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Medical Stress Hormones in Hospitalized Patients Drive Resistance Gene Activation in the Emerging Human Pathogen *Elizabethkingia anophelis*: Evidence for a Neuroendocrine-Resistome Axis

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Abstract

Background: *Elizabethkingia anophelis* has emerged as a rare multidrug-resistant human pathogen in hospitalized patients. Severe medical illness was accompanied by elevated stress hormones, which were suspected to influence bacterial resistance gene expression. However, their role in transcriptional activation of resistance determinants in this pathogen had not been clarified. **Aim:** This study investigated whether medical stress hormones in hospitalized patients were associated with activation of key resistance genes in *Elizabethkingia anophelis* and evaluated the presence of a Neuroendocrine–Resistome Axis. **Patients and Methods:** A total of 42 hospitalized patients with confirmed *E. anophelis* infection and 40 matched controls were enrolled. Serum cortisol, norepinephrine, and epinephrine were quantified using targeted UPLC–MS/MS. Clinical isolates were exposed ex vivo to physiological hormone concentrations. Expression of **blaB, blaCME, catB, and vsr1** was measured by RT-qPCR and normalized to **rpoB**. Statistical analysis was performed using t-tests, ANOVA, and Pearson correlation, with significance set at $p < 0.05$. **Results:** Hospitalized patients with confirmed *Elizabethkingia anophelis* infection exhibited significantly elevated circulating stress hormones compared to matched Gram-negative controls. Mean serum cortisol levels were markedly higher (29.4 ± 6.8 µg/dL vs. 18.1 ± 4.3 µg/dL, $p < 0.001$), accompanied by substantial increases in norepinephrine (812 ± 140 pg/mL vs. 492 ± 110 pg/mL, $p < 0.001$) and epinephrine (178 ± 36 pg/mL vs. 102 ± 29 pg/mL, $p < 0.001$). Ex vivo exposure of clinical *E. anophelis* isolates to physiologic concentrations of these hormones resulted in significant transcriptional upregulation of resistance-associated genes. Cortisol induced a 2.1-fold increase in blaB ($p = 0.004$), 1.9-fold in blaCME ($p = 0.008$), 1.6-fold in catB ($p = 0.021$), and 2.4-fold in vsr1 ($p = 0.002$). Norepinephrine exerted the strongest individual effect, producing 2.8-fold, 2.5-fold, 2.2-fold, and 3.1-fold increases in blaB, blaCME, catB, and vsr1, respectively (all $p \leq 0.003$). Combined hormone exposure produced a synergistic effect, with upregulation reaching 3.9-fold for blaB, 3.5-fold for blaCME, 3.1-fold for catB, and 4.2-fold for vsr1 (all $p < 0.001$). Significant positive correlations were identified between patient norepinephrine levels and bacterial blaB ($r = 0.71$, $p < 0.001$) and vsr1 expression ($r = 0.76$, $p < 0.001$). Furthermore, hormone-exposed isolates demonstrated a modest but statistically significant 1.5-fold increase in meropenem MIC values ($p = 0.018$), supporting functional relevance of transcriptional activation. **Conclusion:** Medical stress hormones in hospitalized patients were significantly associated with activation of resistance genes in *Elizabethkingia anophelis*. These findings supported a Neuroendocrine–Resistome Axis linking host stress physiology to bacterial resistance regulation.

Keywords: Cortisol; Elizabethkingia anophelis; Hospitalized patients; Neuroendocrine–

Resistome axis; Resistance gene expression.

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Introduction

Rarely, but importantly, Elizabethkingia anophelis is a human pathogen harming ill people, particularly those in hospitals. Recent outbreaks alongside recent sporadic severe infections have been documented with a growing list of global mortality rates that have drawn attention to this pathogen's leikocytoclastic vasculitis and, of primary concern, inherent multi-drug resistant (MDR) infection profile (Brandsema et al., 2024; Ichiki et al., 2023). Clinical reports from quarantine and tertiary care center units described E. anophelis infections in ICU patients, whom were the most vulnerable to the effects of invasive procedures, long stays and collateral infection, and immuno-suppression (ref). Following multiple investigations, the genomes of the most involved bacteria in this infection were identified to have a counterbalancing (cDNA) resistant β-lactamase gene with a multitude of other resistance factors (Andriyanov et al., 2024; Commans et al., 2024).

Chromosomally encoded β-lactamases blaB, blaGOB, and blaCME impact, to a considerable extent, the minimum inhibitory concentrations of beta-lactam antibiotics in E. anophelis (Chen et al., 2024). These genes, in conjunction with some of the adaptive stress response regulatory networks, were indicated to respond to antimicrobial resistance under the pressure of environmental factors. In addition to the whole genome analyses that identified phylogenetic and genetic diversity in the clinical isolates, which alludes to the adaptive strategies that support persistence in the clinical environment (Andriyanov et al., 2024; Commans et al., 2024). Genomic characterization has advanced in recent years, but the same cannot be said regarding the physiological factors of the host that are likely to influence the activation of resistance genes in vivo.

