J Med Biosci Suppl 2025;7: S149 - S156 DOI: 10.58837/CHULA.JMBS.7.S1.21

Original article

Immunoinformatic profiling of AdeR unveils novel diagnostic and therapeutic targets against multidrug-resistant *Acinetobacter baumannii*

Jindaporn Deeto[1], Siriphan Boonsilp[2], Worrapoj Oonanant[3], Duangtip Moonmangmee[1], Apisitt Thaiprayoon[4], Phoomintara Longsompurana[4], Jirayut Euanoraseth[1], Dujduan Waraho[4,*]

[1]*Department of Microbiology, Faculty of Science, King Mongkut's University of Technology Thonburi, Bangkok, Thailand*
[2]*Department of Basic Medical Science, Faculty of Medicine Vajira Hospital, Navamindradhiraj University, Dusit, Bangkok, Thailand*
[3]*Department of Clinical Pathology, Faculty of Medicine Vajira Hospital, Navamindradhiraj University, Dusit, Bangkok, Thailand*
[4]*Biological Engineering Program, Faculty of Engineering, King Mongkut's University of Technology Thonburi, Bangkok, Thailand*

Abstract

Background: Multidrug-resistance *A. baumannii* is a Gram-negative, opportunistic pathogen and a major contributor to the global healthcare crisis, particularly in nosocomial infections.

Objective: To identify conserved epitopes in AdeR protein of MDR-*Acinetobacter baumannii* for the development of antibody-based sensors for early detection and intervention.

Methods: The AdeR gene from four MDR-*A. baumannii* isolates was cloned into the pET21a vector, sequenced, and aligned using BioEdit. B-cell and T-cell epitope predictions were made using the IEDB servers. The three-dimensional structure of AdeR was modeled using the Swiss-Model server, with molecular visualization performed in PyMOL.

Results: The analysis of AdeR sequences from four MDR-*A. baumannii* isolates revealed a high degree of conservation, particularly in the N-terminal domain, and identified key epitopes for immune recognition. The three-dimensional structures of AdeR were identical across isolates, despite minor sequence variations. B- and T-cell epitope predictions showed strong conservation, supporting the potential of AdeR as a target for diagnostic tools. These findings suggest that antibodies targeting AdeR could provide an effective candidate for detecting MDR infections. Finally, AdeR's conserved structure and immunogenic regions make it a promising candidate for early detection and management of MDR-*A. baumannii* infections.

Conclusion: The conserved structure and immune recognition sites of AdeR across MDR-*A. baumannii* isolates make it a promising target for antibody-based diagnostic tools. This approach could enable early detection and more effective management of MDR infections.

Keywords: *Acinetobacter baumannii*, AdeR, immunoinformatic profiling, molecular modeling.

*Correspondence to: Dujduan Waraho, Biological Engineering Program, Faculty of Engineering, King Mongkut's University of Technology Thonburi, Bangkok 10800, Thailand.
E-mail: Caramal12345678@gmail.com, Dujduan.war@mail.kmutt.ac.th

Open Access 2025 Deeto et al ., published by Faculty of Medicine, Chulalongkorn University. This work is licensed under the Creative Commons Attribution 4.0 International License.

