



ORIGINAL ARTICLE

Prevalence of human herpesvirus 8 in schizophrenia patients and blood donors: A single-center study

Muhammed Furkan Kürkcü ^{a,*}, Eylül Beren Tanık^b, Mehmet Rıdvan Varlı^c,
Gülsüm Yılmaz^c, Ayfer Bakır^a

^a Department of Medical Microbiology, Ministry of Health, Ankara Etlik City Hospital, Ankara, Turkey

^b Ministry of Health, General Directorate of Public Health, National Virology Reference Laboratory, Ankara, Turkey

^c Department of Psychiatry, Ministry of Health, Ankara Etlik City Hospital, Ankara, Turkey

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Seroprevalence;
Polymerase chain
reaction

Abstract

Objective: Human herpesvirus-8 (HHV-8) causes lymphoproliferative diseases, but recent studies suggest it may also be linked to neuropsychiatric disorders. This study aimed to determine HHV-8 IgG seroprevalence in individuals with schizophrenia and blood donors, as well as to evaluate whether HHV-8 constitutes a risk factor for schizophrenia. A total of 135 patients diagnosed with schizophrenia based on DSM-V criteria and 200 healthy blood donors were included. HHV-8 IgG antibodies were analyzed using ELISA, and molecular detection of HHV-8 DNA was performed by real-time PCR. The seropositivity rates between the groups were statistically compared. HHV-8 IgG positivity was detected in 6.7% of schizophrenia patients (9/135, CI: 3.1–12.3) and 5.0% of blood donors (10/200, CI: 2.4–9.0) ($p = 0.518$). HHV-8 DNA was not detected in any of the serum samples that tested positive for HHV-8 IgG. No statistically significant association was observed between HHV-8 seropositivity and schizophrenia (OR = 1.36; 95% CI: 0.53–3.43; $p = 0.519$). Brief Psychiatric Rating Scale (BPRS) total scores were similar between IgG-positive (median 27, IQR 17–42; mean 47.0 ± 13.3) and IgG-negative patients (median 26, IQR 20–34; mean 45.0 ± 10.8) ($p = 0.678$). Although some BPRS parameters appeared higher in IgG-positive patients, none were statistically significant after multiple testing correction. In this study, HHV-8 IgG seroprevalence was similar between schizophrenia patients and blood donors, and no statistically significant association was observed between HHV-8 seropositivity and schizophrenia. To enhance the generalizability of the findings, larger studies with increased sample size, as well as comparative, regional, and multicenter designs, are needed.

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* Corresponding author.

E-mail address: furkan.kurkcugmail.com (M.F. Kürkcü).

PALABRAS CLAVE

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inmunoenzimático;
Seroprevalencia;
Reacción en cadena
de la polimerasa

Prevalencia del herpesvirus humano 8 en pacientes con esquizofrenia y donantes de sangre: Un estudio unicéntrico

Resumen El virus del herpes humano 8 (VHH-8) causa enfermedades linfoproliferativas, pero estudios recientes sugieren que también podría estar relacionado con trastornos neuropsiquiátricos. Este estudio tuvo como objetivo determinar la seroprevalencia de IgG contra VHH-8 en individuos con esquizofrenia y en donantes de sangre sanos, así como evaluar si el VHH-8 constituye un factor de riesgo para la esquizofrenia. Se incluyeron un total de 135 pacientes diagnosticados con esquizofrenia según los criterios del DSM-V y 200 donantes de sangre sanos. Los anticuerpos IgG contra VHH-8 se analizaron mediante ELISA, y la detección molecular del ADN de VHH-8 se realizó mediante PCR en tiempo real. Se compararon estadísticamente las tasas de seropositividad entre los grupos. La positividad de IgG contra VHH-8 se detectó en el 6,7% de los pacientes con esquizofrenia (9/135; IC: 3,1–12,3) y en el 5,0% de los donantes de sangre (10/200; IC: 2,4–9,0) ($p = 0,518$). El ADN de VHH-8 no se detectó en ninguna de las muestras de suero que resultaron positivas para IgG contra VHH-8. No se observó una asociación estadísticamente significativa entre la seropositividad para VHH-8 y la esquizofrenia (OR = 1,36; IC 95%: 0,53–3,43; $p = 0,519$). Las puntuaciones totales de la Escala Breve de Evaluación Psiquiátrica (BPRS) fueron similares entre los pacientes IgG positivos (mediana 27, RI 17–42; media $47,0 \pm 13,3$) y los IgG negativos (mediana 26, RI 20–34; media $45,0 \pm 10,8$) ($p = 0,678$). Aunque algunos parámetros de la BPRS parecieron más elevados en los pacientes IgG positivos, ninguno fue estadísticamente significativo tras la corrección por pruebas múltiples. En este estudio, la seroprevalencia de IgG contra VHH-8 fue similar entre los pacientes con esquizofrenia y los donantes de sangre, y no se observó una asociación estadísticamente significativa entre la seropositividad para VHH-8 y la esquizofrenia. Para mejorar la generalización de los hallazgos, se necesitan estudios con mayor tamaño muestral, así como diseños comparativos, regionales y multicéntricos.

