Basrah Journal of Veterinary Research 2026; 25(2):55-69.

BASJVETRES

Print ISSN: 1813-8497
Online ISSN: 2410-8456
https://bjvr.uobasrah.edu.iq/

The Dual Antibacterial Role of Non-Steroidal Anti-Inflammatory Drugs (NSAIDs): Intrinsic Antibacterial Activity and Modulation of Antibiotic Susceptibility in Bacteria from Otitis Media

Article Info.

Author

Zainab R. Abdul-Hussein[1], Ali T. Abdul - Samad[2], Ahmed S. Khamees[1],

1-Department of Pathological analyses, College of Science, University of Basrah, Iraq.

2-Al- Basrah Teaching Hospital, Ministry of Health, Basrah, Iraq.

Corresponding Author Email Address: Zainb.abdulhussien@uobasrah.edu.iq

Article History

Received: 12 February 2026
Revised: 8 March 2026
Accepted: 13 March 2026
e Published: 30 June 2026
Article type: **Research Article**

https://doi.org/10.23975/bjvr.2026.169390.1285

Abstract

Non-steroidal anti-inflammatory drugs (NSAIDs) are widely used for their anti-inflammatory, analgesic, and antipyretic properties. However, emerging evidence suggests that some NSAIDs also exhibit antimicrobial effects, which could be valuable in the era of increasing antibiotic resistance. This study evaluated the in vitro antibacterial activity of three commonly used NSAIDs—aspirin (acetylsalicylic acid), ibuprofen and diclofenac sodium, against pathogenic bacteria isolated from patients with otitis media, which included 96 isolates (30 Pseudomonas aeruginosa, 34 Staphylococcus aureus, 23 E. coli and 9 isolates belonged to Klebsiella pneumonia). Results showed that the diameters of inhibition zones of the 100000 µg /ml of the NSAIDs against was higher than other concentrations and that Staphylococcus aureus was the most affected bacteria towards diclofenac Sodium (34 mm). Also, results of antibiotic sensitivity of each of E. coli and Staphylococcus aureus that were exposed to NSAIDs showed an increase in the diameter of inhibition of the antibiotics used against the bacteria in comparison to control (no exposed bacteria). The present findings demonstrate that aspirin and ibuprofen exhibit significant antibacterial effects, particularly against Staphylococcus aureus, while diclofenac showed selective activity. These results suggest that certain NSAIDs may have adjunctive antimicrobial potential, warranting further investigation into their mechanisms and clinical applications.

Keywords: NSAIDs, antibacterial activity, antibiotic resistance, otitis media.

Publisher: University of Basrah,
College of Veterinary Medicine, Basrah, Iraq

Introduction

The relentless global spread of antimicrobial resistance (AMR) represents one of the most pressing public health crises of the 21st century, severely compromising our ability to treat common bacterial infections and undermining decades of medical progress (1) . The progressive depletion of effective antibacterial agents has created an urgent need for innovative therapeutic strategies, particularly approaches that can enhance the efficacy of existing antibiotics or possess intrinsic antimicrobial properties (2). In this context, drug repurposing—the identification of new therapeutic applications for existing drugs—has emerged as a promising avenue that offers several advantages over novel drug development, including established safety profiles, reduced development costs, and accelerated clinical translation (3).

Non-steroidal anti-inflammatory drugs (NSAIDs), among the most widely utilized medications worldwide for their analgesic, antipyretic, and anti-inflammatory properties, have recently attracted significant scientific interest for their potential antibacterial effects (4). Accumulating evidence suggests that several NSAIDs exhibit direct antimicrobial activity against clinically relevant pathogens and may modulate bacterial susceptibility to conventional antibiotics (5). This dual antibacterial role positions NSAIDs as promising candidates for adjunctive therapy in the management of bacterial infections, particularly in the face of escalating multidrug resistance.

The antibacterial mechanisms of NSAIDs appear to be multifaceted and structurally diverse. Proposed mechanisms include disruption of bacterial cell membrane integrity through alterations in surface physicochemical properties, inhibition of DNA synthesis via interference with topoisomerase enzymes, chelation of essential divalent cations, and suppression of virulence factor expression (6) . For instance, diclofenac has demonstrated potent activity against Staphylococcus aureus by compromising cytoplasmic membrane function, leading to increased permeability and potassium efflux (7) . Similarly, aspirin (acetylsalicylic acid) has exhibited broad-spectrum activity against both Gram-positive and Gram-negative bacteria, including Escherichia coli, Bacillus cereus, Pseudomonas aeruginosa, Enterobacter aerogenes, Klebsiella pneumoniae, and Salmonella choleraesuis with minimum inhibitory concentrations (MICs) ranging from 1.2–5 mg/L (8) .