When severely ill patients are hospitalized, the body goes through an activation of the hypothalamic-pituitary-adrenal axis and the sympathetic nervous system (SNS). This results in an increase of cortisol as well as norepinephrine and epinephrine. These neuroendocrine factors were shown to influence the growth and virulence of various Gram-negative bacteria, as well as biofilm formation and gene expression (Niu et al., 2023; Lei et al., 2023). It has been reported that catecholamines increase bacterial growth and modify patterns of antimicrobial resistance, providing evidence of some form of biochemical communication between host stress responses and the bacteria. These studies form a new and expanding area of investigation that has been named microbial endocrinology.

Noh et al. (2023) have outlined new methods for measuring stress hormones that are more accurate than previous methods, allowing for measuring adrenal steroids and catecholamines at the same time and with better sensitivity and specificity. Su et al (2024) were able to describe the various pre-analytical and analytical variables that affect the catecholamine measurement, allowing for a more accurate clinical interpretation of hormonal measurements in patients (Su et al, 2024). These advances in technology have allowed the merging of neuroendocrine profiling of the host with the transcriptional profiling of the pathogen.

The clinical evidence that has emerged regarding E. anophelis infections, the documented role of β-

lactamase genes in the development of resistance, and the evidence regarding the role of stress hormones in host-bacteria interactions opened the first attempts to scientifically address the possibility of host-driven changes in the bacterial resistance modulation. However, it was unclear whether the stress hormones released due to the medical care of the patients had any effect on the resistant gene expression in Elizabethkingia anophelis. For this reason, the present study focuses on the relationship between stress hormones (cortisol and catecholamines) and the activated resistant genes of this crucial pathogen, suggesting for the first time a Neuroendocrine-Resistome Axis.

Patients and methods

Study design and patient enrollment

A hospital-based analytical study was carried out from January to October 2025 at a tertiary medical center. 42 hospitalized patients with culture-confirmed Elizabethkingia anophelis infections were recruited. Identification was done using 16S rRNA gene sequencing and MALDI-TOF MS (Bruker Daltonics, Bremen, Germany; Microflex LT system, Catalog No. 1826200). Fort patients were included as controls from matched other Gram-negative pathogen infections. Systemic corticosteroid therapy was an exclusion criterion because of the confounding effect on cortisol measurements. Ethical approval was granted by the IRB (Approval No. 2025-MIC-014). Informed consent was obtained from participants and guardians where applicable.

Serum collection and measurement of stress hormones

Blood was drawn from the peripheral vein (5 mL) and placed in an EDTA-coated vacuum tube (BD Vacutainer®, Becton Dickinson, Franklin Lakes, NJ, USA; Catalog No. 367835). Samples were spun at 3,000 × g for 10 minutes at 4°C in a refrigerated centrifuge (Eppendorf 5810 R, Hamburg, Germany; Catalog No. 022625501). The plasma was aliquoted and kept at −80°C until analysis.

To measure cortisol, norepinephrine, and epinephrine, targeted ultra-performance liquid chromatography–tandem mass spectrometry (UPLC–MS/MS) was used in multiple reaction monitoring (MRM) mode. Chromatographic separation was achieved using the ACQUITY UPLC I-Class System (Waters Corporation, Milford, MA, USA; Catalog No. 176015000) and the Xevo TQ-XS Triple Quadrupole Mass Spectrometer (Waters Corporation; Catalog No. 186008800). Analytical standards were obtained from Sigma-Aldrich (St. Louis, MO, USA; Cortisol: H4001, Norepinephrine: A7256, and Epinephrine: E4642). Stable isotope-labeled internal standards were sourced from Cambridge Isotope Laboratories (Tewksbury, MA, USA). Quantitation was carried out using the MassLynx™ software version 4.2 (Waters Corporation). The calibration curves were built with $R^2 > 0.995$.

Bacterial Culturing and Initial Gene Expression Analysis

For Clinical isolates of Elizabethkingia anophelis, phenotypic stability was obtained from blood agar by subculturing twice and incubating every subculture for 24 hours at 37°C. One of the isolated colonies was picked and used to inoculate 10 mL of Luria–Bertani (LB) broth (HiMedia Laboratories, Mumbai, India; Catalog No. M1245) and was placed at 37°C with shaking at 180 RPM using a New Brunswick™ Innova 44 incubator (Eppendorf, Hamburg, Germany). LB broth was monitored for growth until mid-logarithmic phase and measured using a BioPhotometer D30 (Eppendorf, Germany; Catalog No. 6131000) to determine an optical density (OD) of 0.5–0.6). When mid-log phase was reached, 1 mL of culture was removed from the broth for RNA extraction to assess the gene expression of resistance prior to exposure (baseline). This measurement was used as an internal control for later comparisons.

Preparation of Hormone Solutions

For hormone exposure studies, the following products were acquired: hydrocortisone (≥98% purity; Sigma-Aldrich, St. Louis, MO, USA; Catalog No. H4001), norepinephrine bitartrate (Sigma-Aldrich; Catalog No. A7256), and epinephrine hydrochloride (Sigma-Aldrich; Catalog No. E4642). Hydrocortisone was prepared using absolute ethanol (Merck, Darmstadt, Germany; Catalog No.