Acinetobacter baumannii (*A. baumannii*) is a Gram-negative, opportunistic pathogen and a major contributor to the global healthcare crisis, particularly in nosocomial infections. It is a key member of the ESKAPE pathogen group, responsible for a significant proportion of multidrug-resistant (MDR) infections worldwide.[1] The rise of MDR strains of *A. baumannii*, resistant to multiple antibiotic classes, leads to millions of infections annually and imposes an estimated economic burden of $742 million in healthcare costs.[2] *A. baumannii* exhibits resistance to a broad spectrum of antibiotics, including aminoglycosides, tetracyclines, fluoroquinolones, chloramphenicol, tigecycline, and colistin, substantially limiting available treatment options.[3] The mechanisms underlying resistance and virulence in *A. baumannii* include overexpression of efflux pumps, mutations in porins, â-lactamase *production*, biofilm formation, and horizontal gene transfer.[4] Among the major contributors to multidrug resistance in *A. baumannii* are the resistance-nodulation-division (RND) efflux pumps, specifically AdeABC, AdeFGH, and AdeIJK.[4] The expression of the AdeABC efflux pump is tightly regulated by the AdeRS two-component regulatory system (TCS).[5] AdeR (Acinetobacter drug efflux Regulator) is a response regulator protein in *A. baumannii* that, once activated by its partner protein AdeS (Acinetobacter drug efflux pump sensor), promotes the expression of the AdeABC efflux pump. This pump contributes to multidrug resistance by actively expelling antibiotics from the bacterial cell. Mutations in the *adeRS* genes, which encode the sensor kinase AdeS and the response regulator AdeR, have been associated with overproduction of the AdeABC pump and reduced susceptibility to antimicrobial agents. The AdeRS system functions by detecting environmental signals through the histidine kinase AdeS, which then phosphorylates AdeR. Once phosphorylated, AdeR upregulates target genes, including those involved in efflux pump production.[6,7] In addition to regulating the AdeABC efflux pump, AdeRS has been implicated in the regulation of biofilm formation and virulence factors, although this regulation appears to be strain-specific. Given the critical role of the AdeR response regulator in antimicrobial resistance, it is considered a promising target for the development of novel therapeutic strategies. The AdeR protein consists of two main domains: a CheY-like receiver domain (amino acids 1–127) and an OmpR/PhoB-type DNA-binding domain (amino acids 138–247). Unlike typical OmpR/PhoB-type response regulators, which bind to the promoter regions of their target genes, AdeR specifically recognizes a 10-base-pair perfect direct-repeat DNA sequence located in the intercistronic region between adeR and adeABC.[8-10] This study investigated the three-dimensional structures of AdeR proteins from four clinical isolates of MDR-*A. baumannii* to assess their similarities and potential as antigenic targets. Although previous research has focused on proteins such as Psfr, LptE, SerA, OmpH, CarO, FimF, CsuB, MlaA, the AdeABC efflux pump, and OmpA, the antigenicity of AdeR has not yet been explored.[11] Structural comparison revealed a high degree of conservation among isolates, particularly in regions associated with B- and T-cell epitope recognition. These findings suggest that AdeR could serve as a novel candidate for vaccine development and antibody-based diagnostic development. Furthermore, conserved antigenic regions highlight its potential application in biosensors for broad-spectrum detection and early diagnosis of MDR-*A. baumannii* infections. To our knowledge, this is the first study to predict the antigenicity of AdeR among clinical isolates, offering new strategies for intervention, management, and diagnostic innovation.

Materials and Methods

Bacterial strains

The mature AdeR of *A. baumannii* 19606 strain, a non-drug-resistant strain, was obtained from ATCC. Two genonic DNA of multidrug-resistant clinical isolates, H1074 and R560, were derived from blood and urine samples, respectively, of patients in the ICU at Vajira Hospital, Thailand.[12,13] The AdeR from the Uniprot database with ID E1A0Z5· E1A0Z5_ACIBA, which corresponds to a Carbarpenem-resistant bacterium isolated by Cologne University hospital in Cologne, Germany was used to campare the amino acid sequemce variability to Thai clinical isolates.

AdeR amino acid sequence alignment

The AdeR gene fragment was amplified using specific primers (AdeR FW: 5'GCGATGCCATGG ATGTTTGATCATTCTTTTTCTTTTGATTGC-3', AdeR RW: 5'GCGATGGTCGACGGCGTCATCTT TTACAGC-3') and then cloned into the pET21a vector at the NcoI and SalI restriction sites. The recombinant vectors containing the AdeR variants were subsequently sent for DNA sequencing. The resulting AdeR sequences were aligned using the BioEdit program for further analysis.

B-cell and T-cell epitope predictions

The potential for AdeR to be recognized by B- and T-cells was evaluated by predicting linear epitopes using the B-cell recognition server (http://tools.iedb.org/bcell/)(14) and assessing peptide binding to MHC class I molecules for T-cell recognition using the MHC class I binding server (http://tools.iedb.org/mhci/). (15)

Molecular modeling and protein three-dimensional structure analysis

All AdeR sequences were submitted to the Swiss-Model server (https://swissmodel.expasy.org) to generate the three-dimensional structure of AdeR, using model A0A8B4N001.1.A as a template for structure prediction. The molecular labeling was performed using the PyMOL modeling program, version 2.5. (16)

Results

Comparison of AdeR amino acid sequences among four clinical isolates of MDR-A. baumannii