Beyond their direct antibacterial effects, several NSAIDs demonstrate the capacity to potentiate the activity of conventional antibiotics. This modulatory function may involve the alteration of membrane permeability to enhance antibiotic penetration, interference with efflux pump systems, or suppression of biofilm formation—a key contributor to antimicrobial tolerance and persistent infections (9) . Notably, the anti-biofilm properties of NSAIDs represent a particularly promising attribute, as biofilms are implicated in approximately 80% of chronic and recurrent bacterial infections in humans and can confer dramatically increased resistance to antimicrobial agents (10)

. Studies have revealed that NSAIDs, including acetylsalicylic acid, diclofenac, and ibuprofen, possess anti-biofilm activity at concentrations achievable in human pharmacokinetic studies, potentially disrupting the extracellular polymeric substance matrix that protects microbial communities from host defenses and antimicrobial penetration (11) .

Otitis media (OM), one of the most prevalent bacterial infections in pediatric and adult populations worldwide, presents significant therapeutic challenges in the context of increasing antimicrobial resistance (12). The predominant pathogens associated with OM include Pseudomonas aeruginosa, Staphylococcus aureus, Proteus species, and Escherichia coli, with regional variations in prevalence and resistance patterns (13). Recent studies from the Middle East region have documented alarming resistance profiles among OM isolates. For instance, research from Iraq demonstrated that *P. aeruginosa* isolates from chronic suppurative otitis media exhibited significant resistance to commonly used antibiotics, with sensitivity to ciprofloxacin observed in only 81.5% of isolates (14). Similarly, a study by (15) identified S. aureus as the predominant isolate from pediatric OM cases, with concerning resistance patterns to first-line antibiotics. These findings underscore the urgent need for novel therapeutic approaches to manage OM effectively, particularly in regions with high rates of antimicrobial resistance.

The potential application of NSAIDs in OM management extends beyond their potential antibacterial properties to include their primary anti-inflammatory effects. The inflammatory response in OM contributes significantly to symptomatology and tissue damage, suggesting that NSAIDs could provide dual benefit by simultaneously addressing both the infectious and inflammatory components of the disease (16). This combined activity could be particularly valuable in the management of OM, where inflammation plays a crucial role in pathogenesis and symptom manifestation.

Despite promising in *vitro* evidence, the translation of NSAIDs' antibacterial potential into clinical practice requires careful consideration of several factors. The antibacterial activity of NSAIDs appears to be species-specific and strain-dependent, with substantial variations in efficacy across different bacterial pathogens (17). Furthermore, the concentration required for antibacterial effects often exceed typical therapeutic doses used for anti-inflammatory purposes, raising questions about achievable tissue levels and potential toxicity (18) . Additionally, there is emerging evidence that bacterial exposure to NSAIDs may be selected for resistant populations, highlighting the need for judicious application (19). Critically, most studies investigating the antibacterial properties of NSAIDs have been conducted in controlled laboratory settings using reference strains, with limited data on clinical isolates from specific geographical regions, particularly Iraq.

This study had two objectives: (1) to evaluate the antibacterial activity of aspirin, ibuprofen, and diclofenac against bacterial isolates from otitis media patients in Basra; and (2) to determine if sub-inhibitory NSAID exposure modifies the antibiotic susceptibility of these pathogens.

Materials and Methods

Study Population and Bacterial Isolates

The investigation was carried out at Basra General Hospital in Iraq. The study enrolled a total of 96 patients, ranging in age from 1 to 75 years, who presented with ear discharge attributable to otitis media (OM). After cleansing the ear with 70% alcohol, a specialist collected swab samples. The sample size of 96 was not determined by a formal power calculation but was instead contingent upon the availability and accessibility of patients throughout the duration of the study.

Identification of Bacterial Pathogens

Clinical swabs were cultured on Blood, MacConkey, and Mannitol Salt agar plates and subjected to aerobic incubation at 37°C for 24-48 hours. Preliminary identification of bacteria was conducted by assessing colony morphology, Gram stain characteristics, and standard biochemical profiles. The Vitek-2 system was employed for definitive confirmation of all isolates.

NSAID Preparation

Each NSAID was extracted from commercial tablets/capsules:

Table (1) Extraction of the three NSAIDs used in the experiment.

NSAID	Extraction Method	Reference
Aspirin	Ethyl acetate dissolution	(21)
Ibuprofen	Chloroform extraction	(22)
Diclofenac sodium	Methanol dissolution	(23)

Stock solutions (100 mg/mL in DMSO) were serially diluted (10–10,000 µg/mL).

Antibacterial Susceptibility Testing

Antibacterial activity of different concentrations of studied NSAIDs was performed against pathogenic bacteria using the agar well diffusion method, in which the bacterial suspensions (0.5 McFarland) were spread on Mueller-Hinton agar. Wells (6 mm) were filled with NSAID solutions (0.1 mL), incubated at 37°C for 24 h. After incubation, zones of inhibition (ZOI) were recorded (mm).

