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Hawraa Ibrahim Abdul Kareem
Department of Clinical Laboratories, College of Applied Medical Sciences, University of Kerbala, Iraq

Zahraa Hussien Ali Alawadi
Department of Clinical Laboratories, College of Applied Medical Sciences, University of Kerbala, Iraq

Ayat Mohammed Obaid Alzumaili
Department of Clinical Laboratories, College of Applied Medical Sciences, University of Kerbala, Iraq

Suhad Hadi Mohammed
Department of Clinical Laboratories, College of Applied Medical Sciences, University of Kerbala, Iraq

Aia Haider Qahtan Al-Saowdy
Department of Clinical Laboratories, College of Applied Medical Sciences, University of Kerbala, Iraq

Huda Khalid Ghazi
Department of Clinical Laboratories, College of Applied Medical Sciences, University of Kerbala, Iraq

Fatma Mukhtar Ismail
Department of Clinical Laboratories, College of Applied Medical Sciences, University of Kerbala, Iraq

Rosul Harbi Ghafel
Department of Clinical Laboratories, College of Applied Medical Sciences, University of Kerbala, Iraq

Corresponding Author:
Hawraa Ibrahim Abdul Kareem
Department of Clinical Laboratories, College of Applied Medical Sciences, University of Kerbala, Iraq

In vitro antibacterial and antibiofilm activity of prednicort and dexamethasone against clinical bacterial isolates, a pilot study

Hawraa Ibrahim Abdul Kareem, Zahraa Hussien Ali Alawad, Ayat Mohammed Obaid Alzumaili, Suhad Hadi Mohammed, Aia Haider Qahtan Al-Saowdy, Huda Khalid Ghazi, Fatma Mukhtar Ismail and Rosul Harbi Ghafel

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Abstract
Background: Preliminary research suggests that steroids may improve direct antimicrobial effects or enhance the efficiency of conventional antibiotics.
Aim of the study: The present study aims to evaluate the antibacterial and antibiofilm actions of some steroids (Prednicort and Dexamethasone) on clinical bacterial isolates.
Material and Method: A total of 17 isolated bacteria were obtained from clinical specimens between February to April 2023. The antibacterial and Antibiofilm activity of Dexamethasone and Prednicort was measured by using a microtiter plate assay.
Results: Both drugs exhibited notable antibacterial activity against Staphylococcus isolates. Whereas their effects were less pronounced against *Escherichia coli* and Pseudomonas spp. Moreover, pre-incubation of bacterial isolates with the drugs resulted in a significant reduction in their biofilm-forming ability. However, when both drugs were applied after a 24-hour incubation period (eradication phase), their impact on disrupting established biofilms was considerably diminished.
Conclusion: Dexamethasone and prednisolone had an antibacterial effect, and the effect of prednisolone as an antibacterial was more and better than Dexamethasone. Furthermore, the antimicrobial efficacy was better on Staphylococcus aureus than on Pseudomonas spp and *Escherichia coli*.

Keywords: Antibacterial, Antibiofilm, Prednicort, Dexamethasone, *Staphylococcus aureus*, *Escherichia coli*, *Pseudomonas*

Introduction
Rising antibiotic resistance among microorganisms represents a major global public health concern, potentially signaling a regression toward a pre-antibiotic era. Furthermore, the current sluggish rate of antibiotic discovery may result in a shortage of effective therapeutic options for managing bacterial infections caused by resistant microbes in the near future [1].
Multi drug resistant bacterium (MDR) is described as non-susceptibility to at least a single chemical from three or more antimicrobial groups [2]. MDR bacteria are primarily found in hospitals and frequently harm individuals who are older or very ill. Common MDR organisms include; MRSA (Methicillin-resistant Staphylococcus aureus) Streptococcus pneumoniae and Vancomycin-resistant Staphylococcus aureus (VRSA) [3].
There is a critical need to develop alternative therapeutic strategies to effectively manage infections caused by antibiotic-resistant bacteria. The steroids are natural compounds, a collection of compounds of both plant and animal origin with a distinctive tetracyclic backbone, which serve many roles and functions in multicellular organisms [4, 5].
Steroid hormones and their derivatives constitute one of the most extensively utilized categories of therapeutic compounds. These medications are generally used for birth control, hormone replacement therapy (HRT), inflammatory diseases, and cancer treatment. The majority of these agents are chemically similar and share a structural backbone. Although they share a basic structural organization, differences in the structures give selectivity for the individual [6].

