ORIGINAL ARTICLE

Repurposing an Anticonvulsant Drug against Pediatric Pathogens: Antibacterial, Antibiofilm, Antioxidant and Synergistic Properties of Gabapentin

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Abstract

Aim: Multidrug-resistant bacterial infections present a significant challenge in pediatric cystic fibrosis (CF), where *Staphylococcus aureus* and *Pseudomonas aeruginosa* commonly establish persistent airway colonization through biofilm formation. Biofilms confer enhanced tolerance to antibiotics and host defences, complicating infection management. So, the present study was aimed to investigate the antibacterial, antibiofilm, synergistic, and antioxidant potential of gabapentin, a second-generation neuroleptic drug, against the mentioned CF-associated pathogens. **Materials and Methods:** To find the antibacterial activity of the drug and its minimum inhibitory concentrations, agar diffusion assays were performed. Crystal violet assays were conducted to understand the biofilm prevention and eradication potentials of the drug. The synergic potential of the drug was analysed with ampicillin and rifampicin. To study the anti-oxidant activities of the drug, DPPH assay and catalase assay were performed. **Results:** The drug gabapentin shown promising antibacterial activities with minimum inhibitory concentrations of 250 µg/mL for S. aureus and 125 µg/mL for *P. aeruginosa*. It also inhibited the bacterial adhesion and early biofilm formation, and significantly disrupted mature biofilms, reducing *S. aureus* and *P. aeruginosa* biofilm mass by up to 86% and 82%, respectively, at 3X MIC. It also showed synergistic interactions with the tested antibiotics, ampicillin and rifampicin. Additionally, gabapentin exhibited notable antioxidant activity. **Conclusion:** The above-mentioned results suggest that gabapentin has multifunctional potential as an adjunctive therapeutic agent for managing biofilm-mediated, multidrug-resistant infections in paediatric cystic fibrosis, offering a promising strategy to improve clinical outcomes.

Key words: Antibiofilm, drug repurposing, gabapentin, pediatric cystic fibrosis, synergism

INTRODUCTION

Antimicrobial resistance has emerged as a significant obstacle in the successful management of infectious diseases and is associated with increased rates of morbidity and mortality.[1] A major limitation in addressing this problem lies in the reliance on conventional antimicrobial susceptibility testing, which is typically performed on single bacterial species under planktonic conditions.[2,3] Such testing often fails to accurately reflect treatment outcomes observed in clinical settings. Increasing evidence suggests that many chronic and persistent infections are polymicrobial and involve microorganisms growing within structured communities known as biofilms, which exhibit enhanced tolerance to antimicrobial agents.[4]

In pediatric patients with cystic fibrosis (CF), airway colonization commonly begins with *Staphylococcus aureus* during early childhood.[5] With advancing age, this organism

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Received: 18-01-2026
Revised: 04-03-2026
Accepted: 13-03-2026

is frequently replaced by *Pseudomonas aeruginosa*, often following a transitional phase in which both bacteria coexist.[6,7] This pattern of succession indicates potential biological interactions between these pathogens that may influence disease progression and treatment response. Early acquisition of *P. aeruginosa* is of particular clinical concern, as chronic colonization is associated with declining pulmonary function and poorer long-term outcomes.[8] Consequently, aggressive antibiotic therapy is initiated upon first detection in an effort to eliminate the organism before chronic infection becomes established.

Despite the use of inhaled antibiotics such as tobramycin, which achieve high concentrations within the airways, eradication of *P. aeruginosa* is unsuccessful in a significant proportion of pediatric CF patients. Reported failure rates range from approximately 10% to 40%, underscoring the complexity of infection management in this population.[9-12] Notably, studies have been unable to consistently identify specific host factors or bacterial traits that reliably predict treatment failure. This suggests that additional mechanisms, including interactions between co-colonizing organisms within biofilms, may play an important role in the persistence of infection. However, limited research has focused on how interspecies interactions, particularly between *S. aureus* and *P. aeruginosa*, contribute to reduced antibiotic effectiveness and the failure of eradication strategies in CF airways.[4]

Biofilm formation is a major survival strategy employed by pathogenic bacteria, enabling them to withstand hostile conditions more effectively than planktonic cells. Biofilms are organized microbial communities encased within a self-produced extracellular matrix composed of polysaccharides, proteins, and other organic components that promote firm adherence to biotic and abiotic surfaces.[13] This matrix acts as a physical and chemical barrier, protecting bacteria from antimicrobial agents and host immune responses while also facilitating the exchange of genetic material, including antimicrobial resistance determinants, among biofilm-associated cells.[14] Consequently, biofilms are implicated in a substantial proportion of persistent and recurrent infections, accounting for more than half of all microbial infections and nearly two-thirds of bacterial infections in humans.[15] These characteristics make biofilm-associated infections particularly difficult to eradicate in healthcare settings.