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Introduction

Schizophrenia is a chronic psychiatric disorder affecting approximately 1% of the global population and is characterized by delusions, hallucinations, cognitive impairments, and emotional blunting. In addition to genetic and environmental determinants, infectious agents have been proposed as potential contributors to its pathogenesis^{2,16}. Given their neurotropic properties and lifelong latency, herpesviruses have been suggested to play a role in schizophrenia. Supporting this hypothesis, Hannachi et al. reported a significantly higher HHV-8 IgG prevalence among schizophrenia patients (28.7%) compared to healthy controls (14.8%) in Tunisia, indicating a possible association between HHV-8 and the disorder¹⁵. Nevertheless, epidemiological data regarding this relationship remain scarce.

Human herpesvirus 8 (HHV-8), also known as Kaposi's sarcoma-associated herpesvirus (KSHV), belongs to the *Gammaherpesvirinae* subfamily of the *Herpesviridae* family. It primarily infects B lymphocytes but can also target endothelial and epithelial cells¹². HHV-8 follows a biphasic life cycle consisting of acute and latent phases, and its latency mechanisms may contribute to oncogenesis^{3,4,30}. Clinically, the virus is most strongly linked to Kaposi's sarcoma, primary effusion lymphoma, and multicentric Castlemann disease^{7,14}.

Global HHV-8 seroprevalence exhibits wide geographic variation. High prevalence rates have been reported in sub-Saharan Africa, whereas lower rates are observed in Europe and the Americas¹⁹. Transmission occurs mainly through saliva in childhood, while sexual transmission plays a more prominent role in adulthood, especially among men who have sex with men. Blood transfusion and organ transplantation are rare but possible transmission routes, and evidence for intravenous drug use remains inconclusive^{11,26}.

Considering the limited evidence on the role of HHV-8 in psychiatric disorders, alongside conflicting findings reported for other herpesviruses^{8,10,17}, further studies are required. The present study aimed to investigate HHV-8 IgG seroprevalence in patients with schizophrenia compared with blood donors in Türkiye and to assess whether HHV-8 infection may be associated with schizophrenia.

Materials and methods**Study design and participants**

This prospective cross-sectional study was conducted at Ankara Etlik City Hospital between July 1, 2024, and October 1, 2024. The study group consisted of 135 patients aged 18–65 years who had been diagnosed with schizophrenia by board-certified psychiatrists at the Psychiatry Clinic

of Ankara Etlik City Hospital based on DSM-V diagnostic criteria. The diagnoses were confirmed through clinical evaluation and consensus among the psychiatrists. These patients were regularly followed up at the hospital's psychiatry outpatient clinic and voluntarily agreed to participate in the study.

To evaluate the psychiatric symptoms of the patients, a psychiatrist administered the 18-item Brief Psychiatric Rating Scale (BPRS). The BPRS scoring ranged from 1 (Absent) to 7 (Severe) based on the severity of each symptom¹³. For the purposes of this study, scores ≥ 2 were considered to indicate the presence of a symptom, regardless of severity, in order to capture the full spectrum of psychiatric manifestations from mild to severe.

During the same period, 200 healthy blood donors, aged 18–65 years, who met the eligibility criteria for blood donation and voluntarily agreed to participate in the study, were recruited from the Ankara Etlik City Hospital Blood Center as the control group.