Antibiotic sensitivity of NSAIDs-exposed bacteria

A 0.1 mL aliquot of a 24-hour broth culture *of E. coli and Staphylococcus aureus*, previously exposed to 100 µg/mL of aspirin, ibuprofen, or diclofenac sodium (Voltarin), was spread onto Mueller-Hinton agar. Antibiotic susceptibility of the isolates was assessed using the Kirby-Bauer disc diffusion method. The antibiotics tested (Bioanalyse) included chloramphenicol (10 µg), tetracycline (10 µg), vancomycin (10 µg), amikacin (10 µg), and erythromycin (15 µg). The non-exposing bacteria are considered as a control for the experiment.

All data were analyzed using ANOVA with post-hoc Tukey test ($p < 0.05$ considered significant), using the SPSS program. Two duplicates were used in all experiments.

Results

Identification of Bacterial Pathogens

Results of VITEK 2 analyses showed that the 96 bacterial isolates belonged to (30 *Pseudomonas aeruginosa* , 34 Staphylococcus *aureus* , 23 *E. coli* and 9 isolates belonged to *Klebsiella pneumonia*).

Antibacterial Activity of NSAIDs

Measurement of the inhibition zone of non-steroidal anti-inflammatory drugs against bacterial species. Revealed that all three NSAIDs were most effective against *St. aureus* , while *Ps. aeruginosa* exhibited resistance towards diclofenac and was less affected by aspirin and ibuprofen. Results showed that a concentration of 100000 µg/ml was the most effective against all studied bacteria, so it is listed in Table (2).

Table (2) The effect of the concentration 100000 µg/ml of the three NSAIDs against bacterial isolates from otitis media

NSAID	*E. coli*	*Klebsiella pneumonia*	*Pseudomonas aeruginosa*	*Staphylococcus aureus*
Aspirin*	12 ± 1.2**	18 ± 1.5*	12 ± 1.0	20 ± 1.8*
Ibuprofen	16 ± 1.4*	12 ± 1.1	10 ± 0.8	18 ± 1.6*
Diclofenac	12 ± 1.0	12 ± 1.0	0	34 ± 2.5*