The development of novel antimicrobial agents from natural sources such as plants, soil microorganisms, and marine environments is often limited by high costs, prolonged timelines, and extensive regulatory requirements. In contrast, drug repurposing has emerged as a practical and efficient alternative, as repurposed drugs possess established safety profiles and well-characterized pharmacological properties.[16,17] Gabapentin, a second-generation neuroleptic approved by the U.S. Food and Drug Administration, is a structural analogue of gamma-aminobutyric acid and is widely prescribed for neuropathic pain and certain neurological disorders. Given its favorable safety profile, the present study investigated the antibacterial, antibiofilm, and antioxidant potential of gabapentin against *S. aureus* and *P. aeruginosa*, key biofilm-forming pathogens associated with pediatric CF.

MATERIALS AND METHODS

Inoculum preparation

Overnight bacterial cultures of *S. aureus* and *P. aeruginosa*, standardized to 0.5 McFarland, were obtained from inpatients in the pediatric department of a tertiary care hospital and used for all experiments. *S. aureus* was cultured in Brain Heart Infusion (BHI) broth, whereas *P. aeruginosa* was grown in Mueller-Hinton broth (MHB). All assays were conducted in triplicate to ensure reproducibility. Ampicillin and rifampicin were included as positive controls.

Antibacterial activity of gabapentin

The antibacterial effect of gabapentin against *S. aureus* and *P. aeruginosa* was assessed using the well diffusion method described previously.[18] Briefly, overnight cultures were uniformly spread on sterile BHI or Mueller–Hinton agar plates. Wells were punched aseptically, and different concentrations of gabapentin were added. Plates were incubated, and the zones of inhibition were measured to determine antibacterial activity.

Minimum inhibitory concentration (MIC) determination

MIC values of gabapentin against the test organisms were determined using the microdilution method.[19] A stock solution of 250 µg/mL gabapentin was prepared and serially diluted to obtain concentrations down to 1.9 µg/mL in the appropriate broth (BHI or MHB). After inoculation with overnight cultures of *S. aureus* and *P. aeruginosa*, plates were incubated, and optical density was recorded at 600 nm to determine MIC values.

Effect on bacterial adhesion

The effect of gabapentin on the adhesion of *S. aureus* and *P. aeruginosa* to polystyrene surfaces was assessed using the crystal violet assay as described previously.[19] Briefly, overnight cultures of the test organisms were exposed to varying concentrations of gabapentin (250–1.9 µg/mL) and allowed to adhere to polystyrene plates for 3 h. Following incubation, non-adherent cells were removed, and the attached cells were fixed with methanol and stained with crystal violet. The bound dye was then solubilized using an ethanol–acetone mixture, and absorbance was measured at 570 nm to quantify bacterial adhesion.

Effect of gabapentin on biofilm formation

Gabapentin's effect on biofilm formation by the test organisms (*S. aureus* and *P. aeruginosa*) was examined using the crystal violet method.[20] Biofilms were allowed to develop for 5 days in wells containing different concentrations (250–1.9 µg/mL) of gabapentin. After incubation, the biofilms were fixed with methanol, stained with crystal violet, and destained with an ethanol–acetone mixture. Absorbance of the final solution was recorded at 570 nm.

Effect of gabapentin on biofilm eradication

The ability of gabapentin to disrupt established *S. aureus* and *P. aeruginosa* biofilms was assessed following the procedure.[18] Five-day-old mature biofilms of all test pathogens were treated with ×1, ×2, and ×3 MIC concentrations of gabapentin. After treatment, the remaining biofilm was fixed with methanol, stained with crystal violet, and destained with an ethanol–acetone mixture. The absorbance was read at 570 nm.

Synergistic activity

The synergistic interaction between gabapentin and selected commercial antibiotics, ampicillin for *S. aureus* and rifampicin for *P. aeruginosa* was evaluated using the checkerboard assay as previously described.[18] Briefly, gabapentin and the respective antibiotics were prepared at concentrations corresponding to one level above the MIC, the MIC, and three levels below the MIC. These combinations were inoculated with overnight cultures of *S. aureus* and *P. aeruginosa* and incubated. Following incubation, optical density was measured at 600 nm. The degree of interaction was assessed by calculating the fractional inhibitory concentration index (FICI), obtained by adding the FIC values of gabapentin and the respective antibiotics. The FIC of gabapentin was determined by dividing its MIC in combination by its MIC when tested alone.

