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Judul Artikel	: Attenuation of alginate lyase by chlorhexidine in the alginate pathway of Pseudomonas aeruginosa biofilm formation
Jurnal	: Journal of Advanced Pharmacy Education & Research (JAPER)
Penulis	: Mohammad Subkhan , Sukardiman Sukardiman , Isnin Anang Marhana , Laily Irfana

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2	Bukti revisi minor	1 Desember 2024
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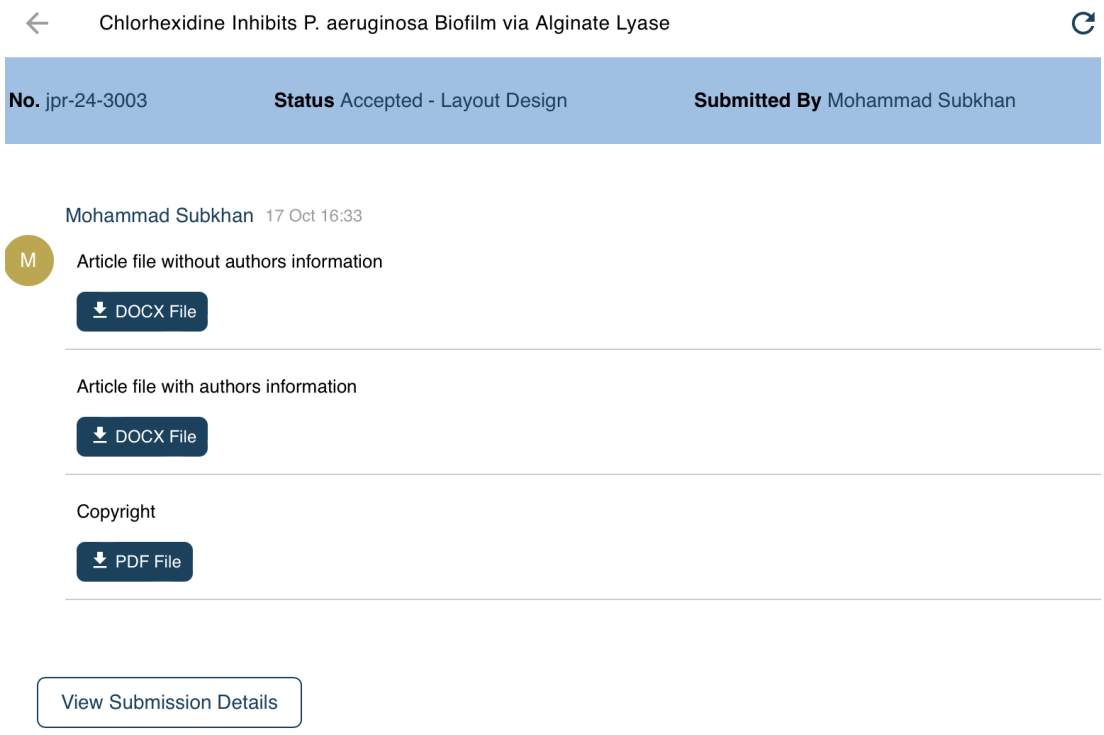

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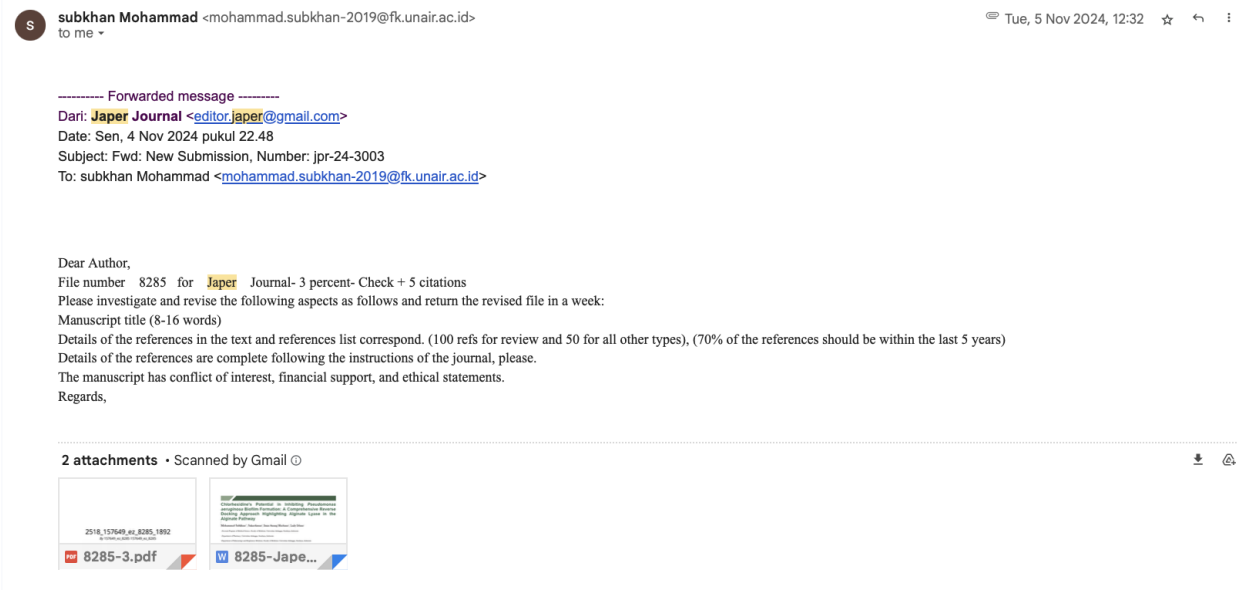

