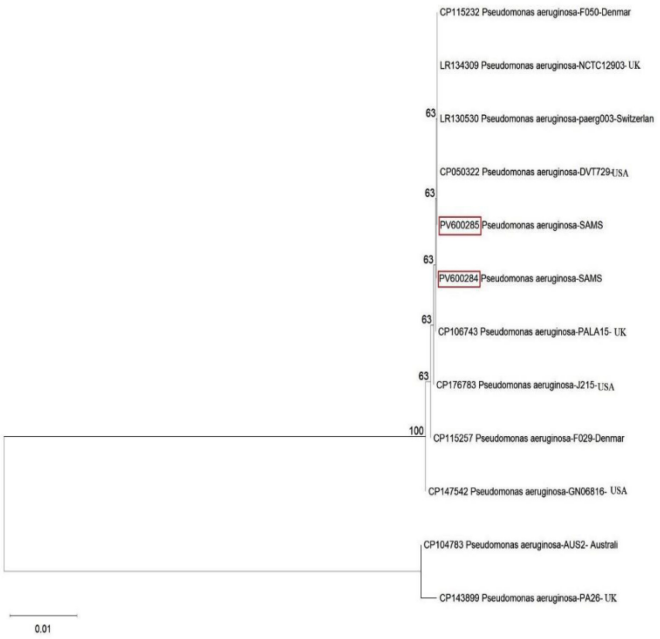



**Figure 2.** Phylogenic relationships among *Pseudomonas aeruginosa* carrying *(Exo S*) gene. The ClustalW method, which is associated with nucleotide substitution models (Tamura 3-parameter), was used to align the Maximum Likelihood tree. by using megax version 11 with a bootstrap setting of 1000 replicates the tree was created. The nodes' numbers are bootstrapped values. The sequences were generated and indicated as red rectangle in this investigation.




with other sequences that were deposited in the GenBank.

*The Distribution of Exo S and Las B Virulence Genes in Patients of Variable Ages and Genders*

The distribution of the virulence genes among patients of both genders and all ages is displayed in Table 8. The *Exo S*, was found in all specimens 22/22, while *Las B*, was found in 21/22 of *P. aeruginosa* isolates. As illustrated in the table, females had a higher non-significant ($p$ = 0.546) rate of these genes than males (54.5 vs. 45.5%) for *Exo* and (50 vs. 45.5%) for *Las B*. As regards to age, the highest rate for *Exo S* and *Las B* was 45.5% detected among ages > 20-30 years. While the lowest rate (13.6%) for *Exo S* and *Las B* was observed among ages > 30-40 years, the relationships between age and the rate of detection of these genes were statistically non-significant ($p$ = 0.071).

## Discussion

Infections in burn patients may occur due to a number of factors, including immunocompromised individuals, skin rash, invasive hospital surgeries, as well as long hospital stays [4]. According to the study's

**Table 7.** Accession numbers of *Pseudomonas aeruginosa* isolates carrying *Las B* genes and rate of similarity with other references.

| Species | Accession number | Strain | Region | % Similarities |
|---|---|---|---|---|
| *Pseudomonas aeruginosa* | PQ963870 | SAMS Kurd | Iraq | |
| *Pseudomonas aeruginosa* | PQ963871 | SAMS Kurd | Iraq | |
| *Pseudomonas aeruginosa* | LR657304 | PAK | UK | 100 |
| *Pseudomonas aeruginosa* | CP143899 | PA26 | UK | 100 |
| *Pseudomonas aeruginosa* | LR130530 | paerg003 | Switzerland | 100 |
| *Pseudomonas aeruginosa* | CP100761 | PA0011 | Ghana | 100 |
| *Pseudomonas aeruginosa* | CP096665 | PAO1 | USA | 100 |
| *Pseudomonas aeruginosa* | CP093013 | H19 | Germany | 100 |
| *Pseudomonas aeruginosa* | CP181746 | A24357 | Germany | 100 |
| *Pseudomonas aeruginosa* | CP075826 | PaLo29 | Belgium | 100 |