Antioxidant activity

2,2-diphenyl-1-picrylhydrazyl (DPPH) assay

The antioxidant activity of gabapentin was evaluated using the DPPH free radical scavenging assay, following the method.[21] Various concentrations of gabapentin (100, 200, 400, 600 and 1000 µg/mL) were mixed with the DPPH solution and incubated for 30 min. The absorbance of the reaction mixture was then measured at 517 nm to determine the percentage of radical scavenging activity.

Catalase assay

Catalase activity in the presence of gabapentin was analysed as standardized protocol.[22] Various concentrations of gabapentin (100, 200, 400, 600 and 1000 µg/mL) were mixed with 1.5 mL of phosphate buffer, followed by the addition of 60 mM hydrogen peroxide. The reduction in absorbance at 240 nm, corresponding to hydrogen peroxide breakdown, was used to determine gabapentin's radical scavenging capacity.

Statistical analysis

All experiments were conducted in triplicate. Mean values and standard deviations were calculated, and standard error was derived accordingly.

RESULTS

Antibacterial activity of gabapentin

Gabapentin's ability to inhibit bacterial growth was evaluated against *S. aureus* and *P. aeruginosa*, and the results are shown in Figure 1. Both tested concentrations produced clear inhibition zones around the wells, and the diameter of these zones increased in a dose-dependent manner for each organism.

MIC

The MIC values of gabapentin for *S. aureus* and *P. aeruginosa* were determined using the microdilution method [Figure 2]. Gabapentin inhibited the growth of *S. aureus* at 250 µg/mL, whereas *P. aeruginosa* required only 125 µg/mL for measurable growth suppression.

Effect on bacterial adhesion

Gabapentin's influence on bacterial attachment to non-biological surfaces was quantitatively assessed [Figure 3]. Up to the MIC level, gabapentin markedly reduced the adhesion of both test species to polystyrene surfaces. As the concentration decreased and exposure time increased to 3 h, adhesion gradually began to rise.

Effect on biofilm formation

Biofilm-forming ability in the presence of varying gabapentin concentrations (250–1.9 µg/mL) is presented in Figure 4. No detectable biofilm formation occurred at MIC levels, while a progressive increase in biofilm biomass was observed when concentrations fell below the MIC. However, even at lower doses, the total biofilm formation remained limited, suggesting that small quantities of gabapentin can still hinder biofilm development.

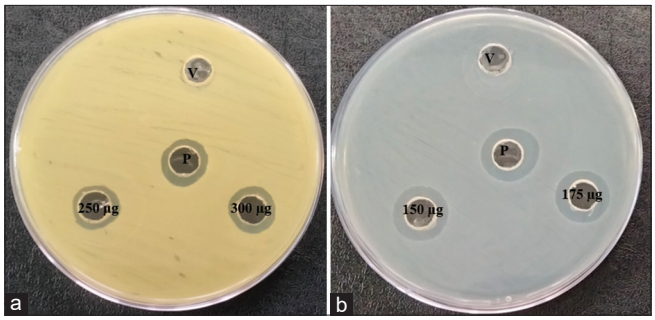

Figure 1: Antibacterial activity of gabapentin against (a) *Staphylococcus aureus* and (b) *Pseudomonas aeruginosa*. P=Positive control; V=Vehicle control (water)

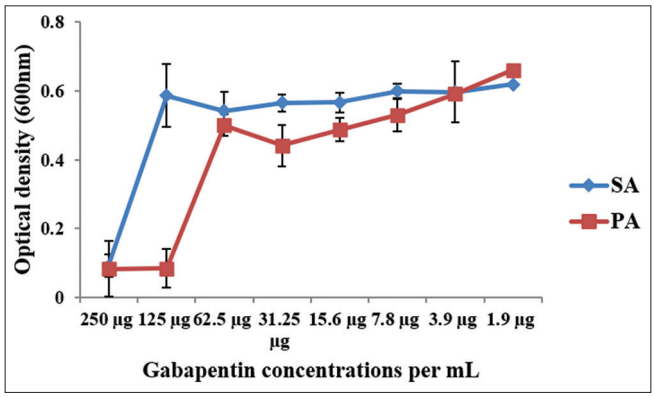

Figure 2: Minimum inhibitory concentration determination of gabapentin against *Staphylococcus aureus* and *Pseudomonas aeruginosa*

Effect on biofilm eradication

Gabapentin's ability to disrupt mature biofilms was evaluated at ×1, ×2, and ×3 MIC [Figure 5]. For *S. aureus*, mature biofilms were reduced by 65%, 81%, and 86%, respectively. For *P. aeruginosa*, the reductions were 68%, 72%, and 82%. These findings indicate that gabapentin can effectively eradicate established biofilms in a concentration-dependent manner.