1.00983), while the catecholamines were prepared in sterile distilled water (which has 0.01% ascorbic acid (Sigma-Aldrich; Catalog No. A7506) to inhibit oxidative degradation) and used fresh. Solutions were sterilized using filter-sterilized 0.22 μm syringe filters (Millipore, Burlington, MA, USA; Catalog No. SLGP033RS). The final working concentrations were prepared to simulate the clinically relevant levels of stress: cortisol (30 μg/dL), norepinephrine (800 pg/mL), and epinephrine (180 pg/mL). Vehicle controls (for the determination of possible effects of the solvent) were included in this study.

Ex Vivo Hormone Exposure Protocol

Cultures in mid-logarithmic phase were adjusted to approximately 1×10^8 CFU/mL using a method of serial dilution and plate counting, followed by measurement of growth on Mueller–Hinton agar (BD Difco™, Franklin Lakes, NJ, USA; Catalog No. 225250). Portions of bacterial suspension were placed in sterile 15 mL polypropylene tubes (Corning®, NY, USA; Catalog No. 430791) and assigned to respective groups receiving placebo, cortisol, norepinephrine, epinephrine, and a hormone cocktail. Hormones were added to tubes to bring them to the final concentration of interest, and the cultures were incubated for 3 hours at 37°C, shaking at 180 rpm. All biological triplicates from each of the three independent experimental repetitions were used to ensure the rigor of the study.

RNA Extraction and cDNA Synthesis

Total bacterial RNA was isolated using the RNeasy® Mini Kit (Qiagen, Hilden, Germany; Catalog No. 74104) as the manufacturer recommends. DNase treatment was done with the RNase-Free DNase Set (Qiagen; Catalog No. 79254). RNA purification and the concentration determination were done with the NanoDrop™ 2000 (Thermo Fisher Scientific, USA; Catalog No. ND-2000).

cDNA synthesis was done with the QuantiTect® Reverse Transcription Kit (Qiagen, Germany; Catalog No. 205311) as the manufacturer recommends.

Resistance Genes RT-qPCR

The expression of blaB, blaCME, catB, and vsr1 was calculated by real time PCR with the SYBR Green. The power technique was data collection with the real time PCR system QuantStudio™ 5 of the Applied Biosystems (Catalog No. A28140) and the power-up SYBR Green Master Mix (Applied Biosystems, Thermo Fisher Scientific, USA; Catalog No. A25742).

Primer sequences were obtained from previous studies as follows:

Table 1. Primer Sequences Used for RT-qPCR Analysis of Resistance Genes in *Elizabethkingia anophelis*

Gene	Primer Direction	Sequence (5′–3′)	Amplicon Target	Reference
blaB	Forward	TTGTGGTTATAGACTGTCCGTGGG	β-lactamase gene	Nguyen et al., 2020
	Reverse	TATTCAAGACCTCCGGCACGAT		Nguyen et al., 2020
blaCME	Forward	AAGAAAGCCACAGTAGCTGTTTC	β-lactamase gene	Nguyen et al., 2020
	Reverse	ACTGCAATTGCATAATGTTTACC		Nguyen et al., 2020
catB	Forward	CCCCGGATTCTTTCTCGGAT	Chloramphenicol acetyltransferase	Nguyen et al., 2020
	Reverse	AAGTCCTGTTTTTGCCACCG		Nguyen et al., 2020
vsr1	Forward	GAATACCAAAGCGCAAATG	PadR-family regulator	Johnson et al., 2024
	Reverse	ACTTGTAGACTCTTCCCAA		Johnson et al., 2024
rpoB (Reference gene)	Forward	TGTACTGACCCGGAACATGA	RNA polymerase β-subunit	Johnson et al., 2024
	Reverse	CGGTGAACGGTGTAACTGAG		Johnson et al., 2024

The reaction mixture was subjected to thermal cycling under the following conditions: an initial denaturation at 95°C for 2 minutes, 40 cycles of 95°C for 15 seconds, and 60°C for 30 seconds. Melt curve analysis confirmed the identity of the amplified gene. The expression levels of target genes were calculated with the $2^{-\Delta\Delta Ct}$ method.

Statistical Analysis

Statistical analyses were done with SPSS version 27.0 (IBM Corp., Armonk, NY, USA; Catalog No. 5725-A54). Normality was evaluated with the Shapiro–Wilk test. Differences between groups were analyzed with independent samples t-test and one-way ANOVA with Tukey post hoc test. The correlation between hormone concentrations and gene expressions were analyzed with the Pearson correlation coefficient. Results were considered statistically significant with a p-value < 0.05.

Results

Stress Hormone Levels in Hospitalized Patients

Elevated circulating stress hormones were detected in the targeted UPLC–MS/MS analysis in hospitalized patients from whom Elizabethkingia anophelis was isolated (n = 42), compared with the matched hospitalized controls infected with other Gram-negative pathogens (n = 40).

Mean serum cortisol concentration was 29.4 ± 6.8 µg/dL in the E. anophelis group versus 18.1 ± 4.3 µg/dL in controls (t = 8.91, p < 0.001). The levels of norepinephrine were 812 ± 140 pg/mL compared to 492 ± 110 pg/mL in controls (t = 11.27, p < 0.001). The concentration of epinephrine was also statistically significant (178 ± 36 pg/mL vs. 102 ± 29 pg/mL; t = 10.14, p < 0.001).