results, *P. aeruginosa* was found to be detected in 37.5% of the patient specimens. While in a prior study in the same region, a higher rate of 64.6% was isolated from burn patients [14]. Previous studies from Iraq and other nations reported various rates of *P. aeruginosa* isolates, such as: In Baghdad, a study revealed that the isolated rate of infection was 36% which is slightly lower than that of the current study [15]. While in Basra, a higher rate of 50.63% was isolated [16]. In both Saudi Arabia and Egypt, rates of 30% and 32.8% [17]. Variations in the incidence of *P. aeruginosa* across studies could be due to variations in sanitary methods, geographic areas, and the type of clinical specimens used [18]. As regards to gender, the rate of isolates from females was significantly higher in comparison to males (45.7% vs 26%), as shown in Table 5. A study conducted in Duhok observed that the highest rate of isolation from females in the same location was 38.18%, compared to 26.36% for males [14]. On the other hand, another study carried out in the same hospital reported higher isolate rate from males than females (33.3 vs 41.8) [19]. This variation in the rates of *P. aeruginosa* isolates among specimens from both genders may be due to the patient's hygiene application, residency, sample type and quantity, technique of collection and season of sample collection [20]. Usually, females are more prone to burn injury than males due to their home duties [21].

Ages between 20-30 years showed the highest rate of *P. aeruginosa* isolates, accounting for 55%, while the lowest rate (12.5%) was among ages above 1-10 years, with highly significant differences among them as shown in Table 5. Previous studies reported varied rates among different ages. In Kirkuk, the highest prevalence was 45% among ages 15 to 30 [18]. While Jeschke *et al*. (2020) reported the highest rate among ages of 20-29 years [22].

The *Exo S* gene PCR result showed a band size of 504 bp, which was detected in all samples, as demonstrated in Figure 1. This gene is a cytotoxin that damages various types of host cell, and is known as exoenzyme S *Exo S*, with a molecular weight of 504 bp, and is essential in the acute period of the infection [23].

**Figure 4.** Phylogenic relationships among *Pseudomonas aeruginosa* carrying *Las B* gene. The Muscle method, which is associated with nucleotide substitution models (Tamura 3-parameter), was used to align the Maximum Likelihood tree using mega x version 11 with a bootstrap setting of 1000 replicates the tree was created. The nodes' numbers are bootstrapped values. The sequences were generated and indicated as red rectangle in this investigation.

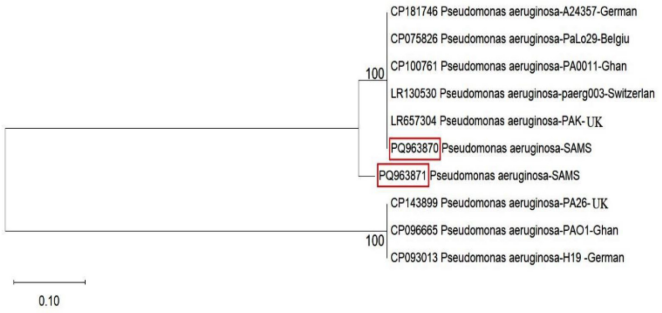



**Table 8.** The correlation between *P. aeruginosa* isolation rates and numbers among varying ages and genders.

| Variables | No. and rate of positive *Exo S* genes among (22) specimens. | No. and rate of positive *Las B* genes among (22) specimens. | *p* |
|---|---|---|---|
| **Gender** | | | |
| Males | 10 (45.5%) | 10 (45.5%) | $p = 0.546$; $\chi^2 = 0.364$ |
| Females | 12 (54.5%) | 11 (50%) | |
| **Age (years)** | **No. and rate of positive *Exo S* genes among 100 % specimens.** | **No. and rate of positive *Las B* genes among 95.5 % specimens.** | |
| < 1-10 | 4 (18.1%) | 4 (18.1) | $p = 0.071$; $\chi^2 = 7.030$ |
| > 10-20 | 5 (22.7%) | 5 (22.7) | |
| > 20-30 | 10 (45.5% | 10 (45.5) | |
| > 30-40 | 3 (13.6%) | 2 (9.1) | |
| Total | 22 (100%) | 21 (95.5%) | |

The same primers used in the current study were also employed by another study to detect this gene among *P. aeruginosa* isolates, with a success rate of 86% [24]. *Exo S* gene segments, the obtained sequences were submitted to GenBank and given accession codes (PV600284, PV600285). Table 6 shows that these sequences displayed similar homologies with other sequences recorded in GenBank.