All participants were residents of Ankara, Türkiye, and were recruited from the same healthcare center. The study population was considered relatively homogeneous in terms of geographic and ethnic background.

This study was conducted in accordance with the Declaration of Helsinki. Ethical approval was obtained from the Ethics Committee of Ankara Etlik City Hospital, University of Health Sciences (Approval No: AEŞH-BADEK-2024-488). Written informed consent was obtained from all participants or their legal guardians prior to inclusion in the study.

Serological analysis

A 5 ml venous blood sample was obtained from each participant. After serum separation, 1 ml aliquots were divided into two Eppendorf tubes and stored at -20°C until analysis.

HHV-8 IgG antibody screening was performed using the ELISA method at the Serology Laboratory of the Medical Microbiology Department, Ankara Etlik City Hospital. For this purpose, a commercially available HHV-8 IgG ELISA kit (Sunlong Biotech, China; Cat. No: SL2685Hu) was used, and the test was conducted and evaluated according to the manufacturer's protocol. Results were evaluated qualitatively according to the manufacturer's instructions, as the assay does not provide semi-quantitative or quantitative antibody measurements. The cut-off (CO) value was determined by calculating the mean optical density (OD) of two negative control wells, dividing this value by 2, and adding 0.15. Serum samples with OD values $\geq \text{CO}$ were considered HHV-8 IgG positive. To ensure accuracy, both positive and negative control samples were included in each test run. The calculated CO value for this study was 0.43, based on the formula described above. Information regarding the sensitivity and specificity of the assay was not available in the manufacturer's documentation.

Molecular analysis

HHV-8 DNA isolation was performed using the Biospeedy® Rapid Nucleic Acid Extraction Kit (Bioeksan R&D Technologies Inc., Istanbul, Türkiye) with the Zybion EXM3000 automatic extraction device (Zybion Inc., Chongqing, China).

The amplification of the obtained DNA samples was carried out using the Human Herpesvirus 8 qPCR Kit (Bioeksan R&D Technologies Inc., Istanbul, Türkiye; Cat. No: BS-HHV8-25) with the Magnetic Induction Cycler (Mic) PCR device (Bio Molecular Systems, Upper Coomera, QLD, Australia). HHV-8 DNA detection targeted the ORF73 (LANA) gene region.

The amplification data obtained from real-time PCR were analyzed using Sigmoida Software (V 8.6) integrated into the device (Bioeksan R&D Technologies Inc., Istanbul, Türkiye). The validity of the results was assessed through automated analysis, and manual interpretation was applied when necessary to ensure result accuracy. To ensure reliability, one positive and one negative control sample were included in each test run. According to the manufacturer's specifications, the PCR kit has a sensitivity of 100% and a specificity of 99.68%.

Statistical analysis

Data were analyzed using SPSS version 27 (IBM Corp., USA). Descriptive statistics were used to characterize the study population. Categorical variables were expressed as frequencies and percentages, whereas continuous variables were summarized as means with standard deviations (SD) or medians with interquartile ranges (IQR), as appropriate. The normality of distribution was assessed using the Kolmogorov-Smirnov test.

For comparisons of categorical data between groups, Pearson's chi-square test or Fisher's exact test was applied as appropriate. For continuous variables, the independent samples *t*-test was used when data were normally distributed, and the Mann-Whitney *U* test was used for non-normally distributed data. The relationship between disease duration and HHV-8 seropositivity was evaluated using Spearman's rank correlation. To account for multiple testing when analyzing the association between Brief Psychiatric Rating Scale (BPRS) parameters and HHV-8 status, both Bonferroni and Benjamini-Hochberg false discovery rate (FDR) corrections were applied. A two-tailed *p*-value < 0.05 was considered statistically significant; adjusted *p*-values are reported where relevant.

The risk estimation for HHV-8 infection in schizophrenia patients was calculated using logistic regression analysis, with odds ratios (ORs) and 95% confidence intervals (CIs). All tests were two-tailed, and a *p*-value < 0.05 was considered statistically significant.