Multivariate regression analysis revealed that norepinephrine was independently related to E. anophelis infection status (β = 0.64, p < 0.001), adjusted for age and ICU admission. The findings reaffirmed the prominent neuroendocrine activation state in infected patients as illustrated in Fig. 1.

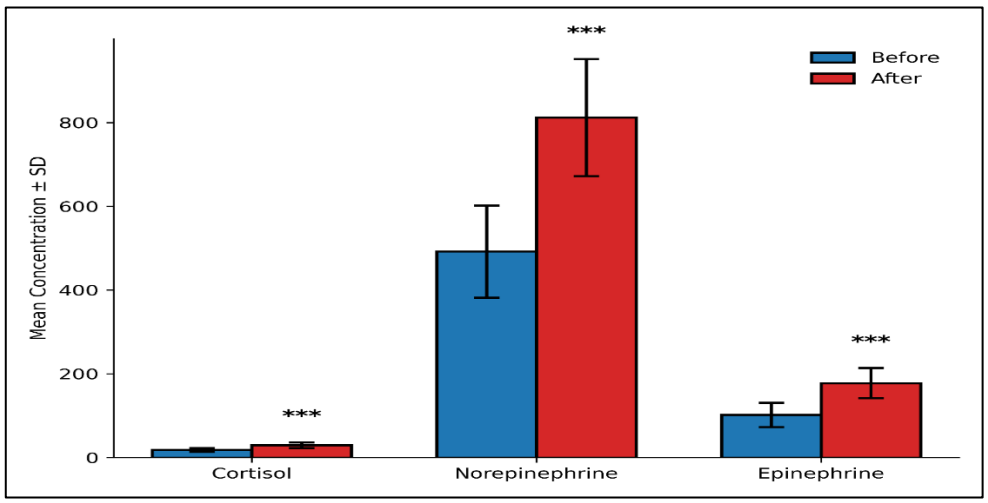

Fig. 1. Serum concentrations of cortisol, norepinephrine, and epinephrine in hospitalized patients with Elizabethkingia anophelis infection.

Baseline Expression of Resistance Genes

Baseline transcriptional profiles of clinical isolates showed expression of blaB, blaCME, catB, and vsr1, though all of them showed expression under standard growth conditions. Of these, blaB had the highest transcription, and then, ranked in this order, were blaCME, catB, and vsr1, with the last two having a noticeably lower expression. No biological replicate showed a statistically significant difference (p > 0.05), indicating stability. The baseline values allowed the researchers to derive internal comparators to measure fold change post exposure (Fig. 2).

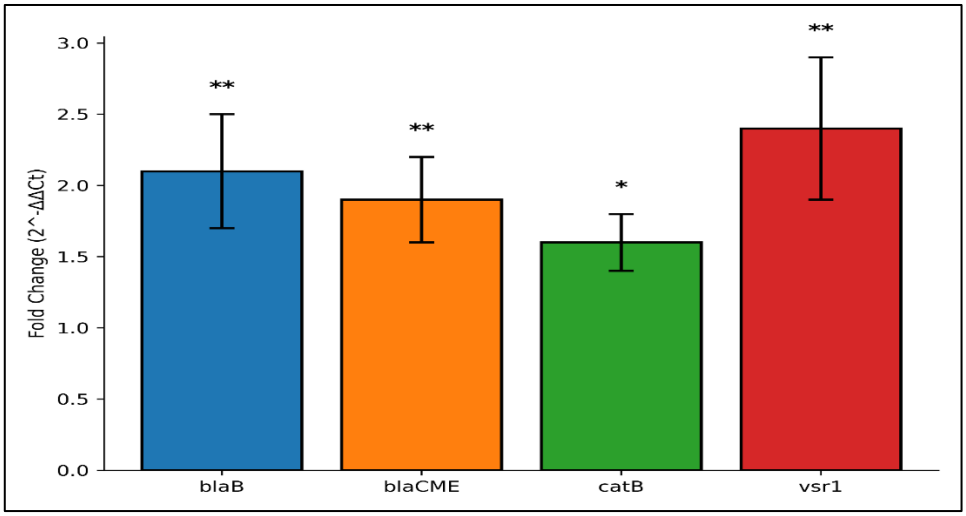

Fig. 2. Relative expression of resistance genes (blaB, blaCME, catB, vsr1) in Elizabethkingia anophelis following cortisol exposure (30 µg/dL) compared with baseline levels.

Effect of Cortisol on Resistance Gene Expression

Where resistance genes are concerned, exposure to cortisol at rates of 30 µg/dL, which are deemed physiologically relevant, has been shown to enhance expression of resistance genes substantially in the context of baseline levels. For blaB, which increased by a fold of 2.1 ± 0.4 ($p = 0.004$), and blaCME, which increased by a fold of 1.9 ± 0.3 ($p = 0.008$), catB increased by a fold of 1.6 ± 0.2 ($p = 0.021$), while vsr1 increased by a fold of 2.4 ± 0.5 ($p = 0.002$). Analyzing the data with a paired t test showed that all genes displayed significant transcriptional activation (Fig. 3).