Synergistic activity

The combined effect of gabapentin with conventional antibiotics is shown in Figure 6. When paired with ampicillin, gabapentin produced a two-fold reduction in MIC for *S. aureus* (from 250 to 125 µg/mL). Similarly, co-treatment with rifampicin lowered the MIC for *P. aeruginosa* from 125 to 62.5 µg/mL. Both combinations yielded a FICI of 0.5, confirming a synergistic interaction and indicating that gabapentin can potentiate the activity of these antibiotics.

Antioxidant activity

DPPH assay

The antioxidant potential of gabapentin was further examined using the DPPH radical scavenging method. As illustrated

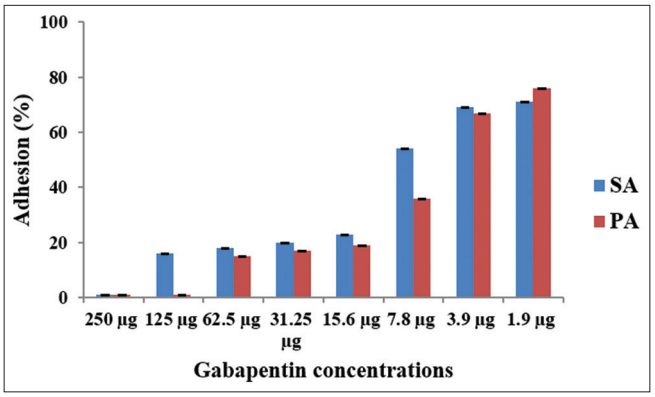

Figure 3: Effect of gabapentin on the adhesion of *Staphylococcus aureus* and *Pseudomonas aeruginosa*

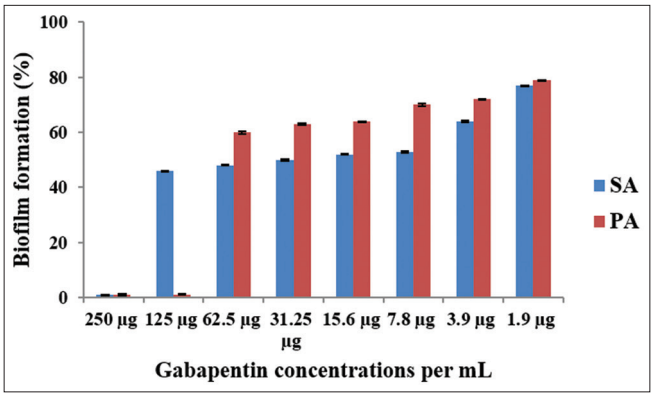

Figure 4: Influence of gabapentin on biofilm formation by *Staphylococcus aureus* and *Pseudomonas aeruginosa*

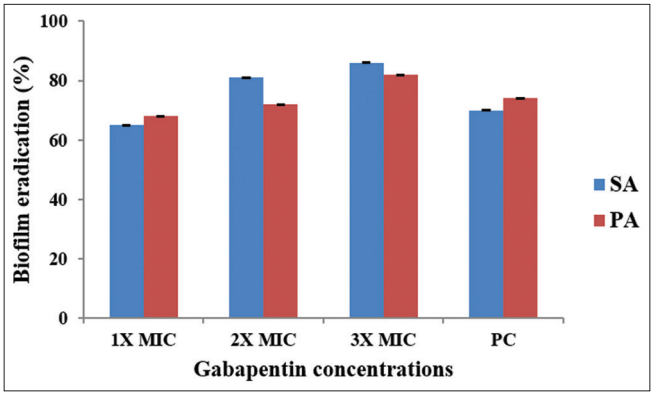

Figure 5: Quantitative analysis of gabapentin-mediated eradication of mature *Staphylococcus aureus* and *Pseudomonas aeruginosa* biofilms. PC=Positive control

in Figure 7, gabapentin showed a progressive increase in its ability to neutralize free radicals as the concentration increased. Starting from 100 µg/mL and rising to 1000 µg/mL, the scavenging activity increased from 20% to 52%. This upward trend indicates that gabapentin becomes more effective at quenching DPPH radicals at higher concentrations.