To investigate the similarity of the AdeR amino acid sequences, the sequences from four clinical isolates of MDR-*A. baumannii* were compared using the BioEdit program, as illustrated in **Figure 1**. The sequence alignment analysis revealed that the similarity between the AdeR proteins from the clinical isolates 19606 and H1074, 19606 and G560, H1074 and G560, 19606 and EZA10ZA, H1074 and EZA10ZA, and G560 and EZA10ZA isolates, were 98.79%, 98.79%, 98.38%, 98.78%, 98.78%, and 99.59% respectively. The AdeR protein comprises two key functional domains: the response regulator domain (amino acids 1-127) and the DNA-binding domain (amino acids 138-247).(8, 9) The comparison of these sequences identified that most of the amino acid substitutions were located within the DNA-binding domain, while only 1 substitution was present in the response regulator domain. This suggests that the N-terminal helix of the protein, which is part of the response regulator domain, is highly conserved across the isolates. In addition to the amino acid alignment, the three-dimensional structures of the AdeR proteins from all four clinical isolates showed identical structural configurations, as illustrated in **Figure 2**.

Figure 1. Amino acid sequence alignment of AdeR protein among four clinical isolates of *A. baumannii*. The alignment compares the AdeR sequences from isolates 19606, H1074, G560, and E1A0Z5, highlighting the conserved and variable regions across the four variants. The figure illustrates the degree of sequence similarity and identifies key amino acid substitutions or conserved motifs within the response regulator and DNA-binding domains of AdeR. The red and green boxes covers response regulator and DNA-binding domain respectively.

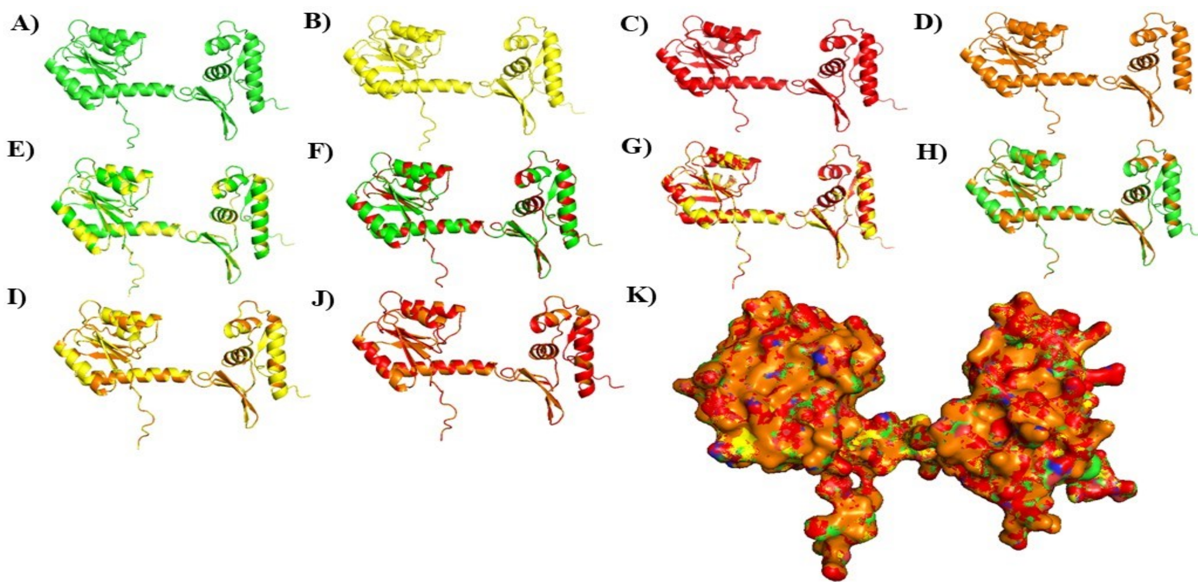

Figure 2. The three-dimensional structures of four variants of AdeR protein from Thailand clinical isolates MDR-*A. baumannii*. The structures of AdeR from isolates (**A**) 19606, (**B**) H1074, (**C**) G560, (**D**) E1A0Z5. The structural comparisons of isolates (**E**) 19606 and H1074, (**F**) 19606 and G560, (**G**) H1074 and G560, (**H**) 19606 and E1A0Z5, (**I**) H1074 and E1A0Z5, (**J**) G560 and E1A0Z5 (**K**). The comparison among four clinical isolates 19606, H1074, G560, and E1A0Z5 presented with surfaced-globular configurations.