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Dear editor of JAPER,

Thank you for your feedback on my manuscript. I have carefully revised the sections addressing the conflict of interest, financial support, and ethical statements as requested.

Please find attached the revised manuscript for your review.

Should you require any further clarification or additional adjustments, please do not hesitate to contact me. I appreciate your guidance throughout this process.

Thank you for your time and consideration.

Best regards,
Mohammad Subkhan
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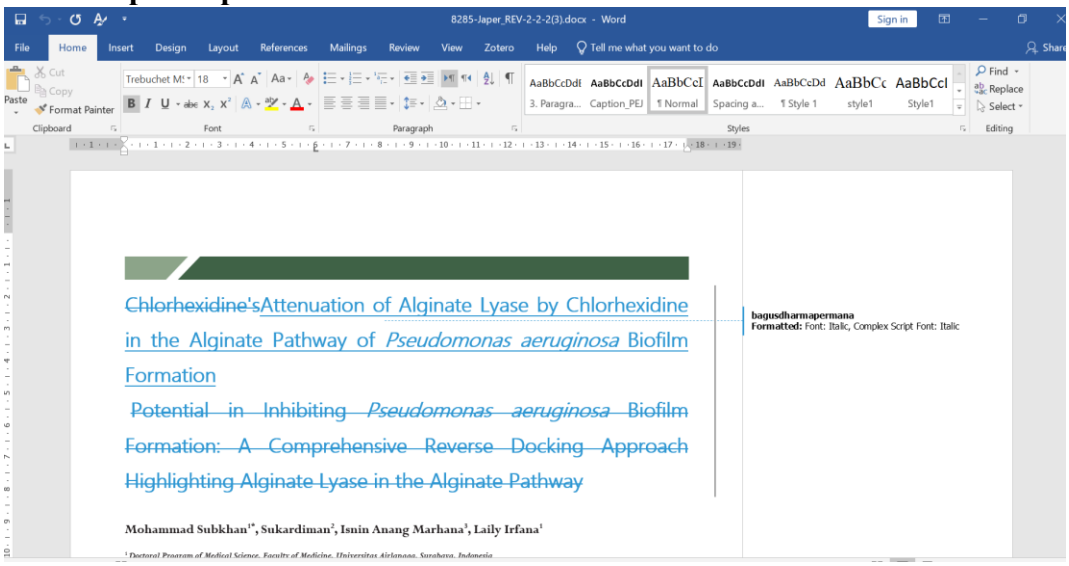

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Dear Authors:

Mohammad Subkhan*, Sukardiman, Isnin Anang Marhana, Laily Irfana

I am pleased to inform you that, your peer reviewed revised article entitled: "Chlorhexidine's potential in inhibiting *Pseudomonas aeruginosa* biofilm formation: A Comprehensive reverse docking approach highlighting alginate lyase in the alginate pathway" is now accepted for publication in (Volume 15, 1) of the Journal of Advanced Pharmacy Education & Research [JAPER].

Thank you for considering JAPER Journal for publication of your valuable work and all your cooperation with the review and publication process.

I look forward to publish your upcoming articles in JAPER and wish you great success in your career endeavor.

Please do not hesitate to contact us if you have any further questions.

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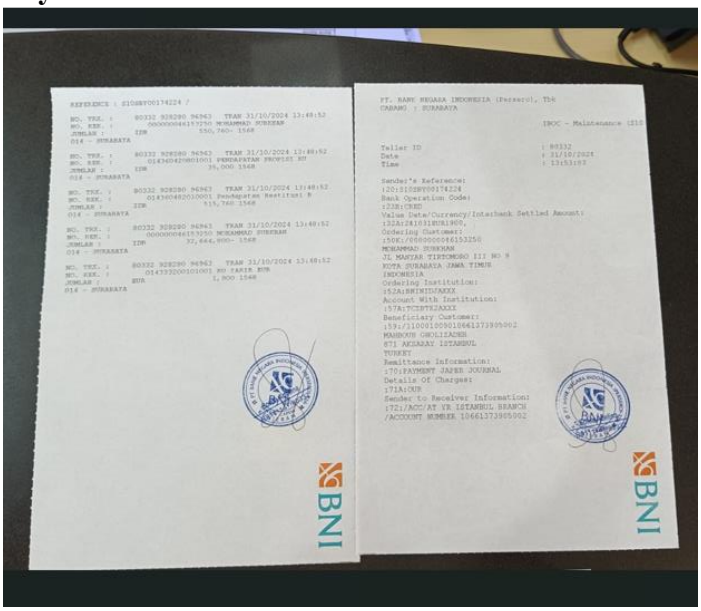