Catalase assay

Gabapentin's influence on catalase-mediated oxidative processes was also measured, and the results are displayed

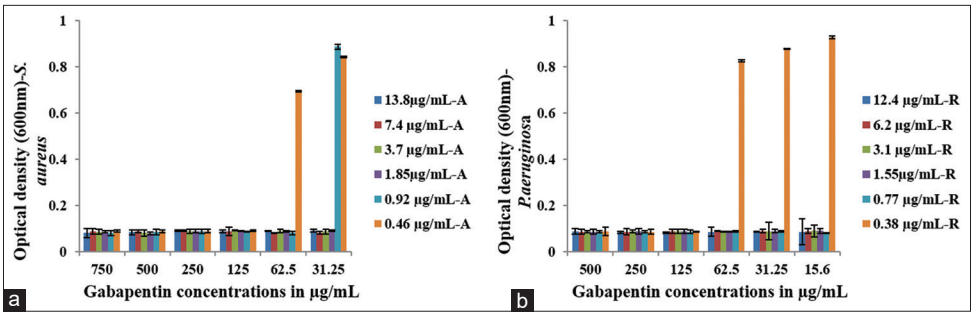

Figure 6: Synergistic effects of gabapentin with (a) ampicillin against *Staphylococcus aureus* and (b) rifampicin against *Pseudomonas aeruginosa*

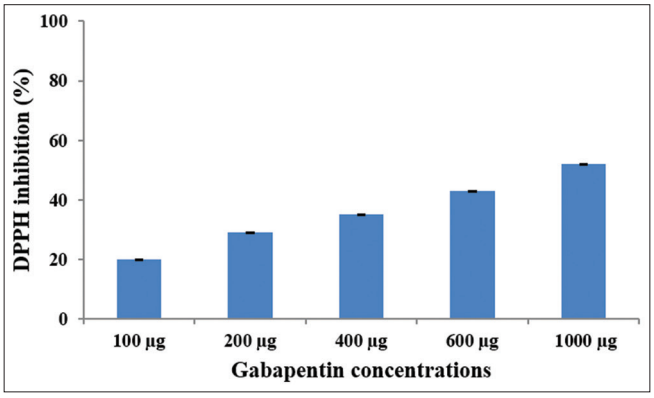

Figure 7: 2,2-diphenyl-1-picrylhydrazyl radical-scavenging activity of gabapentin

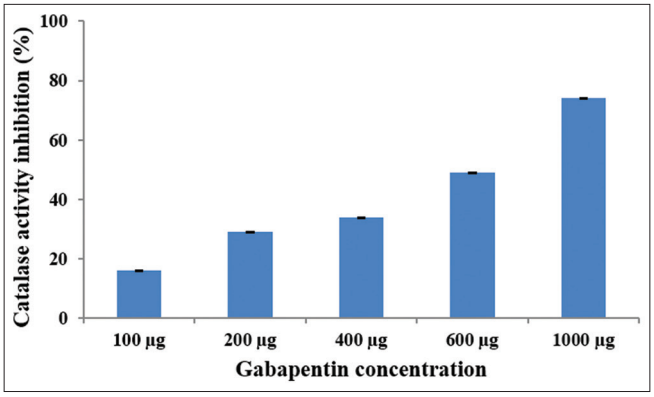

Figure 8: Catalase scavenging activity of gabapentin

in Figure 8. The compound exhibited a clear dose-responsive increase in catalase scavenging ability, ranging from 16% at the lowest concentration to 74% at the highest. These findings suggest that gabapentin can significantly reduce oxidative stress by interfering with catalase activity in a concentration-dependent manner.