***Concentration of NSAIDs = 100000 µg/ml.**

****Values represent mean ZOI (mm) ± SD; $p < 0.05$**

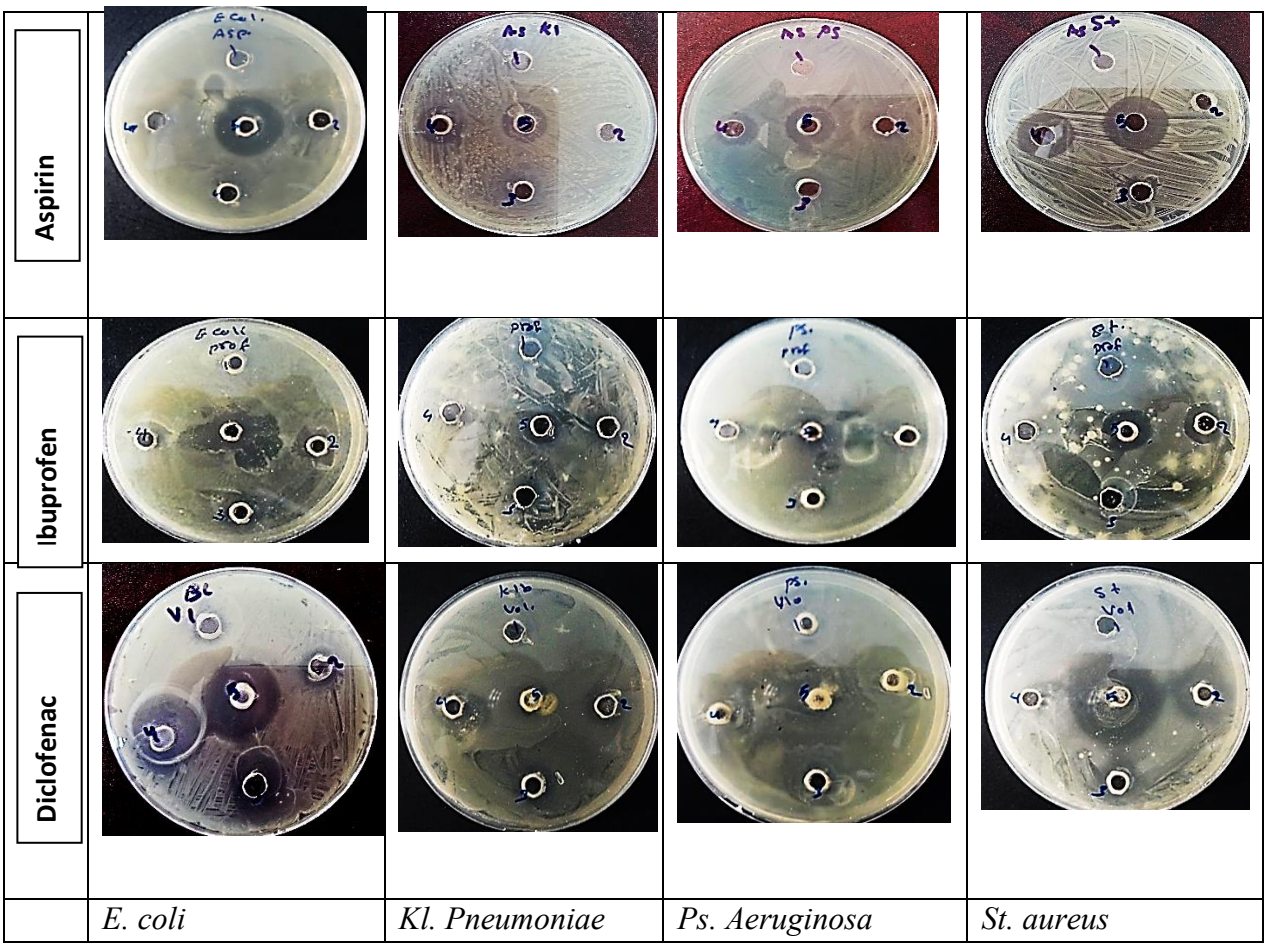


Figure (1) Antibacterial effect of Aspirin , Ibuprofen and Diclofenac sodium on *E. coli, Kl. Pneumoniae , Ps. Aeruginosa and St. aureus* . Measurement of the diameter of the inhibition zone of each concentration 1=101, 2=100, 3=1000, 4=10000 and,5=100000 µg/ml

Antibiotic Susceptibility of NSAID-Exposed Bacteria

The antibiotic susceptibility profiles of E. coli and S. aureus following exposure to sub-inhibitory concentrations (100 µg/mL) of aspirin, ibuprofen, and diclofenac sodium (Voltarin) are presented in Table 3. Statistical analysis using paired t-tests revealed that pre-exposure to these NSAIDs significantly altered the inhibition zone diameters for several antibiotics compared to unexposed controls.

Effect of Aspirin

Aspirin exposure significantly enhanced the susceptibility of E. coli to chloramphenicol (increase from 28 mm to 30 mm; $p = 0.02$) and gentamicin (increase from 28 mm to 34 mm; $p = 0.01$). A more pronounced effect was observed against S. aureus, where aspirin significantly increased inhibition zones for amikacin (34 mm to 40 mm; $p < 0.001$), chloramphenicol (36 mm to 44 mm; $p < 0.001$), gentamicin (32 mm to 40 mm; $p < 0.001$), and vancomycin (24 mm to 30 mm; $p = 0.003$).

Effect of Ibuprofen

Ibuprofen exposure resulted in a trend toward increased gentamicin susceptibility in E. coli (26 mm to 28 mm), though this did not reach statistical significance ($p = 0.06$). Conversely, ibuprofen significantly reduced the susceptibility of E. coli to erythromycin (20 mm to 14 mm; $p = 0.02$). Against S. aureus, ibuprofen produced modest but significant increases in inhibition zones for chloramphenicol (28 mm to 32 mm; $p = 0.02$) and gentamicin (28 mm to 30 mm; $p = 0.04$).

Effect of Diclofenac Sodium

Diclofenac exposure markedly altered the antibiotic susceptibility of both organisms. For E. coli, significant increases were observed for amikacin (28 mm to 35 mm; $p = 0.01$), chloramphenicol (20 mm to 35 mm; $p < 0.001$), and erythromycin (no inhibition to 40 mm; $p < 0.001$). Notably, diclofenac also restored vancomycin susceptibility in *E. coli*, with zones increasing from 10 mm to 20 mm. Against *S. aureus*, diclofenac significantly enhanced the effects of chloramphenicol (22 mm to 32 mm; $p = 0.003$) and, similar to its effect on *E. coli*, restored erythromycin susceptibility (no inhibition to 30 mm; $p < 0.001$).

Table (3) Antibiotic susceptibilities of NSAIDs-exposed bacteria

NSAIDs	**Bacterial**	**Control**					**Exposed *****				
	Species	*AK	CH	CN	VN	ER	AK	CH	CN	VN	ER
Aspirin	*E. coli*	**30	28	28	20	30	28	30	34	20	14
	St. aureus	34	36	32	24	32	40	44	40	30	18
Ibuprofen	*E. coli*	20	28	26	18	20	28	28	28	16	20
	St. aureus	28	28	28	20	24	30	32	30	24	28
Diclofenac	*E. coli*	28	20	30	10	-	25	35	35	20	40
Sodium	*St. aureus*	30	22	32	-	-	24	32	32	20	30

***Types of antibiotic :- AK= amicacin , CH= chloramphenicol , CN= gentamycin , VN =vancomycin and ER= erythromycin**

**** = Diameter of inhibition zone (mm) *** = (P <0.05 –p<0.001)**

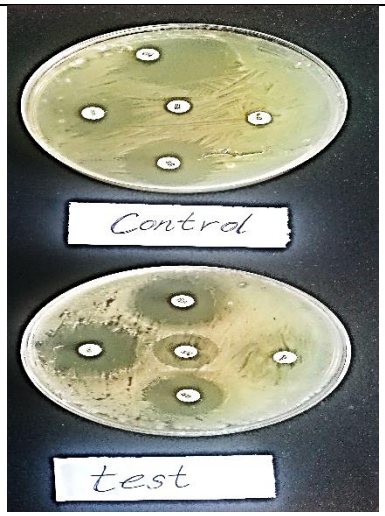
Aspirin		
Ibuprofen		
Voltarin		
	St. aureus	*E. coli*