Naturally occurring steroid hormones are normally categorized into five main groups, including androgen (testosterone), progestin (progesterone), estrogen (estradiol), cortisol/corticosterone (glucocorticoid), and aldosterone (mineralocorticoid). The structural basis for the diversity in actions and the varied therapeutic applications of these substances are investigated [7]. While the synthetic steroids are mostly utilized for therapeutic purposes, such as anti-inflammatory or immunosuppressive. The effects of corticosteroids have generally been classified as physiological or pharmacological. Physiological actions were assumed to occur at regular daily production levels, but the pharmacologic effects were viewed as occurring at levels higher than usual. Chemical modifications to the cortisol molecule have led to the development of synthetic corticosteroids with selective glucocorticoid or mineralocorticoid activity [8].

Among the commonly used synthetic corticosteroids are prednisolone and dexamethasone. Prednisolone is commonly prescribed for the management of various inflammatory and autoimmune diseases, including ulcerative colitis, Crohn's disease, chronic obstructive pulmonary disease, rheumatoid arthritis, and severe dermatological conditions. Dexamethasone, with its strong glucocorticoid and minimal mineralocorticoid activity, is used in managing congenital adrenal hyperplasia, allergic and inflammatory disorders, and for the diagnosis of Cushing's syndrome [9].

Clinical observations have shown that patients undergoing long-term steroid therapy for conditions like eczema, psoriasis, and asthma often present with alterations or reductions in beneficial bacteria (microbial flora) and an increase in potentially harmful bacteria such as Staphylococcus aureus. These findings raise the possibility that synthetic corticosteroids may exhibit direct or indirect antibacterial effects. Consequently, the primary aim of the present study is to assess the antibacterial activity of synthetic corticosteroids (dexamethasone and prednisolone) against selected bacterial strains.

Materials and Methods

Study design and setting

Seventeen bacterial isolates were obtained from diabetic foot swabs collected from patients admitted to Al-Kafeel Hospital. These isolates were subsequently re-identified at the Microbiology Laboratory, Department of Clinical Laboratories, College of Applied Medical Sciences, University of Kerbala over the period of 4 months between February to April 2023.

Bacterial Identification

Reactivation Step

The bacterial isolates were cultured and reactivated using blood agar and MacConkey agar (both manufactured by HIMEDIA, India). Following 24 hours of incubation, the morphological characteristics of the colonies were observed and documented. Observations included colony size, shape, color, and hemolytic activity on blood agar. On MacConkey agar, colony color was assessed to differentiate between lactose-fermenting bacteria, which produced pink colonies, and non-lactose-fermenting bacteria, which formed colorless colonies.

The diagnostic and biochemical tests

Classification of Bacteria according to Gram Reaction

Classification of bacterial gram reaction was done using gram staining for a group of samples to see if they were gram positive or gram negative. The procedure involves four fundamental steps: application of the primary stain (crystal violet), treatment with a mordant (Gram's iodine), rapid decolorization using ethanol, acetone, or a combination of the two, followed by counterstaining with safranin.

Identification and antibiotic susceptibility tests by using the Vitek 2 Compact system

Both bacterial identification and the susceptibility of all isolates to standard antibiotics were evaluated using the VITEK 2 automated system (BioMérieux).

Principle

The VITEK 2 Compact System is an advanced identification tool developed for identifying bacteria and yeast. It utilizes VITEK 2 cards, which contain 64 wells filled with various nutrients and biochemical tests to facilitate microbial identification. A standardized suspension of the microorganism is prepared and used to inoculate the VITEK 2 card. As the organism interacts with the biochemical substrates within the card, a characteristic pattern of positive and negative reactions is generated.