Results

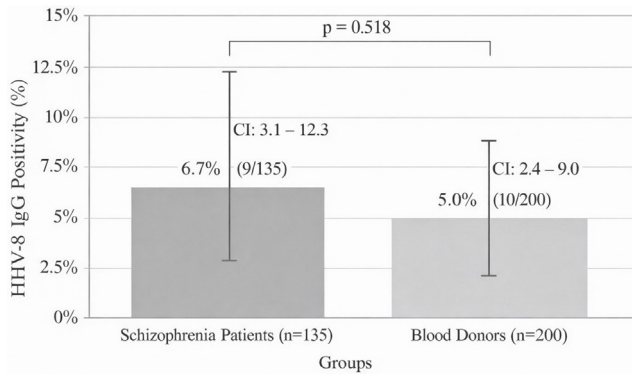
The patients included in the study were between 18 and 65 years old, and detailed demographic data, including gender distribution and mean age, are provided in Table 1.

Among the 135 schizophrenia patients included in the study, HHV-8 IgG positivity was detected in 9 patients (6.7%, CI: 3.1–12.3), while 10 out of 200 blood donors (5.0%, CI: 2.4–9.0) tested positive for HHV-8 IgG (Fig. 1). The seroprevalence rates did not show a significant difference between the groups ($p = 0.518$). In serum samples from individuals who tested positive for HHV-8 IgG, the presence of HHV-8 DNA was not detected in any sample using the PCR method.

Table 1 Demographic data of the study population.

Groups	Number (n)	Male/female	Age*	Age range	Disease duration*	Urban/rural
Schizophrenia	135	85/50	39 (33–49)	18–65	11 (7–20)	124/11
Blood donors	200	161/39	33 (26–42)	19–60	–	200/0
Total	335	246/89	36 (28–46)	18–65	–	324/11

* Age and disease duration (year), median; interquartile range: range between the 25th to 75th. All blood donors were recruited from voluntary donors presenting to the hospital and were residents of urban areas.

**Figure 1** HHV-8 IgG positivity rates in patients with schizophrenia and healthy blood donors.

Among schizophrenia patients, HHV-8 IgG positivity was 8.2% (7/85) in males and 4.0% (2/50) in females, whereas among blood donors, HHV-8 IgG positivity in males was 6.2% (10/161). No HHV-8 positivity was detected in female blood donors. No significant difference in HHV-8 IgG positivity was observed between genders in either group ($p=0.484$ and $p=0.215$, respectively).

In schizophrenia patients, HHV-8 IgG positivity was observed in 12.5% (5/40) of individuals aged 18–34 years, 6.3% (4/64) of individuals aged 35–49 years, and 0% (0/31) of individuals aged 50–65 years ($p=0.114$). Among blood donors, HHV-8 IgG positivity was 7.6% (8/105) in the 18–34 age group, 2.5% (2/80) in the 35–49 age group, and 0% (0/15) in the 50–65 age group ($p=0.321$).

Although the OR was 1.36, this did not reflect a statistically significant difference between schizophrenia patients and blood donors (95% CI: 0.53–3.43; $p=0.519$). The demographic and clinical characteristics of HHV-8 IgG-positive schizophrenia patients are presented in [Supplementary Table 1](#).

Psychopathological symptom distributions are summarized in [Supplementary Figs. 1 and 2](#). The three most common symptoms were depressed mood (90.4%, $n=122$), anxiety (88.9%, $n=120$), and emotional withdrawal (88.1%, $n=117$). In contrast, excitement (31.1%, $n=42$), disorientation (27.4%, $n=37$), and grandiosity (26.7%, $n=36$) were observed at lower rates. Among the schizophrenia patients, 8.1% (11/135) resided in rural areas, whereas all blood donors resided in urban areas.

Although some BPRS parameters appeared higher in HHV-8 IgG-positive individuals, no statistically significant

differences were observed between IgG-positive and IgG-negative groups. Depressed mood and motor retardation were observed in all HHV-8 positive individuals. Additionally, unusual thought content was found at a lower rate in HHV-8 positive individuals compared to HHV-8 negative individuals ($p=0.033$). However, this association did not remain significant after correction for multiple testing (Bonferroni and FDR-adjusted $p>0.05$) ([Supplementary Table 2](#)).