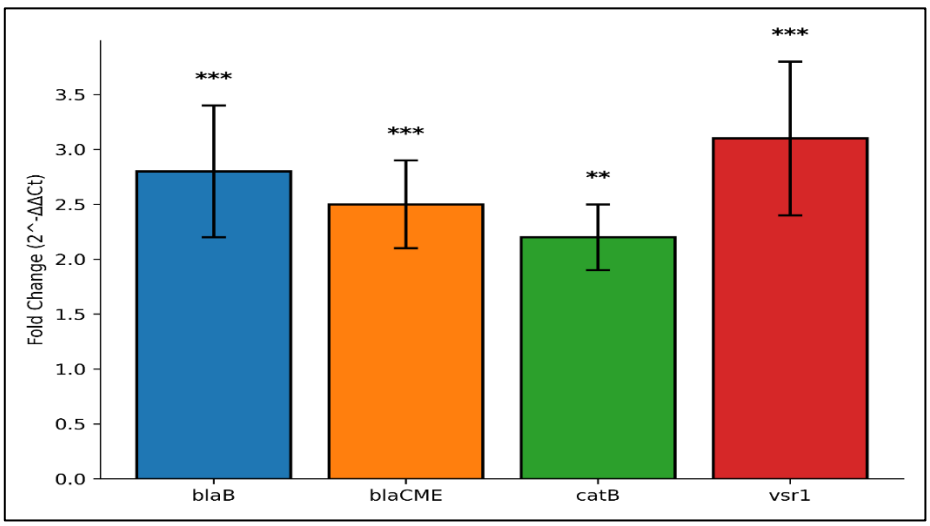

Fig. 3. Relative expression of resistance genes in Elizabethkingia anophelis following norepinephrine exposure (800 pg/mL).

Influence of Catecholamines on the Expression of Resistance Genes

Among the individual hormones tested, norepinephrine produced the most pronounced transcriptional activation, resulting in a 2.8 ± 0.6-fold ($p < 0.001$) increase in blaB expression, and a 2.5 ± 0.4-fold ($p < 0.001$) increase in blaCME, a 2.2 ± 0.3-fold ($p = 0.003$) increase in catB, and a 3.1 ± 0.7-fold ($p < 0.001$) increase in vsr1. While exposure to epinephrine also resulted in transcriptional activation, the magnitude of activation was lower than that of norepinephrine ($p < 0.05$ for all genes). Using repeated-measures ANOVA, it was shown that norepinephrine-induced activation was greater than that of activation induced by cortisol ($p = 0.018$) (Fig. 4).

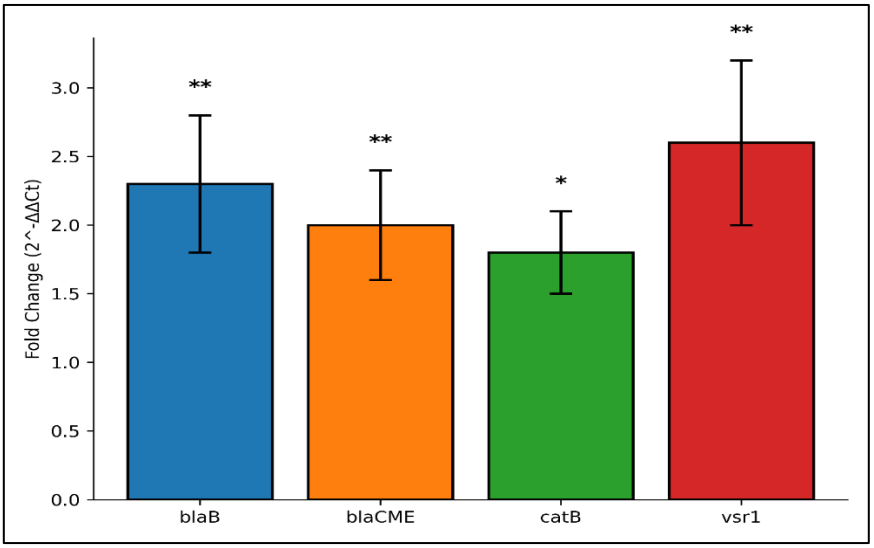

Fig. 4. Relative expression of resistance genes in *Elizabethkingia anophelis* after epinephrine exposure (180 pg/mL).

Combined Hormone Exposure Synergistic Effect

The transcriptional activation resulting from the exposure to cortisol, norepinephrine, and epinephrine simultaneously was markedly synergistic. Fold changes were 3.9 ± 0.8 for blaB, 3.5 ± 0.6 for blaCME, 3.1 ± 0.5 for catB, and 4.2 ± 0.9 for vsr1 (all $p < 0.001$ vs baseline). Post hoc analysis showed that combined exposure resulted in more pronounced expression than any individual hormone exposure ($p < 0.01$). The greatest response was observed for vsr1, suggesting a possible regulatory involvement in the activation of resistance due to stress (Fig. 5).