Procedure*:*

1. **Restoration of Preserved Isolates**
   Frozen bacterial isolates maintained in preserved media (Brain Heart Infusion broth) containing glycerol at -20 °C were retrieved and allowed to thaw gradually at room temperature*.*
2. **Primary Culturing on Solid Media**
   Using a sterile disposable inoculating loop, a small volume of the thawed suspension was streaked onto appropriate agar plates (e.g., Blood Agar, MacConkey Agar) depending on the expected bacterial type. The incubation of the plates was at 37°C for 24 hours to ensure the growing of isolated colonies and to confirm purity*.*
3. **Reparation of the Inoculum**
   From the freshly incubated agar plates, several well-isolated colonies were selected using a sterile cotton swab. These were emulsified into 3 mL of sterile normal saline in a clean test tube to form a homogeneous bacterial suspension*.*
4. **Turbidity Adjustment (McFarland Standard)**
   The bacterial sample suspension was standardized to the McFarland turbidity level specified for the selected VITEK 2 ID or AST card. A DensiCHEK Plus turbidity meter was used to ensure accurate calibration of the bacterial concentration*.*
5. **Card Selection and Inoculation**
   Based on the preliminary identification or suspected bacterial group, the appropriate VITEK 2 ID and AST cards were selected, and prior to use, the cards were brought to room temperature before being opened. The standardized bacterial suspension was then loaded into the card by inserting the card into the test tube containing the suspension, which was then placed into the cassette card holder*.*
6. **Automated Filling of ID and AST Cards**
   The loaded cassette was inserted into the filling chamber (left side) of the VITEK 2 instrument. The "Start Fill" command was initiated, prompting the system to automatically fill all inserted cards within

approximately 70 seconds. The system emitted an audible alert upon successful completion of the filling cycle.

7. **Barcode Scanning and Card Loading**
   Upon completion of the filling step, the load compartment door was unlocked automatically. The cassette was transferred into the loading chamber, where the barcodes were scanned to ensure accurate sample registration within the virtual cassette. Following confirmation, the cards were sealed, straws were trimmed.
8. After successful card loading, used tubes and straws were discarded into a designated biohazard waste container in compliance with laboratory safety protocols.
9. Identification results were automatically transmitted to the designated "Results View" folder on the system. The data were reviewed and printed by the analyst for further interpretation and documentation.

Bacterial Preservation

The isolated bacterial strains were preserved in Brain Heart Infusion (BHI) broth (HiMedia, India) supplemented with 15% (v/v) glycerol. The suspensions were transferred into sterile Eppendorf tubes and stored at -20 °C until further analysis.

Antibacterial and anti-biofilm effects (both anti-biofilm and eradication assay)

1. Activation of bacterial isolates was done by using nutrient agar plates. Activation was done by aseptically transferring one or two colonies and uniformly distributing over the agar surface. The plates were subsequently incubated at 37 °C for 24 h to permit bacterial growth.
2. To prepare a 0.5 McFarland bacterial suspension, 100 mL of sterile distilled water was added to plain test tubes, followed by the addition of a bacterial colony. The suspension was then vortexed for 30 seconds to achieve uniform turbidity. Turbidity was measured using a microtiter plate and an ELISA reader (at wavelength 630nm).
3. A 96-well microtiter plate was obtained. 100 µl of McFarland suspension and 50 µl of Mueller-Hinton broth were added. The first two wells from each column were considered as controls.
4. After the McFarland suspension and Mueller-Hinton agar were added, drug treatments were introduced. Fifty microliters of Prednicort (drug A) and Dexamethasone (drug B) were added to each isolate in separate wells except for control wells.
5. 5-The incubation of the microtiter plate was performed at 37 °C for 24 hours
6. On the following day, the plate was read using an ELISA reader at a wavelength 630 nm.
7. For antibiofilm activity, all contents of the wells were withdrawn from the wells.
8. The wells were subsequently stained with crystal violet by adding 100 µL of the dye to each well and allowing it to incubate for 15 minutes. Then the wells were carefully washed using sterile distilled water. After washing, 100 µl of ethanol was dispensed into each well, and the optical density was recorded using the ELISA reader.
9. For eradication assay, bacterial suspension was incubated with Muller Hinton broth (in a microtiter plate and incubated for 24h in 37c and after that the bacterial suspension with broth was removed and 100 ml of drug A and B were added to each well except for control wells. The plate was incubated for 24h in 37c again. After that, the plate was stained with crystal violet as mentioned in step 8.