In schizophrenia patients, BPRS scores ranged from 19 to 78, with a mean of 45.1 ± 10.9 . Among HHV-8 IgG-positive patients, the mean total BPRS score was 47.0 ± 13.3 (range 28–67), whereas in IgG-negative patients, it was 45.0 ± 10.8 (range 19–78) ($p=0.678$). The presence of IgG antibodies was not associated with symptom severity ($p=0.598$).

Discussion

In this study, the HHV-8 IgG seroprevalence was found to be 6.7% in schizophrenia patients and 5.0% in blood donors. Although the IgG positivity rate was higher in schizophrenia patients compared to blood donors, the difference was not statistically significant.

Global HHV-8 seroprevalence studies have demonstrated substantial regional variations in HHV-8 IgG positivity rates. The highest seroprevalence rates have been reported in sub-Saharan African countries (30–80%). In contrast, lower prevalence rates have been identified in Europe and Mediterranean countries (2–28%) and America (1–5%)^{22,25}. In Türkiye, the limited number of studies conducted have reported HHV-8 seroprevalence among blood donors to range between 5.2% and 7.0%^{1,28}. The seroprevalence detected in blood donors in this study is consistent with data from Türkiye and Europe.

In the literature, only one study investigating HHV-8 IgG seroprevalence in schizophrenia patients has been identified. Hannachi et al. from Tunisia reported that HHV-8 IgG prevalence was significantly higher in schizophrenia patients (28.7%) compared to the healthy control group (14.8%) ($p=0.01$). Furthermore, they observed a significant association between HHV-8 seroprevalence in schizophrenia patients and both positive symptoms (SAPS score) and disease severity (CGI score). However, no relationship was found between BPRS and PANSS scores, disease duration, mode of onset, or schizophrenia subtype¹⁵.

In our current study, we investigated the relationship between HHV-8 IgG prevalence and BPRS scores in schizophrenia patients but did not find any association. The lack of a significant association observed in our study may be

attributed to multiple factors, including regional variations in HHV-8 seroprevalence and limited statistical power due to sample size. Methodological constraints, such as the use of qualitative serological assays and serum-based PCR, may also have limited the detection of latent infection. Serological and molecular assays used for HHV-8 diagnosis have inherent technical limitations. Serological assays, including ELISA-based IgG detection, primarily reflect past exposure and are unable to distinguish between latent and active infection. Moreover, variability in assay performance and potential cross-reactivity may affect the accuracy of seroprevalence estimates. Molecular detection by PCR is also limited, particularly when serum samples are used, as HHV-8 predominantly establishes latency in B lymphocytes and endothelial cells rather than circulating freely in plasma. Therefore, the absence of detectable viral DNA in serum does not exclude latent infection or low-level viral persistence. These limitations have been highlighted in previous studies. Furthermore, the lack of a universally accepted gold standard for HHV-8 diagnosis has been emphasized in the literature²⁴.

Furthermore, unlike neurotropic herpesviruses, the limited neuroinvasive potential of HHV-8 may explain the absence of a measurable association with schizophrenia. These findings highlight the importance of considering both biological and methodological factors when interpreting the potential role of HHV-8 in neuropsychiatric disorders.

Several herpesviruses, including HSV-1/2, CMV, EBV, and HHV-6, have been associated with schizophrenia in previous studies; however, findings remain inconsistent^{9,18,20,21,31}. Some studies suggest a potential role of these viruses in disease pathogenesis, including maternal infection and immune-mediated mechanisms, whereas others have found no significant association^{5,6,9,21,23,27,29}, indicating that the relationship between herpesviruses and schizophrenia is complex and may vary depending on population characteristics and study design. In contrast, there is no evidence suggesting that HHV-8 affects the central nervous system through a similar mechanism. Unlike neurotropic herpesviruses such as HSV-1 and HHV-6, HHV-8 predominantly targets B lymphocytes and endothelial cells, with limited evidence supporting its ability to establish latency or replication within the central nervous system. Accordingly, HHV-8 may have a lower likelihood of establishing persistent infection in neural tissues compared to other herpesviruses¹². In this study, the absence of HHV-8 DNA in serum samples is not unexpected and should be interpreted with caution, as HHV-8 establishes latency primarily in B lymphocytes and endothelial cells rather than circulating freely in serum. This discrepancy may be explained by the fact that HHV-8 IgG seropositivity reflects past exposure or latent infection, whereas the absence of detectable viral DNA in serum likely indicates a lack of active viremia at the time of sampling. Accordingly, the use of serum samples may have limited the detection of latent HHV-8 infection. Future studies should utilize whole blood, peripheral blood mononuclear cells (PBMCs), or lymphocyte fractions to improve detection sensitivity and enable a more accurate assessment of latent viral persistence.