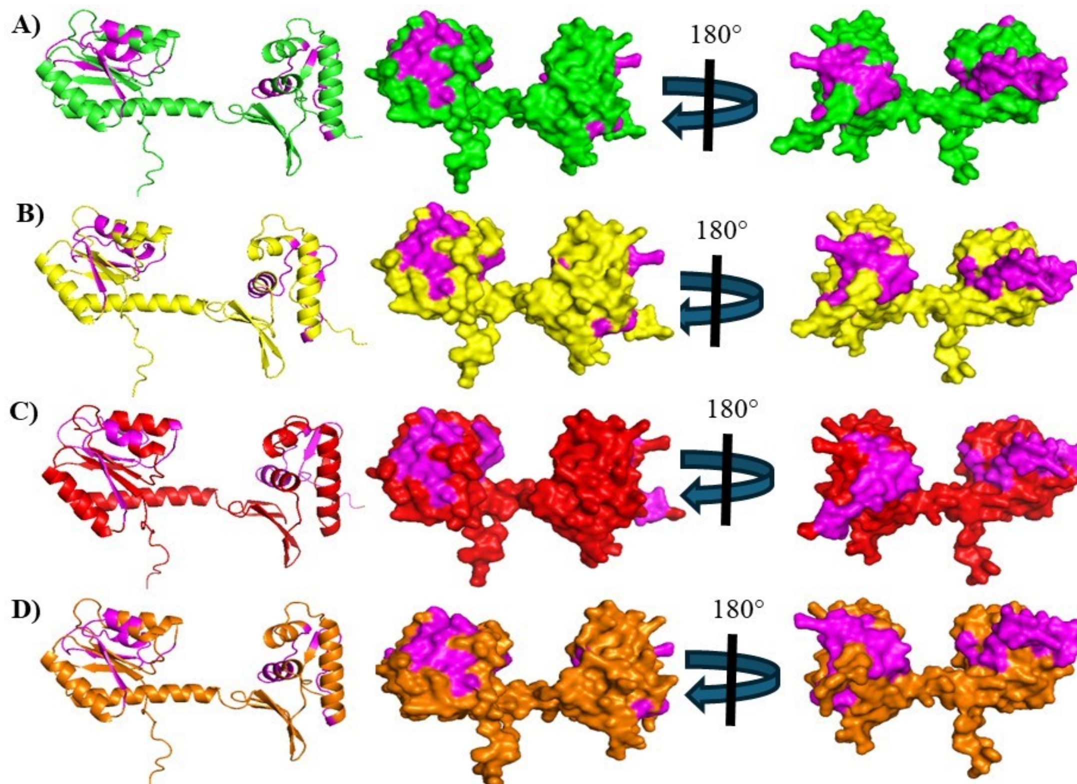

Figure 3. The T-cell epitope regions presented on the AdeR protein were analyzed among MDR- *A. baumannii* isolates. The AdeR protein structures were modeled from the following strains: (**A**) 19606, (**B**) H1074, (**C**) G560 and (**D**) E1A0Z5. The magenta regions indicate the locations of B-cell epitopes.

Table 1. Conservation of T-cell Epitopes on AdeR across Four Clinical Isolates of *A. baumannii.*

Core amino acid	residues	score	MDR-*A. baumannii*			
			19606	H1074	G560	E1A0Z5
MSVIRAMNGKQAIE	37-50	0.9093	+	+	+	+
QGIFQMLINVRGVG	221-234	0.8550	+	+	-	-
GVGYRLDNPLAVKDD	232-246	0.8175	-	-	+	+
DQDIDKVMALRIGAD	93-107	0.7621	+	-	+	+
LDQDIDKVMALRIGAD	92-107	0.7076	-	+	-	-
IISFMIDQPHKVFTR	175-189	0.6779	+	-	+	+
KIISFMIDQPHKVFTR	174-189	0.6258	-	+	-	-
QTPVIMLTALDQDID	83-97	0.4730	+	+	+	+

The prediction of T-cell epitopes localized on four isolates of MDR-A. baumannii AdeR structures