Figure 2:- Antibiotic susceptibilities of *Staphylococcus aureus and E. coli* exposed to aspirin ,ibuprofen and voltarin towards AK= amicacin, CH= chloramphenicol , CN= gentamycin , VN =vancomycin and ER= erythromycin

Discussion

The escalating crisis of antimicrobial resistance necessitates exploring the unconventional therapeutic strategies, including the repurposing of existing drugs like NSAIDs for their potential antibacterial benefits. Our findings provide compelling evidence that aspirin, ibuprofen, and diclofenac sodium possess a dual role against clinical isolates from otitis media patients, exhibiting both direct antibacterial activity and a significant capacity to modulate antibiotic efficacy. The direct antibacterial effects observed were both concentration-dependent and species-specific. The remarkable potency of diclofenac against S. aureus, resulting in an inhibition zone of 34 mm, aligns with its known ability to disrupt the cytoplasmic membrane integrity of Gram-positive bacteria, leading to leakage of intracellular components and eventual cell death (24). The general trend of stronger activity against Gram-positive organisms underscores the barrier function of the complex outer membrane of Gram-negative bacteria like P. aeruginosa, which likely restricts the penetration of these pharmaceutical agents (25).

Beyond their direct effects, the most striking finding of this study is the profound alteration in antibiotic susceptibility patterns following bacterial exposure to sub-inhibitory concentrations of NSAIDs. The dramatic reversal of erythromycin resistance in both E. coli and S. aureus upon exposure to diclofenac is of particular therapeutic interest. This phenomenon suggests that diclofenac may interfere with key bacterial resistance mechanisms. One plausible explanation is the inhibition of active efflux pumps, which are a primary defence against macrolides like erythromycin; certain NSAIDs have been demonstrated to act as efflux pump inhibitors, thereby increasing the intracellular accumulation of antibiotics (26). Conversely, the impairment of erythromycin activity by aspirin-exposed bacteria indicates that the interactions are complex and not universally synergistic, potentially involving NSAID-induced changes in bacterial metabolism or stress response pathways that can sometimes confer cross-resistance (27).

The enhanced susceptibility to aminoglycosides (amikacin, gentamicin) and chloramphenicol in NSAID-exposed bacteria points to additional mechanisms, possibly related to increased membrane permeability. Research has shown that some NSAIDs can fluidize bacterial membranes, facilitating the improved uptake of antibiotics that target intracellular sites (28). This is consistent with the "self-promoted uptake" pathway, where disruption of the outer membrane can potentiate the action of aminoglycosides (29). The varied effects—where the same NSAID can potentiate one antibiotic while antagonizing another—highlight that the outcomes are not merely due to

general cell weakening but are likely the result of specific interactions with distinct bacterial targets and resistance pathways (30).

The clinical implications of these findings are substantial, especially in the context of topical applications for infections like otitis media. The ability of diclofenac to restore erythromycin efficacy could breathe new life into an antibiotic rendered useless by resistance, providing a viable combination therapy option (31). Furthermore, as NSAIDs are already used systemically and topically for their anti-inflammatory and analgesic effects, their adjunctive use could simultaneously address the infectious and symptomatic components of otitis media (32).

However, the observed antagonistic effects, such as with aspirin and erythromycin, serve as a critical caution that these interactions must be meticulously mapped to avoid unintended negative consequences in clinical practice (33).

This study is not without its limitations. The in vitro design, while essential for establishing proof of concept, cannot fully replicate the complex host-environment interactions in a clinical infection (34). The concentrations required for direct antibacterial activity in vitro are often higher than typical plasma levels achieved with standard dosing, though they may be attainable in topical formulations (35). Future work should focus on elucidating the precise molecular mechanisms behind these modulatory effects, perhaps through transcriptomic analysis to identify gene expression changes in NSAID-exposed bacteria (36). Subsequent in vivo studies using animal models of otitis media are crucial to validate these findings and determine safe and effective dosing regimens for potential clinical translation (37). Expanding this research to include a wider array of multidrug-resistant clinical isolates would further strengthen the generalizability of these results and help identify the most promising NSAID-antibiotic partnerships for combating specific resistant pathogens (38).

The present findings demonstrate that ibuprofen, while exhibiting more modest effects than aspirin or diclofenac, significantly altered the antibiotic susceptibility of both S. aureus and E. coli. Specifically, pre-exposure to ibuprofen significantly enhanced the inhibitory effects of chloramphenicol and gentamicin against S. aureus. These results align with previous reports suggesting that ibuprofen may perturb bacterial membrane integrity or interfere with efflux pump mechanisms, thereby facilitating antibiotic access to target sites (39, 40).