Ethical consideration

This study had been approved by the committee of College of Applied Medical Science, Department of Clinical Laboratories and the research was done in the microbiology lab, University of Kerbala (Certificate Number 259 on September 24, 2023). Each bacterial isolates utilized in this study were collected following informed consent from the participating patients.

Statistical analysis

The Statistical Package for Social Sciences (SPSS) version 22 program (IBM Corp., NY, USA) was utilized to conduct all statistical analyses. Descriptive statistics were utilized to determine frequencies, the mean, standard deviation. A $p<0.05$ was considered statistically significant.

Results

In this research, 20% of Staphylococcus isolates (both COP and CON groups) exhibited resistance to gentamicin and vancomycin. Additionally, resistance was observed in 70% of isolates to erythromycin, tetracycline, and fusidic acid. Resistance rates to rifampicin, clindamycin, oxacillin, and teicoplanin were 40%, 60%, 80%, and 11.1%, respectively. Additionally, 30% of the strains were resistant to both moxifloxacin and trimethoprim/sulfamethoxazole. Notably, all Staphylococcus isolates (100%) were resistant to benzylpenicillin, whereas complete susceptibility (100%) was recorded for tigecycline and linezolid, as shown in Table 1.

All of the *Escherichia coli* (100%) exhibited resistance to trimethoprim/sulfamethoxazole, ceftazidime, cefepime, levofloxacin, ticarcillin, piperacillin, and aztreonam. Only 25% were resistant to gentamicin. In contrast, all isolates were sensitive to ciprofloxacin, tobramycin, imipenem, Piperacillin tazobactam, ticarcillin clavulanic acid, meropenem, and amikacin, as illustrated in Table 2.

All of the Pseudomonas were resistant to cefazolin, and all of them were sensitive to gentamicin, trimethoprim/sulfamethoxazole, ciprofloxacin, ceftazidime, cefepime, tobramycin, levofloxacin, imipenem, piperacillin tazobactam, meropenem, and amikacin, as shown in Table 3.

Table 1: Results of Antibacterial Susceptibility of Staphylococcus Species Using VITEK 2 Compact system.

Bacterial group	Gentamicin	Erythromycin	Vancomycin	Tetracycline	Tigecycline	Rifampicin	Trimethoprim sulfamethxazole	Benzylpenicillin	Oxacillin	Moxifloxacin	Clindamycin	Linezolid	Fusidic acid	Teicoplanin
Staphylococcus COP	S	S	R	R	S	S	S	R	S	S	S	S	R	S
Staphylococcus COP	R	R	S	R	S	S	S	R	R	I	R	S	R	S
Staphylococcus COP	S	R	S	R	S	R	R	R	S	R	R	S	S	S
Staphylococcus COP	S	S	S	S	S	S	S	R	R	S	S	S	S	S
Staphylococcus COP	S	R	S	S	S	S	S	R	R	S	R	S	S	S
Staphylococcus CON	S	R	S	R	S	S	S	R	R	I	S	S	R	S
Staphylococcus CON	S	R	S	R	S	R	S	R	R	R	R	S	R	-
Staphylococcus CON	S	R	R	R	S	R	R	R	R	I	R	-	R	R
Staphylococcus CON	R	R	S	R	S	R	R	R	R	R	R	S	R	S
Staphylococcus CON	S	S	S	S	S	S	S	R	R	S	S	S	R	S
Total Percentage of Resistance	20%	70%	20%	70%	0%	40%	30%	100%	80%	30%	60%	0%	70%	11.1%

COP, coagulase positive; CON, coagulase negative; R, resistance; S, sensitive; I, intermediate

Table 2: Results of Antibacterial susceptibility of *Escherichia coli* using VITEK 2 Compact system.