Phylogenetic investigation based on *Exo S* gene showed that *P. aeruginosa* isolates from human (PV600284 and PV600285) form clade with other sequences from Denmark (CP115232), United Kingdom (LR134309), Switzerland (LR130530), and USA (CP050322) with bootstrap 63% as shown in Figure 2. *Las B,* the second virulence gene, degrades tissue in respond to host defenses [25]. Elastase breaks down the arteries' and arterioles' elastic lamina [26]. This gene was amplified in 95.5% of the tested specimens using species specific primer and produced band sizes each of 300 bps. The produced segments were sent for sequencing, and the received sequences were recorded in GenBank under accession numbers (PQ963870, PQ963871). These sequences displayed comparable homologies with other sequences that were deposited in the GenBank. Another study in Duhok also, recorded this gene within 50% of her specimens [14], while other study estimated the *las B* gene at 82% among tested specimens [24]. Phylogenetic investigation based on *Las B* gene showed that *P. aeruginosa* isolates from human (PQ963870 and PQ963871) are sister group and form a well-supported clade with other sequences from UK (LR65704), Switzerland (LR130530), Ghana (CP100761), Belgium (CP075826), and Germany (CP181746), with 100% of the bootstraps.

The distribution of the virulence genes among patients of both genders and all ages is displayed in Table 8. As illustrated in the table, females had a higher non-significant rate of these genes than males for *Exo s* and *Las B*. As regards age, the highest rates for *Exo S* and *Las B* were detected among ages > 20-30 years. While the lowest rate for *Exo S* and *Las B* was observed among ages > 30-40 years, the relationships between age and the rate of detection of these genes were statistically non–significant. The elevated frequency of virulence genes among the *P. aeruginosa* isolates from infected burn specimens of patients who were challenging treatment due to high inactivity of medication used for treatment might be attributed to the misuse or overuse of antibiotics, as clearly explained by this study. This condition necessitates the introduction of strict preventive measures and the introduction of health education programs that warn the community about the dangers of the misuse and overuse of antibiotics.

The underlying rationale of this investigation is grounded in the clinical and epidemiological importance of *Pseudomonas aeruginosa* as a predominant pathogen in burn patients. The impressed scientific impetus for targeting *ExoS* and *LasB* genes relies in their role as major virulence determinants [16]. *ExoS* encodes an exoenzyme that disrupts host cell signaling and promotes cytotoxicity, while *LasB* encodes elastase, a protease that degrades host tissues and immune components. Together, these factors contribute to tissue destruction, impaired wound healing, and progression to sepsis, which remains a leading cause of mortality in severe burn cases. Detecting these genes therefore provides insight into the pathogenic potential of circulating *P. aeruginosa* strains and their capacity to cause invasive infections of resistant isolates [1].

Clinically, the identification of *ExoS* and *LasB* confers tangible benefits by enabling early recognition of high-risk infections and facilitating the design of targeted antimicrobial or adjunctive therapies. For affected patients, this translates into reduced complication rates, improved treatment outcomes, shorter hospital stays, and lower mortality. At the community level, molecular surveillance of these virulence genes supports infection-control strategies in hospitals, strengthens antibiotic stewardship by preventing indiscriminate drug use, and contributes to regional and global genomic databases that monitor the evolution and spread of pathogenic strains. Consequently, this detection not only improves patient care but also mitigates the broader public health threat posed by multidrug-resistant *P. aeruginosa* [14].

The inclusion of the phylogenetic tree is justified as it provides a comparative framework for situating the *ExoS* and *LasB* gene sequences obtained from local *Pseudomonas aeruginosa* isolates within the global genetic landscape. By illustrating evolutionary relationships with strains from other countries, the tree validates the molecular identity of the isolates, confirms sequence homology, and highlights possible routes of genetic similarity or divergence [13,20].

This is directly relevant to the study's objectives, as it not only establishes the presence of virulence genes in burn-associated isolates but also demonstrates their phylogenetic relatedness to internationally reported strains. Such analysis strengthens the epidemiological significance of the findings, enhances understanding of the genetic diversity and potential origins of these

virulent isolates, and underscores the public health importance of monitoring pathogen evolution in clinical settings [20].

## Conclusions

This study highlights the importance of detecting ExoS and LasB virulence genes in *Pseudomonas aeruginosa* isolates from burn patients. The findings suggest the contribution of these genes to the bacteria's pathogenic potential and global dissemination. Early detection can guide treatment and improve patient outcomes. The study emphasizes the need for molecular diagnostics in clinical practice to control infections and promote antibiotic stewardship.

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The author thanks the Polytechnic University of Duhok for providing some of the research facilities. Thanks are also due to the Burn Hospital for allowing me to collect patient specimens and take their data; without this help, the work would not have been achieved.

### Ethical Consideration

This study was performed according to the ethical standards of Helsinki declaration, and an ethical approval was taken before carrying the study from Duhok General Directorate of Health (27112024-10-10) for taking patient samples their consent and related information.

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### Conflict of interest

No conflict of interest is declared.

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