To evaluate the potential for the human immune system to recognize AdeR, we predicted the location of T-cell epitopes on the three-dimensional structure of AdeR proteins using IEDB T-cell epitope prediction server, as presented in **Table 1.** T-cell epitopes are regions of an antigen that are recognized by the T-cell receptor (TCR) on the surface of T-cells. When the TCR binds to a T-cell epitope on an antigen, it activates naïve T-cells, which then differentiate into cytotoxic T-cells. These cytotoxic T-cells play a crucial role in immune defense by destroying foreign substances through the secretion of hydrolytic enzymes. The structural analysis of the T-cell epitopes on the AdeR protein revealed conserved regions among the four MDR-*A. baumannii* isolates. The T-cell epitopes shared core sequence similarities, including the sequences MSVIRAMNGKQAIE, DQDIDKVMALRIGAD, IISFMIDQPHKVFTR, and QTPVIMLTALDQDID. These epitopes are distributed across both the N- and C-terminal regions of the AdeR proteins (**Figure 3**). Notably, when comparing the amino acid sequences from the previous analysis, the N-terminal helix emerges as a particularly promising target for AdeR protein detection. This region is highly conserved, containing both conserved amino acid sequences and a similarity in structural configuration. Additionally, it is the area that harbors the conserved T-cell epitopes across all four clinical isolates of MDR-*A. baumannii*, making it an appealing candidate for immune targeting.

The prediction of B cell epitopes presented on four species of MDR-A. baumannii AdeR structure

To assess the potential for the AdeR protein to be recognized by antibodies, the amino acid sequences of all four AdeR variants were analyzed using a B-cell epitope prediction program to identify the locations of B-cell epitopes on the AdeR structure. B-cell epitopes are specific regions on an antigen that are recognized by the B-cell receptor (BCR) on the surface of naïve B-cells. The interaction between the BCR and the B-cell epitope triggers a signaling cascade that leads to the differentiation of naïve B-cells into plasma cells. [17,18] These plasma cells are responsible for producing antibodies, which play a crucial role in immune defense by eliminating specific antigens through mechanisms such as: 1) opsonization, 2) antigen neutralization and precipitation, and 3) antibody-dependent cellular cytotoxicity (ADCC).[19-20] The results of the B-cell epitope prediction indicated that the B-cell epitopes for all AdeR variants are highly conserved, particularly in the N- and C-terminal domains **(Figure 4).** Notably, the DNA-binding domain of AdeR exhibited the highest antigenicity, suggesting that this region is particularly prone to antibody recognition. These findings imply that mutations in the amino acid residues across the four AdeR variants have minimal impact on the B-cell epitope regions. This may be due to the mutations occurring in positions where amino acids with similar properties are substituted, resulting in only slight changes in the interactions between amino acids within the three-dimensional structure of AdeR. As a result, the mutations in the AdeR structure appear insufficient to significantly alter its overall shape or antigenicity, as evidenced by the high structural similarity between the N- and C-terminal helices, as well as the overlap of B- and T-cell epitope positions.