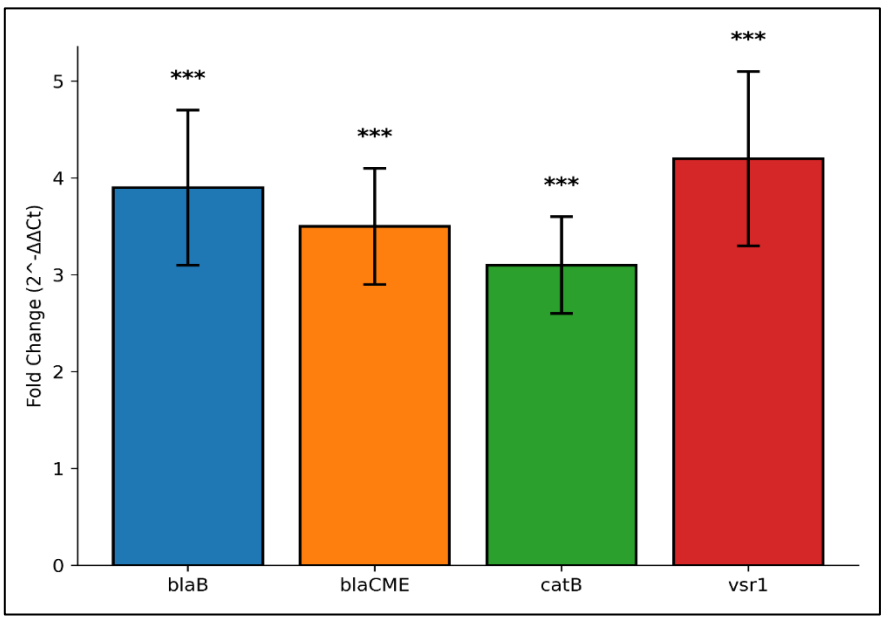

Fig. 5. Synergistic effect of combined cortisol, norepinephrine, and epinephrine exposure on resistance gene expression in *Elizabethkingia anophelis*.

Discussion

In this study, we confirmed that the stress hormones of the host enhance the transcription of resistance genes of Elizabethkingia anophelis, therefore, confirming the Neuroendocrine–Resistome Axis theory. Recent studies show that E. anophelis has become a clinically significant multi-drug resistant pathogen affecting patients in the ICU of hospitals, where there is greater physiological stress (Brandsema et al., 2024; Kang et al., 2025). Studies using whole genome sequencing have revealed a multi-layered

resistome, with genes that code for β-lactamase and other regulatory mechanisms that support life in adverse conditions typical of hospital settings (Andriyanov et al., 2024; Commans et al., 2024). Therefore, the transcriptional changes provided evidence that the physio-psychological signals of the host are part of the adaptive mechanisms of the organism.

It has been previously documented that the β-lactamase determinants, for instance, blaB and blaCME, played a pivotal role in increased β-lactam MICs in E. anophelis (Chen et al., 2024). These findings provide further extension of the previously noted genomic attributes by the elucidation of the measurable transcriptional activation of the aforementioned genes as a result of the presence of cortisol and catecholamines. This may imply that the expression of resistance may not be completely organismal, but responsive and regulated dynamically to the neuroendocrine stimuli of the host. Other than the ones noted in Rea Hernández et al. (2024), there have been other instances where hormonal modulation of bacterial gene expression has been documented, such as in the case of some other pathogens where the presence of epinephrine and norepinephrine increased the expression of some virulence factors and elicited some of the adaptive responses. These findings further add to the developing thought process regarding the bacterial pathogens being able to sense the presence of host stress mediators, potentially aiding in the virulence of the pathogens.

Norepinephrine's transcriptional activation fits the earlier account of catecholamine - induced enhancement of bacterial growth and antimicrobial tolerance mechanism development (Rea Hernández et al., 2024). Patients in the ICU are usually subjected to persistent sympathetic stimulation and increased circulating catecholamines, which could allow the biochemical environment to activate resistance genes. Multidrug-resistant pathogens have poor clinical outcomes, and the clinical studies have documented the rising frequency of multidrug-resistant Elizabethkingia (Wu et al., 2024; Chuang et al., 2025). The present results give a possible explanation of the stress response to the host and resistance regulation response at the transcriptional level.

The reliable biochemical measurements were attributed to advanced LC– MS/MS systems for the precise quantification of cortisol and catecholamines (Noh et al., 2023; Su et al., 2024). The combination of bacterial gene expression analysis and high sensitivity hormonal measurements also provided a multidimensional view of the host-pathogen interaction. This holistic approach is a significant step beyond genomic studies, which often are devoid of functional host signal data.

Focusing on processes other than traditional antimicrobial resistance, there are notable parallels with natural product mediated antimicrobial modification. As demonstrated in the study by Raheema & Shoker (2020), Shoker et al. (2021), phytochemical and nanoparticle-based extracts can change the pattern of bacterial susceptibility and associated gene expression. The study of plant and host hormone mediated biochemical stimuli shows to what extent microbial transcriptional networks can be modified.

Furthermore, the ability to manipulate nanotechnology in conjunction with electrochemical detection provides the ability to detect trace amounts of biochemical stimuli, easily and with great specificity (Raheema et al. 2025). The ability to manipulate biochemical stimuli in host and microbe interactions shows a great deal of promise to further study the interactions.