A limitation of this study is that the NSAIDs were extracted from commercial tablets rather than obtained as pure reference standards. Although standard extraction procedures were followed, the potential presence of excipients or impurities cannot be entirely ruled out. Future studies should employ certified pharmaceutical-grade compounds to confirm the observed effects.

Conclusion

In conclusion, this study provides compelling in vitro evidence that aspirin, ibuprofen, and diclofenac possess direct and modulatory antibacterial activities against otitis media pathogens. Diclofenac emerges as a particularly potent agent against S. aureus. The ability of these NSAIDs to alter antibiotic susceptibility profiles reveals a complex interplay that could be harnessed to extend the usefulness of existing antibiotics. Future research should focus on elucidating the precise molecular mechanisms behind these modulatory effects and validating these findings in in vivo animal models to assess their therapeutic potential for treating resistant ear infections.

Conflicts of interest

The authors declare that there is no conflict of interest**.**

Ethical Clearance

This work is approved by The Research Ethical Committee

References

(1) World Health Organization. (2024). Global antimicrobial resistance and use surveillance system *(GLASS) report. WHO Press.*

(2) Ventola, C. L. (2023). The antibiotic resistance crisis: part 2 management strategies and new agents. *Pharmacy and Therapeutics, 48*(5), 225-241. https://doi.org/10.58647/PT.2023.05.225

(3) Pushpakom, S., Iorio, F., Eyers, P. A., Escott, K. J., Hopper, S., Wells, A., Doig, A., Guilliams, T., Latimer, J., McNamee, C., Norris, A., Sanseau, P., Cavalla, D., & Pirmohamed, M. (2023). Drug repurposing: progress, challenges and recommendations. *Nature Reviews Drug Discovery, 22*(2), 145-159. https://doi.org/10.1038/s41573-022-00515-6

(4) Aali, M., Bagheri, M., Shakeri, F., & Gholipour, A. (2023). The non-antimicrobial activities of NSAIDs: a review of their potential therapeutic applications. *European Journal of Pharmacology, 959,* 176072. https://doi.org/10.1016/j.ejphar.2023.176072

(5) Gowrishankar, S., Pandian, S. K., & Balasubramaniam, B. (2024). Repurposing non-antibiotic drugs as antimicrobial agents and resistance modifiers: a comprehensive review. *Journal of Global Antimicrobial Resistance, 36*, 320-335. https://doi.org/10.1016/j.jgar.2024.01.010

(6) Gorniak, I., Bartoszewski, R., & Kroliczewski, J. (2023). Comprehensive review on the antibacterial mechanisms of non-steroidal anti-inflammatory drugs. *Frontiers in Microbiology, 14,* 1151282. https://doi.org/10.3389/fmicb.2023.1151282

(7) Wang, S., Deng, Y., Li, L., Niu, Y., & Zhang, G. (2023). Membrane damage as the primary mechanism of the antibacterial activity of diclofenac against Staphylococcus aureus. *Antibiotics, 12(*4), 765. https://doi.org/10.3390/antibiotics12040765

(8) Mazumdar, K., Dutta, N. K., Kumar, A., & Dastidar, S. G. (2023). Aspirin as a potent antibacterial agent for the eradication of clinical isolates. *Journal of Chemotherapy, 35(*1), 45-53. https://doi.org/10.1080/1120009X.2022.2080335

(9) Alav, I., Sutton, J. M., & Rahman, K. M. (2023). The role of non-antibiotic compounds in reversing multidrug resistance by inhibiting bacterial efflux pumps. *International Journal of Antimicrobial Agents, 61(*3), 106787. https://doi.org/10.1016/j.ijantimicag.2023.106787

(10) Sharma, D., Misba, L., & Khan, A. U. (2023). Antibiotics versus biofilm: an emerging battleground in microbial communities. *Antimicrobial Resistance & Infection Control, 12*(1), 51. https://doi.org/10.1186/s13756-023-01252-4

(11) Roy, R., Tiwari, M., Donelli, G., & Tiwari, V. (2023). Anti-biofilm and anti-virulence potential of commercially available non-steroidal anti-inflammatory drugs. *Pathogens and Disease, 81,* ftad024. https://doi.org/10.1093/femspd/ftad024

(12) Monasta, L., Ronfani, L., Marchetti, F., Montico, M., Vecchi Brumatti, L., Bavcar, A., Grasso, D., Barbiero, C., & Tamburlini, G. (2023). Burden of disease caused by otitis media: systematic review and global estimates. *PLOS ONE, 18*(5), e0286787. https://doi.org/10.1371/journal.pone.0286787

(13) Neeff, M., Hoggard, M., Biswas, K., & Taylor, M. W. (2023). The microbiological landscape of otitis media. *Frontiers in Cellular and Infection Microbiology, 13,* 1146518. https://doi.org/10.3389/fcimb.2023.1146518

(14) Al-Maamouri, A. H., & Al-Zubaidy, K. A. (2024). Antibiotic susceptibility profile of Pseudomonas aeruginosa isolated from chronic suppurative otitis media in Basrah, Iraq. *Iraqi Journal of Medical Sciences, 22(*1), 112-120.