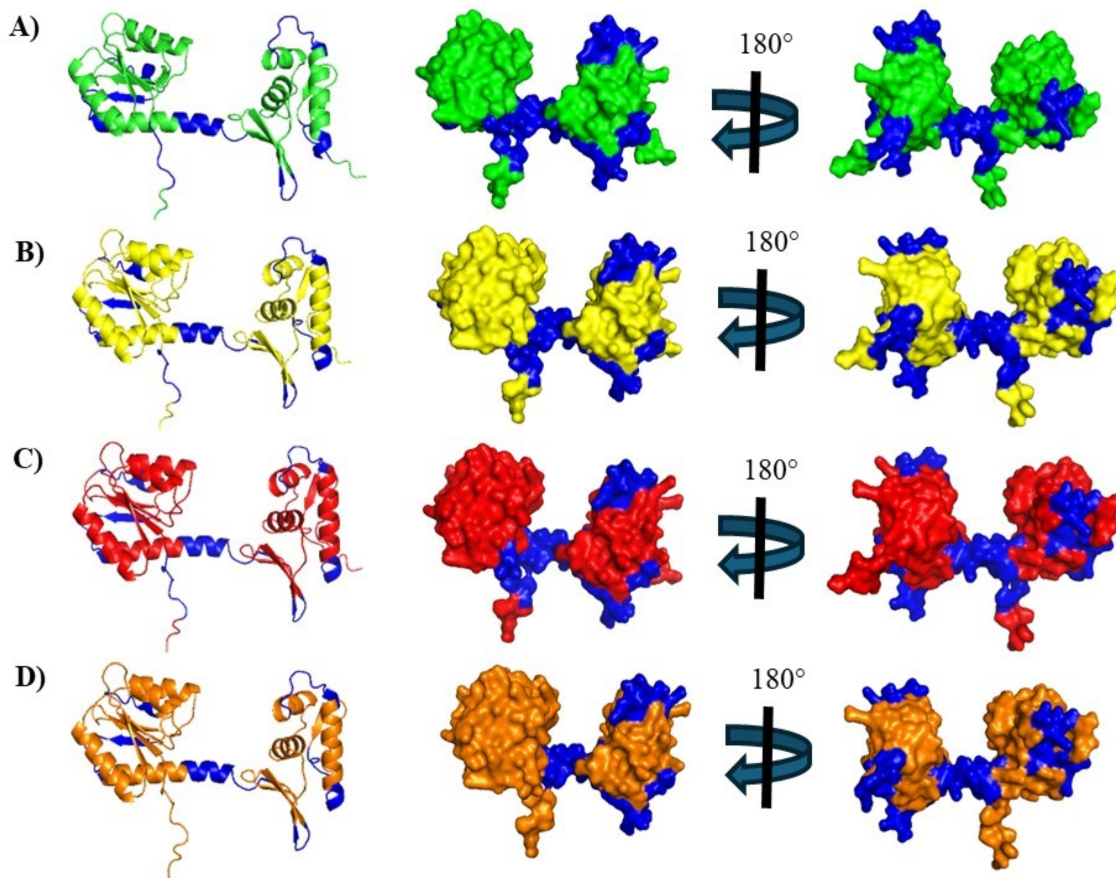

Figure 4. The B-cell epitope regions presented on the AdeR protein were analyzed among MDR- *A. baumannii* isolates. The AdeR protein structures were modeled from the following strains: (**A**) 19606, (**B**) H1074, (**C**) G560, and (**D**) E1A0Z5. The blue regions indicate the locations of T-cell epitopes.

Discussion

The comparative analysis of AdeR amino acid sequences from four clinical isolates of MDR-*A. baumannii* demonstrated a high level of conservation across the isolates. The AdeR proteins from the Thai and German clinical isolates showed 98% identity in their primary structure and exhibited similar tertiary structures. Furthermore, the T-cell and B-cell epitopes were found to be highly comparable between the Thai and German isolates. Several putative proteins from MDR-*A. baumannii* including the putative ferric siderophore receptor protein (Psfr), lipopolysaccharide transport protein E (LptE), surface antigen protein (SerA), outer membrane protein H (OmpH), carbapenem resistance outer membrane protein (CarO), fimbrial protein F (FimF), pilus protein (CsuB), putative phospholipid-binding lipoprotein MlaA, the AdeABC efflux pump, and outer membrane protein A (OmpA) have previously been investigated for B- and T-cell epitope prediction in the search for suitable antigenic targets.[12] However, to date, no studies have reported the prediction of antigenic epitopes for the AdeR protein. Here, we present, for the first time, an in-depth prediction and analysis of the antigenicity of the AdeR protein among MDR-*A. baumannii* clinical isolates, highlighting its potential as a novel vaccine and antibody-based diagnostic development candidate. The AdeR protein is composed of two key functional domains including the response regulator and the DNA-binding domain. Most of the amino acid substitutions were localized within the DNA-binding domain, while only a few changes were observed in the response regulator domain, emphasizing the conservation of the N-terminal helix. Additionally, the three-dimensional structures of the AdeR proteins from all isolates were identical, indicating that the overall structure remains consistent despite minor sequence variations. The analysis of B- and T-cell epitopes further supports the potential of AdeR as a target for immune recognition. Both B- and T-cell epitopes were found to be highly conserved across isolates, particularly within the N- and C-terminal regions, with a conservation threshold set above 0.5. In epitope prediction, the threshold score serves as a predefined cutoff to differentiate potential epitope