DISCUSSION

In pediatric CF, persistent airway infections with *S. aureus* and *P. aeruginosa* remain a major clinical challenge due to biofilm-mediated antimicrobial resistance. The failure of eradication therapies highlights the role of interspecies interactions within biofilms, which protect bacteria from antibiotics and immune responses. Repurposed agents like gabapentin show promise in targeting these biofilm-forming pathogens, potentially reducing infection persistence and improving long-term outcomes in children with CF. In the present study, we examined the antibacterial potential of gabapentin, a second-generation neuroleptic medication against *S. aureus* and *P. aeruginosa*. The findings indicated clear inhibitory effects on both pathogens, as demonstrated by the measured MICs. In line with our results, gabapentin sulfonamide and its novel derivatives have also been evaluated for antibacterial activity against several bacterial strains, including *Escherichia coli*, *Streptococcus faecalis*, *Salmonella* Typhi, *S. aureus*, and *P. aeruginosa*. These compounds exhibited strong inhibitory activity at 12.18 mg/mL.[23] Similarly, newly synthesized isoindole-1,3 (2H)-dione derivatives produced through molecular hybridization of gabapentin and pregabalin with phthalic anhydride derivatives were assessed for their antimicrobial potential. All synthesized compounds demonstrated concentration-dependent antibacterial activity against *E. coli* and *S. aureus*, whereas the parent drugs showed no antibacterial effect at 20 µg/mL.[24]

Biofilm formation plays a crucial role in the emergence of antibiotic resistance, progressing through well-defined stages that include initial adhesion, surface colonization, and maturation each contributing to the persistence of infections. In the present study, we evaluated the effect of gabapentin on these stages of biofilm development and found that it exerted a pronounced inhibitory action, particularly by preventing biofilm establishment on polystyrene surfaces. In addition, gabapentin was capable of disrupting mature, preformed biofilms produced by the test pathogens, further supporting its potential as an effective antibiofilm agent. In addition, combining repurposed compounds with conventional antibiotics is a valuable strategy for overcoming drug resistance. Accordingly, our study examined the potential synergistic effects of gabapentin in combination with ampicillin and rifampicin. The results revealed clear synergistic interactions, further supporting gabapentin's role as an effective adjunct to existing antimicrobial therapies. Similarly, a growing number of

studies have investigated the repurposing of existing drugs for antimicrobial applications. For example, hexestrol, a non-steroidal synthetic estrogen, was recently shown to possess strong antibacterial and antibiofilm activity against methicillin-resistant *Staphylococcus aureus* (MRSA), with an MIC of 16 µg/mL. It also significantly reduced biofilm formation and displayed synergistic interactions with conventional antibiotics.[25] Likewise, selamectin demonstrated antibacterial activity against *S. aureus*, including noticeable alterations in cell morphology following treatment. It also inhibited biofilm formation and acted synergistically with ampicillin.[26] Another repurposed drug, paroxetine an antidepressant was tested against both methicillin-sensitive and MRSA. It exhibited antibacterial activity with an MIC of 64 µg/mL and showed additive effects in combination with oxacillin, with morphological damage observed as part of its mechanism of action.[27] Similarly, amlodipine displayed *in vitro* antibacterial activity against *S. aureus* with an MIC of 128 µg/mL. It also exhibited synergy with oxacillin and demonstrated the ability to disrupt mature biofilms, likely through membrane damage leading to cell death.[28] Furthermore, the antimalarial drug amodiaquine has been tested against two ESKAPE pathogens, *S. aureus* and *P. aeruginosa*, showing notable antibacterial activity with MIC values of 50 µg/mL and 100 µg/mL, respectively, along with synergistic effects when combined with ciprofloxacin.[29] Overall, gabapentin shows promise as both an antibacterial and antibiofilm agent against clinically relevant pathogens.

CONCLUSION

This study assessed the antibacterial and antibiofilm potential of gabapentin against *S. aureus* and *P. aeruginosa*, major pathogens implicated in pediatric CF airway infections. Gabapentin demonstrated significant antibacterial activity, effectively inhibiting the growth of both organisms. Notably, it suppressed biofilm formation on abiotic surfaces and disrupted mature, established biofilms, which are central to persistent and recurrent infections in CF patients. Combination assays further revealed that gabapentin enhanced the effectiveness of commonly used antibiotics, suggesting synergistic interactions that could improve therapeutic outcomes. In addition, gabapentin exhibited antioxidant properties, which may help mitigate oxidative stress associated with chronic airway infections. These findings support the potential repurposing of gabapentin as a multifunctional agent for managing biofilm-mediated, multidrug-resistant infections in pediatric CF, offering a promising adjunct to conventional antimicrobial therapy.

CONFLICTS OF INTEREST

The author declares no conflicts of interest relevant to this article.

ACKNOWLEDGMENT

The authors are grateful to the Deanship of Scientific Research, Prince Sattam bin Abdulaziz University, Al-Kharj, Saudi Arabia, for its support and encouragement in conducting the research and publishing this report.

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Source of Support: Nil. **Conflicts of Interest:** None declared.