Despite the lack of statistical significance, reporting HHV-8 seroprevalence by age group may provide epidemiological insight into potential exposure and transmission

patterns. However, due to the limited sample size, these findings should be interpreted with caution, and larger studies are required to clarify possible age-related differences. This study has several limitations. The current virological approach, based on serum serology and PCR, is not sufficient to assess latent infection or causal relationships. The first limitation is that due to its cross-sectional design, it is not possible to determine whether HHV-8 infection is a causal factor in the development of schizophrenia. Additionally, as this is a regional and single-center study, the findings should not be generalized to the entire country. Lastly, the study only included serum samples. This limitation further supports the restricted ability of serum-based assays to detect latent HHV-8 infection. Therefore, latent HHV-8 infection or low-level viral persistence cannot be excluded based on the current methodology. Quantitative or semi-quantitative antibody levels were not available, which limited the evaluation of differences in immune response intensity between groups. To determine whether HHV-8 establishes latent infection in the central nervous system, further research involving cerebrospinal fluid (CSF) studies or post-mortem brain autopsy analyses may be necessary. Another limitation is related to the selection of the control group. As the controls were blood donors, their age and gender distributions were partly determined by donation eligibility criteria in Türkiye. Individuals above 65 years of age are not eligible to donate blood, and the proportion of female donors is generally lower compared to males. These restrictions may have contributed to the observed differences in demographic characteristics between patients and controls, and should be considered when interpreting the results. We acknowledge that some studies define clinically significant symptoms at higher thresholds (e.g., ≥ 4). Our categorization (≥ 2) was intended to capture any presence of the symptom, not only clinically severe manifestations. This approach may account for the relatively high prevalence rates reported in our results and should be interpreted with caution.

In conclusion, this study is significant as it represents one of the first studies in Türkiye investigating HHV-8 prevalence in schizophrenia patients and blood donors. The findings indicate that HHV-8 seroprevalence is similar between schizophrenia patients and blood donors, and no statistically significant association was observed between HHV-8 seropositivity and schizophrenia. Therefore, our findings do not support an association between HHV-8 infection and schizophrenia rather than confirming it as a risk factor. These findings should be considered preliminary and region-specific. To improve the generalizability of the results, larger-scale, regional, and multicenter cohort studies are needed.

ORCID ID

Eylül Beren Tanık: 0000-0002-5227-7111

Mehmet Rıdvan Varlı: 0009-0004-0253-4556

Gülsüm Yılmaz: 0000-0003-2612-6173

Ayfer Bakır 0000-0002-9006-5267

Authorship contributions

MFK and AB contributed to the study conception and design. MFK, EBT, MRV and GY were responsible for data collection. MFK, AB and EBT were responsible for analysis. MFK and AB wrote the manuscript. All authors have read and approved the final manuscript.

Ethical approval

This study was conducted in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. Ethical approval for this study was obtained from the Ethics Committee of Ankara Etlik City Hospital, University of Health Sciences (Approval No: AEŞH-BADEK-2024-488).

Informed consent

Informed consent was obtained from all individual participants included in the study. For participants who were not able to provide consent personally, consent was obtained from their legal guardians, in accordance with institutional and ethical guidelines.

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Conflict of interest

The authors declare no conflict of interest.

Data availability

The datasets generated and/or analyzed during the current study are not publicly available due to restrictions imposed by the Institutional Review Board (IRB) and local Ethics Committee. Sharing of the data requires prior approval from the Ethics Committee, in accordance with institutional and legal regulations. However, the data are available from the corresponding author upon reasonable request and with the appropriate permissions from the Ethics Committee.

Appendix A. Supplementary data

Supplementary data associated with this article can be found in the online version available at <https://doi.org/10.1016/j.ram.2026.100738>.

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