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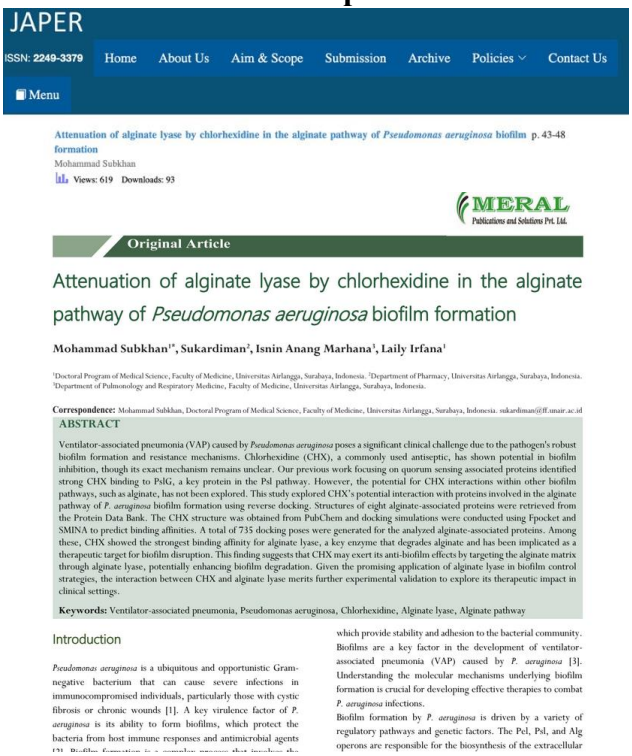

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Attenuation of alginate lyase by chlorhexidine in the alginate pathway of *Pseudomonas aeruginosa* biofilm formation p. 43-48

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Original Article

Attenuation of alginate lyase by chlorhexidine in the alginate pathway of *Pseudomonas aeruginosa* biofilm formation

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ABSTRACT

Ventilator-associated pneumonia (VAP) caused by *Pseudomonas aeruginosa* poses a significant clinical challenge due to the pathogen's robust biofilm formation and resistance mechanisms. Chlorhexidine (CHX), a commonly used antiseptic, has shown potential in biofilm inhibition, though its exact mechanism remains unclear. Our previous work focusing on quorum sensing associated proteins identified strong CHX binding to PslG, a key protein in the Psl pathway. However, the potential for CHX interactions within other biofilm pathways, such as alginate, has not been explored. This study explored CHX's potential interaction with proteins involved in the alginate pathway of *P. aeruginosa* biofilm formation using reverse docking. Structures of eight alginate-associated proteins were retrieved from the Protein Data Bank. The CHX structure was obtained from PubChem and docking simulations were conducted using Fpocket and SMINA to predict binding affinities. A total of 735 docking poses were generated for the analyzed alginate-associated proteins. Among these, CHX showed the strongest binding affinity for alginate lyase, a key enzyme that degrades alginate and has been implicated as a therapeutic target for biofilm disruption. This finding suggests that CHX may exert its anti-biofilm effects by targeting the alginate matrix through alginate lyase, potentially enhancing biofilm degradation. Given the promising application of alginate lyase in biofilm control strategies, the interaction between CHX and alginate lyase merits further experimental validation to explore its therapeutic impact in clinical settings.

Keywords: Ventilator-associated pneumonia, Pseudomonas aeruginosa, Chlorhexidine, Alginate lyase, Alginate pathway

Introduction

Pseudomonas aeruginosa is a ubiquitous and opportunistic Gram-negative bacterium that can cause severe infections in immunocompromised individuals, particularly those with cystic fibrosis or chronic wounds [1]. A key virulence factor of *P. aeruginosa* is its ability to form biofilms, which protect the bacteria from host immune responses and antimicrobial agents which provide stability and adhesion to the bacterial community. Biofilms are a key factor in the development of ventilator-associated pneumonia (VAP) caused by *P. aeruginosa* [3]. Understanding the molecular mechanisms underlying biofilm formation is crucial for developing effective therapies to combat *P. aeruginosa* infections.

Biofilm formation by *P. aeruginosa* is driven by a variety of regulatory pathways and genetic factors. The Pel, Psl, and Alg operons are responsible for the biosynthesis of the extracellular