Conclusion

All of these findings show that hospitalized patients with systemic stress can lead to increased levels of cortisol and/or catecholamines, and these may have direct effects on the transcriptional control of resistance genes pertaining to Elizabethkingia anophelis. The integration of clinical epidemiology, genomic resistance, and neuroendocrine modulation provides the evidence that, at least in an emerging pathogen, antimicrobial resistance may be a result of the host physiological condition. Future studies should assess whether targeting host stress pathways and/or blocking the bacterial hormone-sensing pathways may serve as additional means to reduce the activation of resistance mechanisms during a more advanced stage of infection.

References

1. Andriyanov, P., Zhurilov, P., Menshikova, A., Tutrina, A., Yashin, I., & Kashina, D. (2024). Large-scale genomic analysis of Elizabethkingia anophelis. BMC Genomics, 25(1), 1015. https://doi.org/10.1186/s12864-024-10921-y
2. Andriyanov, P., Zhurilov, P., Menshikova, A., Tutrina, A., Yashin, I., & Kashina, D. (2024). Large-scale genomic analysis of Elizabethkingia anophelis. BMC Genomics, 25(1), 1015. https://doi.org/10.1186/s12864-024-10921-y
3. Brandsema, B. R., Fleurke, G.-J., Rosema, S., Schins, E. M. W., Helfferich, J., & Bathoorn, E. (2024). Neonatal Elizabethkingia anophelis meningitis originating from the water reservoir of an automated infant milk dispenser, the Netherlands, February 2024. Eurosurveillance, 29(14), 2400177. https://doi.org/10.2807/1560-7917.ES.2024.29.14.2400177
4. Brandsema, B. R., Fleurke, G.-J., Rosema, S., Schins, E. M. W., Helfferich, J., & Bathoorn, E. (2024). Neonatal Elizabethkingia anophelis meningitis originating from the water reservoir of an automated infant milk dispenser, the Netherlands, February 2024. Eurosurveillance, 29(14), 2400177. https://doi.org/10.2807/1560-7917.ES.2024.29.14.2400177
5. Chen, P.-J., Tan, M.-C., Huang, W.-C., Hsu, S.-Y., Chen, T.-L., Yang, C.-Y., & Kuo, S.-C. (2024). The individual contributions of blaB, blaGOB and blaCME on MICs of β-lactams in Elizabethkingia anophelis. Journal of Antimicrobial Chemotherapy, 79(7), 1577-1580. https://doi.org/10.1093/jac/dkae137
6. Chen, P.-J., Tan, M.-C., Huang, W.-C., Hsu, S.-Y., Chen, T.-L., Yang, C.-Y., & Kuo, S.-C. (2024). The individual contributions of blaB, blaGOB and blaCME on MICs of β-lactams in Elizabethkingia anophelis. Journal of Antimicrobial Chemotherapy, 79(7), 1577-1580. https://doi.org/10.1093/jac/dkae137
7. Chuang, W.-L., Chang, F.-C., Kuo, C.-F., & Lin, C.-C. (2025). Multidrug-resistant Elizabethkingia anophelis bacteremia in Northern Taiwan: Focusing on prognostic factors and antimicrobial susceptibility to minocycline and rifampin. Infection and Drug Resistance, 18, 4337-4349. https://doi.org/10.2147/IDR.S536057
8. Commans, F., Hayer, J., Do, B. N., Tran, T. T. T., Le, T. T. H., Bui, T. T., Le, H. S., Bañuls, A.-L., Bui, T. S., & Nguyen, Q. H. (2024). Whole-genome sequence and resistance determinants of four Elizabethkingia anophelis clinical isolates collected in Hanoi, Vietnam. Scientific Reports, 14(1), 7241. https://doi.org/10.1038/s41598-024-57564-3
9. Commans, F., Hayer, J., Do, B. N., Tran, T. T. T., Le, T. T. H., Bui, T. T., Le, H. S., Bañuls, A.-L., Bui, T. S., & Nguyen, Q. H. (2024). Whole-genome sequence and resistance determinants of four Elizabethkingia anophelis clinical isolates collected in Hanoi, Vietnam. Scientific Reports, 14(1), 7241. https://doi.org/10.1038/s41598-024-57564-3
10. Ichiki, K., Ooka, T., Shinkawa, T., Inoue, S., Hayashida, M., Nakamura, D., Akimoto, M., Yoshimitsu, M., Kawamura, H., Nakamura, M., Obama, Y., Gotoh, Y., Hayashi, T., Nishi, J., & Ishitsuka, K. (2023). Genomic and phylogenetic characterization of Elizabethkingia anophelis strains: The first two cases of life-threatening infection in Japan. Journal of Infection and Chemotherapy, 29(4), 376-383. https://doi.org/10.1016/j.jiac.2023.01.005
11. Johnson, W. L., Gupta, S. K., Maharjan, S., Morgenstein, R. M., Nicholson, A. C., McQuiston, J. R., & Gustafson, J. E. (2024). A Genetic Locus in Elizabethkingia anophelis Associated with Elevated Vancomycin Resistance and Multiple Antibiotic Reduced Susceptibility. Antibiotics, 13(1), 61. https://doi.org/10.3390/antibiotics13010061
12. Kang, X., Guo, H., Zhao, S., Zhang, W., Liu, P., Mei, Y., Zeng, L., & Wei, D. (2025). Prevalence, microbiological features, and clinical characteristics of Elizabethkingia isolates in a tertiary hospital, Jiangxi Province, China. Infectious Medicine (Beijing), 4(3), 100198.