Bacterial group	Gentamicin	Trimethoprim sulfamethxazole	Ciprofloxacin	Ceftazidime	Cefepime	Tobramycin	Levofloxacin	Imipenem	Piperacillin tazobactam	Ticarcillin	Ticarcillin clavulanic acid	Piperacillin	Aztreonam	Meropenem	Amikacin
Escherichia coli	S	R	S	R	R	I	-	S	S	R	S	R	R	S	S
Escherichia coli	R	R	-	R	R	I	R	-	I	R	-	-	R	S	S
Escherichia coli	S	R	S	R	R	S	-	S	S	R	S	R	R	S	S
Escherichia coli	S	R	-	R	R	S	R	-	S	R	I	R	R	S	S
Total Percentage of Resistance	25%	100%	0%	100%	100%	0%	100%	0%	0%	100%	0%	100%	100%	0%	0%

R, resistance; S, sensitive; I, intermediate

Table 3: Results of Antibacterial susceptibility of *Pseudomonas spp* using VITEK 2 Compact system.

Bacterial group	Gentamicin	Trimethoprim sulfamethxazole	Ciprofloxacin	Ceftazidime	Cefepime	Tobramycin	Levofloxacin	Cefazolin	Imipenem	Piperacillin tazobactam	Meropenem	Amikacin
Pseudomonas spp	S	S	S	S	S	S	S	R	S	S	-	-
Pseudomonas spp	S	-	-	S	S	S	S	R	-	S	S	S
Pseudomonas spp	S	-	S	I	I	S	-	-	S	S	S	S
Total Percentage of Resistance	0%	0%	0%	0%	0%	0%	0%	100%	0%	0%	0%	0%

R, resistance; S, sensitive; I, intermediate

Antibacterial effect of the steroids:

In this study, the results showed that (Prednicort and Dexamethasone) had a good antibacterial effect on Staphylococcus isolates and that Dexamethasone had a better impact than Prednicort, while a lower effect of this drug was observed for *E. coli* and Pseudomonas, as shown in Figure 1.

Antibiofilm assay

This study demonstrated that incubation of bacterial isolates with the steroid resulted in a reduction of biofilm formation by the examined bacteria, as illustrated in Figure 2. However, a diminished effect was observed in the eradication assay, where the drugs were applied after 24 hrs of incubation, as established in Figure 3.

Discussion

The global escalation of bacterial resistance to antibiotics constitutes a profound threat to public health, potentially undoing the advances in treating infectious diseases that modern medicine has achieved [10]. Moreover, the current assessments reveal that the new antibiotics remain grossly insufficient relative to mounting resistance [11].

The drug resistance of Gram-positive organisms has received considerable attention [12]. Additionally, Gram-negative bacteria may pose a similar threat because of their membrane structure. Gram-negative bacteria are inherently resistant to many types of antibiotics [13].

The current study revealed a high resistance rate of Staphylococcus isolates (COP and CON) to Benzylpenicillin (100%), erythromycin (70%), tetracycline (70%), fusidic acid (70%), and oxacillin (80%), whereas all of them were sensitive to tigecycline and linezolid (100%), as shown in Table 1.

Previous study documented that resistance rates among S. aureus isolates were 100%, 78 % and 76 % to benzylpenicillin, erythromycin, and oxacillin, respectively [14]. Additionally, Christina *et al*., (2017) reported a high resistance rate of S. aureus to benzylpenicillin (71.9%) [15]. Similarly, finding was reviewed by Baek *et al*., (2016), which documented the higher resistance of S. aureus isolates to Benzylpenicillin, erythromycin, tetracycline, fusidic acid, and oxacillin, and 0.1% of isolates were resistant to linezolid [16].

Another study reported that resistance to new agents (like linezolid, tigecycline) is low globally but not zero; for example, CoNS had 0.3% resistance to linezolid and 1.6% to tigecycline [17].

All of the *Escherichia coli* were resistant to trimethoprim/sulfamethoxazole, ceftazidime, cefepime, levofloxacin, ticarcillin, piperacillin, and aztreonam. While all of them were sensitive to ciprofloxacin, tobramycin, imipenem, Piperacillin tazobactam, ticarcillin clavulanic acid, meropenem, and amikacin, as shown in Table 2.

The resistance pattern of *E. coli* to antibiotics has been extremely different in various studies. The study documented by Naqid *et al*., (2025) found the *E. coli* isolates exhibited resistance rates, particularly against ceftazidime (67.8%), while the highest sensitivity was observed for meropenem (95.86%) and imipenem (94.92%) [18].