regions from non-epitopic sequences based on their predicted likelihood of antibody recognition. This threshold is typically determined through statistical modeling or machine learning approaches trained on experimentally validated datasets where residues scoring above 0.5 are predicted epitopes, making them candidates for further immunological validation. [21] Furthermore, the mutations observed across the isolates appear to have little effect on the immunogenic regions, suggesting that antibodies targeting AdeR would be effective in detecting MDR-*A. baumannii* infections. The stability of these epitopes, even across different clinical strains, ensures that any diagnostic tools developed to target AdeR will likely be robust and reliable. AdeR presents strong potential as a diagnostic target for the early detection and management of MDR-*A. baumannii* infections. Its conserved structure and predicted immune recognition sites make it a promising candidate for antibody-based detection assays, which could enhance clinical outcomes by enabling rapid and accurate diagnosis. However, a major limitation of B- and T-cell epitope prediction lies in their reliance on in silico models, which may not fully capture the complexity of immune responses in vivo. Predicted epitopes may demonstrate low immunogenicity or fail to elicit protective immunity when evaluated experimentally. [22] Additionally, current prediction tools are often biased toward well-characterized pathogens, potentially limiting their predictive accuracy for novel targets like AdeR. Therefore, experimental validation through approaches such as peptide-based ELISA, T-cell proliferation assays, and animal models is essential. [23,24] Future studies should focus on characterizing the immunogenicity of identified epitopes, evaluating the diagnostic performance of AdeR-specific antibodies in clinical specimens, and integrating AdeR-targeted assays into routine diagnostic workflows. Incorporating AdeR detection into rapid immunoassay platforms, such as lateral flow assay or point-of-care diagnostics, could significantly improve early identification and management of MDR-*A. baumannii* infections in clinical settings.

Conclusion

In this study, we conducted the first in-depth prediction and comparative analysis of the antigenic epitopes of the AdeR protein among clinical isolates of multidrug-resistant (MDR) *Acinetobacter baumannii*. Our findings revealed a high degree of conservation in both the amino acid sequences and three-dimensional structures of AdeR across isolates from different geographic origins. Notably, T-cell and B-cell epitopes were highly preserved, particularly within the N- and C-terminal regions, reinforcing AdeR's potential as a stable and reliable target for immune recognition. Despite minor sequence variations, the structural integrity and predicted immunogenic regions of AdeR remained unaffected, suggesting that antibodies against AdeR could serve as effective tools for the detection of MDR-*A. baumannii*. While in silico predictions provide valuable insights, experimental validation is critical to confirm the immunogenicity and diagnostic utility of the predicted epitopes. Future work should focus on verifying these findings through laboratory assays and developing AdeR-based rapid diagnostic platforms to enhance early detection and management of MDR-*A. baumannii* infections.

Acknowledgements

This research project is supported by King Mongkut's University of Technology Thonburi (KMUTT), Thailand Science Research and Innovation (TSRI), and National Science, Research and Innovation Fund (NSRF) Fiscal year 2025 Grant number FRB680074/0164 (Program Smart Healthcare) to D.W.-Z., and the Royal Golden Jubilee (RGJ) Ph.D. scholarship from the National Research Council of Thailand under the Ministry of Higher Education, Science, Research and Innovation (NRCT); NRCT5-RGJ63006 to A.T..

Conflicts of interest statement

The authors declare no conflict of interest. The funders had no role in the design of the study; in the collection, analyses, or interpretation of data; in the writing of the manuscript, or in the decision to publish the results.

Data sharing statement

The data underlying the findings are included in this published article. Further details are available upon reasonable request to the corresponding author.