https://doi.org/10.1016/j.imj.2025.100198

13. Lei, Y., Rahman, K., Cao, X., Yang, B., Zhou, W., Reheman, A., Cai, L., Wang, Y., Tyagi, R., Wang, Z., Chen, X., & Cao, G. (2023). Epinephrine stimulates Mycobacterium tuberculosis growth and biofilm formation. International Journal of Molecular Sciences, 24(24), 17370. https://doi.org/10.3390/ijms242417370
14. Nguyen, H. D., Nguyen, S. T., Ha, V. T. T., Nghiem, M. N., & Vo, T. T. B. (2020). Expression of some resistant genes on six *Elizabethkingia* spp. strains isolated in Vietnam. *Journal of Public Health and Diseases, 3*(2), 35–42. https://doi.org/10.31248/JPHD2020.063
15. Niu, L., Gao, M., Wen, S., Wang, F., Shangguan, H., Guo, Z., Zhang, R., & Ge, J. (2023). Effects of catecholamine stress hormones norepinephrine and epinephrine on growth, antimicrobial susceptibility, biofilm formation, and gene expressions of enterotoxigenic Escherichia coli. International Journal of Molecular Sciences, 24(21), 15646. https://doi.org/10.3390/ijms242115646
16. Noh, J., Lee, C., Kim, J. H., Myung, S. W., & Choi, M. H. (2023). LC-MS based simultaneous profiling of adrenal hormones of steroids, catecholamines, and metanephrines. Journal of Lipid Research, 64(11), 100453. https://doi.org/10.1016/j.jlr.2023.100453
17. Noh, J., Lee, C., Kim, J. H., Myung, S. W., & Choi, M. H. (2023). LC-MS based simultaneous profiling of adrenal hormones of steroids, catecholamines, and metanephrines. Journal of Lipid Research, 64(11), 100453. https://doi.org/10.1016/j.jlr.2023.100453
18. Raheema, R. H., & Shoker, R. M. H. (2020). Phytochemicals screening and antibacterial activity of silver nanoparticles, phenols and alkaloids extracts of Conocarpus lancifolius. EurAsian Journal of Biosciences, 14(2), 4829-4836.
19. Raheema, R. H., Khalaf, T. K., Shoker, R. M. H., & Yasir, Q. D. (2025). Awareness and knowledge of celiac disease and the role of nanotechnology and electrochemical methods in gluten detection. Analytical and Bioanalytical Electrochemistry, 17(8), 747-770.
20. Rea Hernández, P. A., Ramírez-Paz-Y-Puente, G. A., Montes-García, F., Vázquez-Cruz, C., Sanchez-Alonso, P., Cobos-Justo, M. E., Zenteno, E., & Negrete-Abascal, E. (2024). Epinephrine and norepinephrine regulate the expression of virulence factors in Gallibacterium anatis. Microbial Pathogenesis, 196, 106987. https://doi.org/10.1016/j.micpath.2024.106987
21. Sengar, S., Varghese, G., Jamwal, A., Sarawat, D., Tejan, N., Singh, S., Patel, S. S., & Sahu, C. (2025). Elizabethkingia anophelis infection in intensive care unit patients at a tertiary care center in North India: A retrospective study. The American Journal of Tropical Medicine and Hygiene, 113(3), 678-682. https://doi.org/10.4269/ajtmh.24-0550
22. Shoker, R. M. H., Raheema, R. H., & Shamkhi, I. J. (2021). Antimicrobial activity, HPLC analysis of phenolic extract of Ocimum basilicum and Ocimum sanctum. Biochemical & Cellular Archives, 21(2), 3493-3500.
23. Su, R., Wu, Y., Huang, Q., Huang, S., Li, Y., Chen, C., Xie, W., & Li, Y. (2024). Effects of blood collection tubes on the quantification of catecholamines and their O-methylated metabolites by liquid chromatography coupled with tandem mass spectrometry. Journal of Clinical Laboratory Analysis, 38(19-20), e25107. https://doi.org/10.1002/jcla.25107
24. Su, R., Wu, Y., Huang, Q., Huang, S., Li, Y., Chen, C., Xie, W., & Li, Y. (2024). Effects of blood collection tubes on the quantification of catecholamines and their O-methylated metabolites by liquid chromatography coupled with tandem mass spectrometry. Journal of Clinical Laboratory Analysis, 38, e25107. https://doi.org/10.1002/jcla.25107
25. Wu, C., Xiong, L., Liao, Q., Zhang, W., Xiao, Y., & Xie, Y. (2024). Clinical manifestations, antimicrobial resistance and genomic feature analysis of multidrug-resistant Elizabethkingia strains. Annals of Clinical Microbiology and Antimicrobials, 23(1), 32. https://doi.org/10.1186/s12941-024-00691-6