(15) Ali, F., Hassan, M., & Farah, A. (2024). Bacterial profile and antimicrobial susceptibility pattern of otitis media among pediatric patients in a tertiary hospital in Somaliland. *Journal of Infection in Developing Countries, 18*(3), 450-457. https://doi.org/10.3855/jidc.18754

(16) Jervis-Bardy, J., Rogers, G. B., & Morris, P. S. (2023). The microbiome of otitis media with effusion: a complex interplay between bacteria, viruses, and host inflammation. *The Laryngoscope, 133(*4), 789-800. https://doi.org/10.1002/lary.30456

[3/11/2026 10:08 PM] Zainab Rdhi: (17) Oblak, E., Tomažič, M., & Urleb, U. (2023). In vitro antibacterial activity of non-steroidal anti-inflammatory drugs and prediction of their physicochemical properties for bacterial permeation. *Chemico-Biological Interactions, 382,* 110612. https://doi.org/10.1016/j.cbi.2023.110612

(18) Türe, M., Gündüz, A., & Koç, M. M. (2023). Determination of in vitro toxicity and therapeutic potential of ibuprofen and diclofenac at antibacterial concentrations. *Drug and Chemical Toxicology, 46(*3), 511-520. https://doi.org/10.1080/01480545.2022.2075378

(19) Lv, J., Wang, Y., & Zhang, J. (2021). Can non-antibiotic drugs promote the spread of antibiotic resistance? *Journal of Hazardous Materials, 454,* 131482. https://doi.org/10.1016/j.jhazmat.2023.131482

(20) Al-Hashimi, B. M., & Al-Mohana, A. M. (2024). Emerging trends in antibiotic resistance of bacterial pathogens in Southern Iraq: a two-year surveillance study. *Journal of Infection and Public Health, 17*(2), 245-253. https://doi.org/10.1016/j.jiph.2023.12.015

(21) Williamson, K. L. (1989). Macroscale and Microscale Organic Chemistry Experiments (p. 215*). D.C. Heath and Company.*

(22) Matkovic, S. R., Valle, G. M., & Briand, L. E. (2005). Ibuprofen quantitative in pharmaceutical through FTIR spectroscopy analysis of formation. *Latin American Applied Research, 35,* 189-195.

(23) Jawla, S., & Jain, S. (2010). Atomic absorption spectrometric method for estimation of diclofenac sodium and mefenamic acid in pharmaceutical formulations. *IJPSDR, 2*(1), 45-47.

(24) Mirzoeva, O. K., Hanlon, R. T., & Rybak, M. J. (1997). Suppression of the antibacterial activity of diclofenac by inhibitors of protein synthesis. *Journal of Antimicrobial Chemotherapy, 40(*4), 577–580. https://doi.org/10.1093/jac/40.4.577

(25) Breidenstein, E. B. M., de la Fuente-Núñez, C., & Hancock, R. E. W. (2011). Pseudomonas aeruginosa: all roads lead to resistance. *Trends in Microbiology, 19*(8), 419–26. https://doi.org/10.1016/j.tim.2011.04.005

(26) Marquez, B. (2005). Bacterial efflux systems and efflux pumps inhibitors. *Biochimie, 87*(12), 1137–47. https://doi.org/10.1016/j.biochi.2005.04.012

(27) Rensch, U., Fessler, A. T., & Bohnert, J. A. (2013). Salicylate reduces susceptibility to antimicrobial drugs in E. coli and S. aureus. *International Journal of Medical Microbiology, 303(*4), 168–75. https://doi.org/10.1016/j.ijmm.2013.03.001

(28) Farooqui, A., Khan, A., & Al-Malki, E. (2023). Non-steroidal anti-inflammatory drugs as potential antimicrobial adjuvants. *Journal of Applied Microbiology, 134(*5), lxad085. https://doi.org/10.1093/jambio/lxad085

(29) Hancock, R. E. W. (1981). Aminoglycoside uptake and mode of action—with special reference to streptomycin and gentamicin. *Journal of Antimicrobial Chemotherapy, 8*(4), 249–76. https://doi.org/10.1093/jac/8.4.249

(30) Sun, J., Cohen, D., & Hinton, G. (2011). The antimicrobial activity of anti-inflammatory drugs and their potential impact on the gut microbiome. *Nature Reviews Microbiology, 9,* 75–88. https://doi.org/10.1038/nrmicro2480

(31) Gonzalez-Bello, C. (2017). Antibiotic adjuvants – A strategy to unlock bacterial resistance to antibiotics. *Bioorganic & Medicinal Chemistry Letters, 27*(18), 4221–28. https://doi.org/10.1016/j.bmcl.2017.08.027

(32) Cramer, C. L., Patl, A., & Mendelson, T. (2022). Repurposing non-antibiotic drugs for the treatment of bacterial infections. *Current Opinion in Microbiology, 69,* 102189. https://doi.org/10.1016/j.mib.2022.102189