Polse *et al*., (2016) reviewed that all S. aureus isolates were 100% susceptible to imipenem and meropenem and 100% resistant to aztreonam. This high resistance could be due to the spontaneous and uncontrollable use of these antibiotics [19]. The carbapenems (imipenem and meropenem) are known to be stable against ESBL enzymes and effective in treating infections caused by ESBL-producing bacteria [20]. Mouhammed & Gdoura (2024) reported that 80% of *E. coli* isolates were resistant to trimethoprim/Sulfamethoxazole, while showing resistance to Levofloxacin (60%) and cefepime (72.9%) [21].

Mubita *et al*., (2021) reported comparable results in which 68% and 74% of E coli isolates were resistant to ciprofloxacin and gentamicin, respectively; however, similar results for piperacillin and tazobactam in which 7% of isolates were resistant to this drug [22]. Such high resistance rates to antibiotics in our community can be explained partially by the high rate of abuse of antibiotics in the region [23].

Additionally, comparable results were found by Al-Dulaimi, (2016) was reported that the resistance of *E. coli* was 100% to ticarcillin and piperacillin, 93.4% to ceftazidime, 95.6% to cefepime and aztreonam. However, this study also found high sensitivity to amikacin (97.8%), imipenem (95.6%), and meropenem (95.6%) [24].

All of the Pseudomonas were resistant to cefazolin, and all of them were sensitive to gentamicin, trimethoprim/sulfamethxazole, ciprofloxacin, ceftazidime, cefepime, tobramycin, levofloxacin, imipenem, piperacillin tazobactam, meropenem, and amikacin, as shown in Table 3.

Previous study found a comparable results in which the highest level of resistance was 96.25% towards Cefepime, followed by Amikacin (91.25%), Gentamicin (81.25%), Ciprofloxacin (76.25%), Ceftazidime (66.25%) and Piperacillin (48.8%), however, similar to current study, this study reported the lowest resistance was towards imipenem (3.75%) followed by meropenem (5%) [25].

Liu *et al* (2024) found that P. aeruginosa was highly sensitive to gentamicin, amikacin, and cefepime (100%), 87.5% to piperacillin/tazobactam, imipenem, ceftazidime, and ciprofloxacin, and 77.8% to Levofloxacin [26]. Also, Salman & Hussein (2023) showed that all isolates were resistant by 100% for Cefazolin [27].

The current study revealed that dexamethasone and prednisolone had an antibacterial effect, and that the effect of prednisolone as an antibacterial was more and better than dexamethasone, and its effect was better on Gram-positive bacteria such as staphylococcus aureus than Gram-negative *E. coli* and pseudomonas, Figure 1.

These findings are partly supported by recent studies that investigated the interaction between corticosteroids and antimicrobial agents, or the direct influence of steroid derivatives on bacteria. For example, Rajasekaran *et al*. (2023) reported that pharmaceutical formulations containing prednisolone enhanced the inhibition of biofilm formation and potentiated antimicrobial activity against several microorganisms [28].

Similarly, Vivas *et al*., (2020) demonstrated that certain steroid derivatives related to prednisolone possessed inhibitory effects against S. aureus planktonic cells, suggesting that prednisolone or its analogues may exert a direct or indirect antibacterial influence, particularly against Gram-positive species [29].

Biofilm formation is a key virulence mechanism in many pathogenic bacteria, including Staphylococcus aureus and *Escherichia coli* [30]. The recent study has explored the

potential antibiofilm effects of steroids, particularly corticosteroids, due to their anti-inflammatory and immunomodulatory properties.

The findings demonstrate that both tested steroids were able to inhibit biofilm formation in Staphylococcus aureus and *Escherichia coli* isolates when compared to untreated controls. Among the tested drugs, prednisolone exhibited the highest antibiofilm activity, particularly against S. aureus, followed by *E. coli*, Figure 2 and 3.

The observed difference in efficacy might be related to the structural and physiological variations between gram-positive and gram-negative bacteria. S. aureus, being a gram-positive organism, has a thick peptidoglycan layer and lacks an outer membrane, which may facilitate greater drug interaction with its biofilm-forming mechanism. In contrast, *E. coli* contains an outer membrane that may act as a barrier to steroid penetration, potentially limiting its antibiofilm [31].