References

1. Mulani MS, Kamble EE, Kumkar SN, Tawre MS, Pardesi KR. Emerging strategies to combat ESKAPE pathogens in the era of antimicrobial resistance: a review. Front Microbiol 2019;10:539.
2. De Oliveira DMP, Forde BM, Kidd TJ, Harris PNA, Schembri MA, Beatson SA, et al. Antimicrobial Resistance in ESKAPE Pathogens. Clin Microbiol Rev 2020 ;33:e00181-19.
3. Peleg AY, Seifert H, Paterson DL. Acinetobacter baumannii: emergence of a successful pathogen. Clin Microbiol Rev 2008;21:538-82.
4. Abdi SN, Ghotaslou R, Ganbarov K, Mobed A, Tanomand A, Yousefi M, et al. Acinetobacter baumannii Efflux Pumps and Antibiotic Resistance. Infect Drug Resist 2020;13:423-34.
5. Lin MF, Lin YY, Lan CY. The role of the two-component system BaeSR in disposing chemicals through regulating transporter systems in *Acinetobacter baumannii*. PLoS One 2015 ;10:e0132843.
6. Roy S, Junghare V, Dutta S, Hazra S, Basu S. Differential binding of carbapenems with the adeabc efflux pump and modulation of the expression of adeb linked to novel mutations within two-component system AdeRS in Carbapenem-resistant acinetobacter baumannii. mSystems 2022;7:e0021722.
7. Sharma S, Kaushik V, Kulshrestha M, Tiwari V. Different efflux pump systems in Acinetobacter baumannii and Their Role in Multidrug Resistance. Adv Exp Med Biol 2023;1370:155-68.
8. Lari AR, Ardebili A, Hashemi A. AdeR-AdeS mutations & overexpression of the AdeABC efflux system in ciprofloxacin-resistant Acinetobacter baumannii clinical isolates. Indian J Med Res 2018;147:413-21.
9. Wen Y, Ouyang Z, Yu Y, Zhou X, Pei Y, Devreese B, et al. Mechanistic insight into how multidrug resistant *Acinetobacter baumannii* response regulator AdeR recognizes an intercistronic region. Nucleic Acids Res 2017;45:9773-87.
10. Lucaßen K, Xanthopoulou K, Wille J, Wille T, Wen Y, Hua X, et al. Characterization of amino acid substitutions in the two-component regulatory system AdeRS identified in multidrug-resistant *Acinetobacter baumannii*. Msphere 2021;6:e00709-21.
11. Beiranvand S, Doosti A, Mirzaei SA. Putative novel B-cell vaccine candidates identified by reverse vaccinology and genomics approaches to control Acinetobacter baumannii serotypes. Infect Genet Evol 2021;96:105138.
12. Wongsuk T, Boonsilp S, Homkaew A, Thananon K, Oonanant W. Whole genome sequence of pan drug-resistant clinical isolate of Acinetobacter baumannii ST1890. PLoS One 2022;17:e0264374.
13. Boonsilp S, Homkaew A, Wongsuk T, Thananon K, Oonanant W. Correlation of RND efflux pump expression and AdeRS mutations in tigecycline-resistant Acinetobacter baumannii from Thai clinical isolates. Eur J Microbiol Immunol (Bp) 2024;14:280-8.
14. Ponomarenko J, Papangelopoulos N, Zajonc DM, Peters B, Sette A, Bourne PE. IEDB-3D: structural data within the immune epitope database. Nucleic acids Res 2010;39(suppl_1):D1164-70.
15. Mendes M, Mahita J, Blazeska N, Greenbaum J, Ha B, Wheeler K, et al. IEDB-3D 2.0: Structural data analysis within the Immune Epitope Database. Protein Sci 2023;32:e4605.
16. Schwede T, Kopp J, Guex N, Peitsch MC. SWISS-MODEL: An automated protein homology-modeling server. Nucleic Acids Res 2003;31:3381-5.
17. Parkin J, Cohen B. An overview of the immune system. The Lancet 2001;357:1777-89.
18. Mauri C, Bosma A. Immune regulatory function of B cells. Annu Rev Immunol 2012;30:221-41.
19. Wang W, Singh S, Zeng DL, King K, Nema S. Antibody structure, instability, and formulation. J Pharm Sci 2007;96:1-26.
20. Pieper K, Grimbacher B, Eibel H. B-cell biology and development. J Allergy Clin Immunol 2013;131:959-71.
21. Potocnakova L, Bhide M, Pulzova LB. An introduction to B-Cell epitope mapping and in silico epitope prediction. J Immunol Res 2016;2016:6760830.
22. Cia G, Pucci F, Rooman M. Critical review of conformational B-cell epitope prediction methods. Brief Bioinform 2023;24:bbac567.
23. Jespersen MC, Mahajan S, Peters B, Nielsen M, Marcatili P. Antibody specific B-cell epitope predictions: leveraging information from antibody-antigen protein complexes. Front Immunol 2019;10: 298.
24. Roggen EL. Recent developments with B-cell epitope identification for predictive studies. J Immunotoxicol 2006;3:137-49.