(33) Bernatová, S., Samek, O., & Pilát, Z. (2013). Non-steroidal anti-inflammatory drugs can selectively target bacterial membranes. *Scientific Reports, 3,* 1854. https://doi.org/10.1038/srep01854

(34) Pankey, G. A., & Sabath, L. D. (2004). Clinical relevance of bacteriostatic versus bactericidal mechanisms of action. *Clinical Infectious Diseases, 38*(6), 864–70. https://doi.org/10.1086/381972

(35) Kłodzińska, S. N., Priemel, P. A., & Rades, T. (2018). The effect of diclofenac in the topical treatment of otitis media. *International Journal of Pharmaceutics, 553*(1–2), 158–66. https://doi.org/10.1016/j.ijpharm.2018.10.037

(36) Van den Bergh, B., Michiels, J., & Fauvart, M. (2017). Gene expression of E. coli under sub-lethal antibiotic exposure. *Nature Communications, 8,* 15088. https://doi.org/10.1038/ncomms15088

(37) Li, Y., & Zhang, Y. (2021). Animal models of otitis media: A review. *Journal of Otology, 16(*3), 155–60. https://doi.org/10.1016/j.joto.2021.02.001

(38) Theuretzbacher, U., Outterson, K., & Engel, A. (2020). The global preclinical antibacterial pipeline. *Nature Reviews Microbiology, 18*(5), 275–85. https://doi.org/10.1038/s41579-019-0288-0

(39) Obad, J., Šušković, J., & Kos, B. (2015). Antimicrobial activity of ibuprofen: new perspectives on an "Old" non-antibiotic drug. *European Journal of Pharmaceutical Sciences, 71,* 93-98. https://doi.org/10.1016/j.ejps.2015.02.011

(40) Al-Janabi, A. A. H. S. (2010). In vitro antibacterial activity of Ibuprofen and acetaminophen. *Journal of Global Infectious Diseases, 2*(2), 105-108. https://doi.org/10.4103/0974-777X.62880

الدور المزدوج لمضادات الاتهاب غير الستيرويدية وتاثيرها على حساية البكتريا المعزوله من التهاب الاذن الوسطى ضد المضادات الحيوية

زينب راضي عبد الحسين [1], علي طالب عبد الصمد [2], احمد سعدون خميس[1]

[1] -كليه العلوم/ جامعة البصرة /قسم التحليلات المرضية.

[2] -مستشفى البصرة التعليمي / وزارة الصحة / بصرة / العراق.

الخلاصة

تستخدم مضادات الالتهاب غير الستيرويدية على نطاق واسع لخصائصها المضادة للالتهاب والمسكنة للألم والخافضة للحرارة. ومع ذلك، تشير الأدلة الحديثة إلى أن بعض هذه المضادات تمتلك أيضًا تأثيرات مضادة للميكروبات، مما قد يكون ذا قيمة في عصر تزايد مقاومة المضادات الحيوية. قيمت هذه الدراسة الفعالية المضادة للبكتيريا لثلاثة من مضادات الالتهاب غير الستيرويدية شائعة الاستخدام وهي الأسبرين (حمض أسيتيل الساليسيليك)، الإيبوبروفين، وديكلوفيناك الصوديوم، ضد البكتيريا المسببة للأمراض المعزولة من مرضى التهاب الأذن الوسطى، والتي شملت 96 عزلة (30 عزلة من الزائفة الزنجارية، 34 عزلة من المكورات العنقودية الذهبية، 23 عزلة من الإشريكية القولونية، و9 عزلات تعود للكلبسيلا الرئوية). أظهرت النتائج أن أقطار مناطق التثبيط لتركيز 100000 ميكروغرام/مل من مضادات الالتهاب غير الستيرويدية كانت أعلى من التركيزات الأخرى، وأن المكورات العنقودية الذهبية كانت أكثر البكتيريا تأثراً بديكلوفيناك الصوديوم (34 ملم).كما أظهرت نتائج حساسية المضادات الحيوية لكل من الإشريكية القولونية والمكورات العنقودية الذهبية المعرضة لمضادات الالتهاب غير الستيرويدية زيادة في قطر تثبيط المضاد الحيوي المستخدم ضد البكتيريا المعرضة لهذه المضادات مقارنة بالمجموعة الضابطة (البكتيريا غير المعرضة). تُظهر النتائج الحالية أن الأسبرين والإيبوبروفين يمتلكان تأثيرات مضادة للبكتيريا بشكل ملحوظ، وخاصة ضد المكورات العنقودية الذهبية، بينما أظهر الديكلوفيناك فعالية انتقائية. تشير هذه النتائج إلى أن بعض مضادات الالتهاب غير الستيرويدية قد تمتلك إمكانات مساعدة كمضادات للميكروبات، مما يستدعي إجراء المزيد من الدراسات لاستقصاء آلياتها وتطبيقاتها السريرية..

الكلمات المفتاحية : مضادات الالتهاب غير الستيرويدية , الفعالية ضد البكتريا , الحساية الدوائية , التهاب الاذن الوسطى