Several previous studies have reported similar findings. For instance, Cherian *et al*., found that some corticosteroids were capable of directly reducing S. aureus biofilm growth *in vitro* [32]. Moreover, research by Tabatabaeifar *et al*., showed that anti-inflammatory drugs, when used alone or in combination with antibiotics, could reduce biofilm biomass in *E. coli* and other gram-negative bacteria [33].

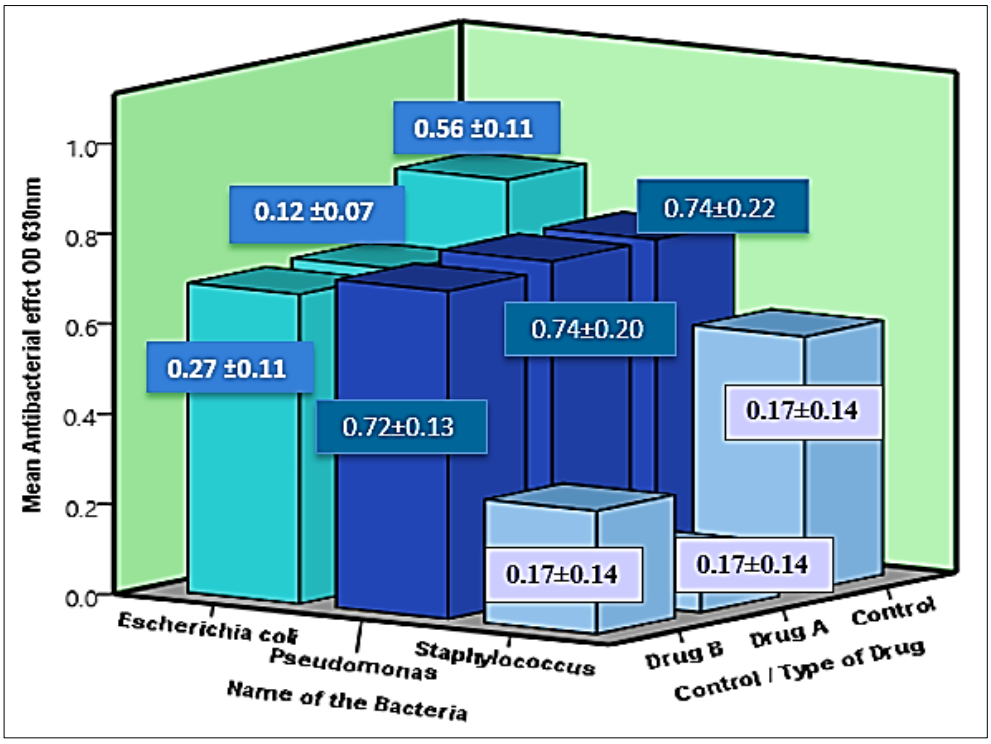

Fig 1: Antibacterial effect of steroids on bacterial isolates, (drug A, prednisolone; drug B, dexamethasone).

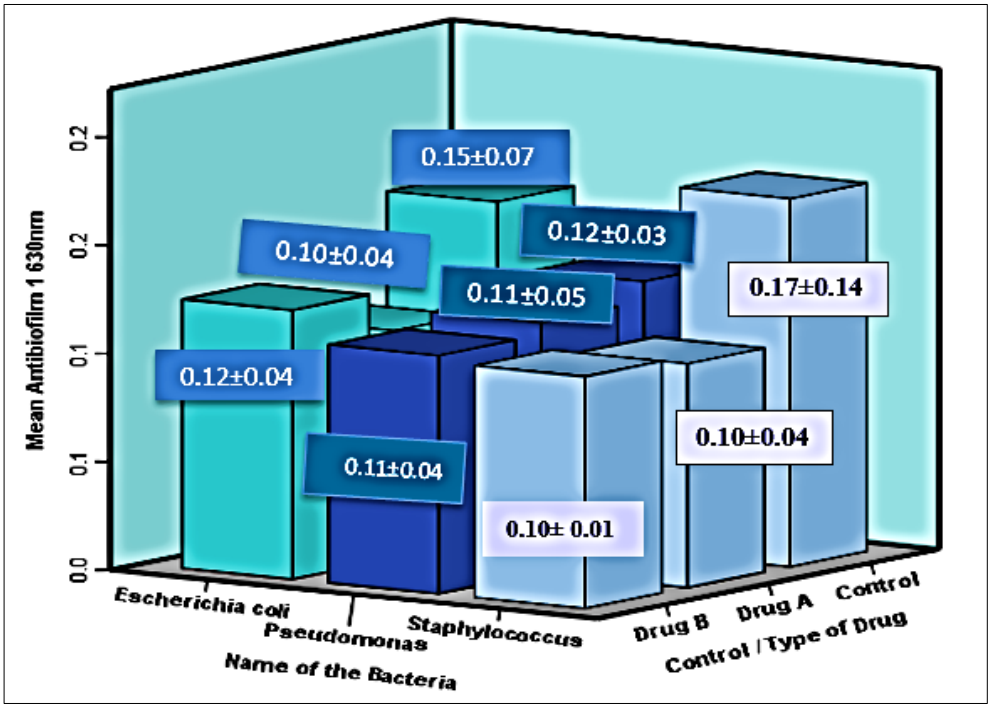

Fig 2: Antibiofilm effect of steroids on bacterial isolates, (drug A, prednisolone; drug B, dexamethasone).

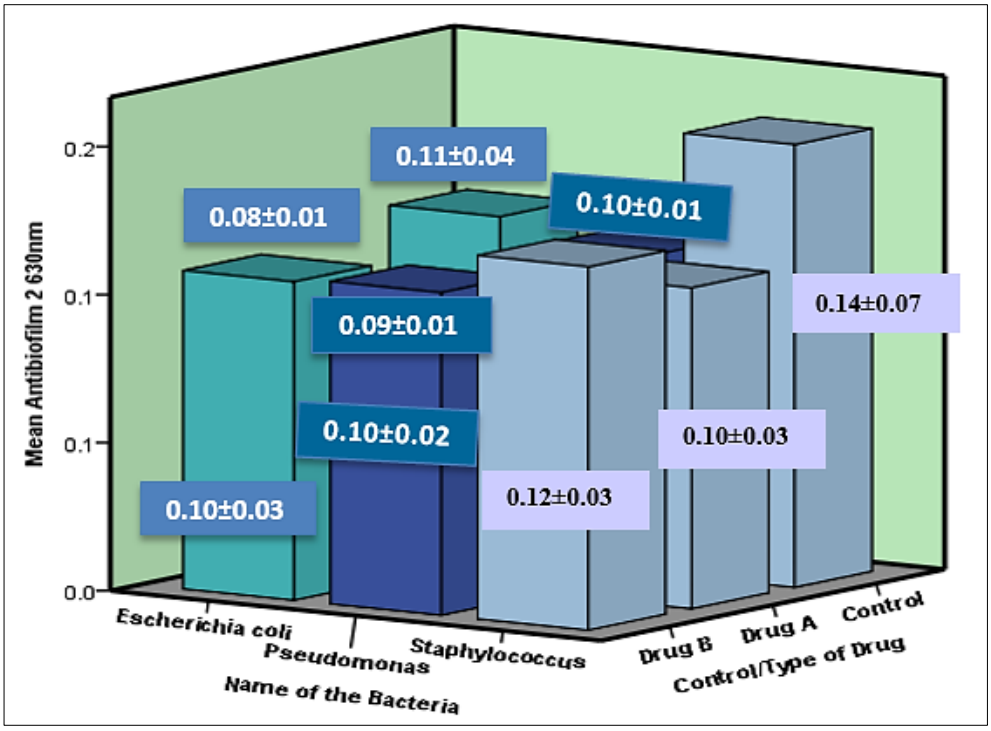

Fig 3: Antibiofilm effect of steroids on bacterial isolates, (drug A, prednisolone; drug B, dexamethasone).

Conclusion

The present study was concluded that the steroids have an antibacterial effect, and this effect on gram - positive bacteria like staphylococcus was more than that of pseudomonas and *E. coli*, also that the drug prednisolone was better than the dexamethasone by inhibiting bacterial growth. Furthermore, the steroids have little effect on preventing biofilm formation and also dismantle the formed membrane.

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Conflicts of Interest

The authors confirm that there are no conflicts of interest.

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