RESEARCH ARTICLE

Decoding the Rabies Phosphoprotein: A First Look at Evolutionary and Structural Dynamics of Rabies Virus in Mumbai, India

Gauri R. Sawant[1], Rajesh R. Pharande[1]*, Rajashree S. Gandge[2], Varsha D. Thorat[1], Shailesh D. Ingole[3], Ravindra J. Zende[4]

Abstract

Rabies remains a significant zoonotic threat in India, where dog-mediated transmission causes substantial human mortality and necessitates robust molecular surveillance. The rabies virus (RABV) phosphoprotein (P) gene is a critical determinant of viral replication, neuro-invasiveness, and host immune evasion. The present study aimed to detect and characterise the full-length P gene from rabies-suspected dog brain samples in Mumbai. RT-PCR demonstrated complete concordance with dFAT, successfully amplifying the full-length 894 bp P gene in 14/20 samples (70%). Automated genotyping and phylogenetic reconstruction of five representative amplicons classified the study sequences within the Arctic-like 1a (AL1a) lineage, clustering distinctly into a localised Peninsular India subclade. Evolutionary pressure analysis indicated strong purifying selection acting on the gene. Deduced amino acid sequence analysis identified 26 non-synonymous mutations across the P protein, including conserved clade-defining mutations E165D and A267G, and novel mutations G54R and A171V. *In silico* 3D structural modelling of critical domains revealed conserved spatial architecture in comparison to the SAD B19 vaccine strain. The study findings highlight the region-specific evolutionary dynamics, genetic diversity, and structural stability of endemic RABV variants in Mumbai, India.

Key words: Arctic-like 1a, Phosphoprotein, Phylogeny, Rabies virus, Structural modeling.

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Introduction

Rabies, caused by the rabies virus (RABV) of the genus *Lyssa virus* within the family *Rhabdoviridae*, remains a relentless and fatal neurological menace in developing nations like India (Fooks *et al.,* 2017). Despite the availability of effective vaccines, dog-mediated rabies continues to cause significant global mortality, with India bearing a disproportionate share of this burden, accounting for an estimated 5,700 human fatalities annually (Thangaraj *et al.,* 2024). The continued prevalence of this zoonotic threat is largely driven by inadequate surveillance and a lack of comprehensive molecular tracking (Varun *et al.,* 2026).

The RABV genome encompasses five genes, among which the highly variable P gene encodes an essential multifunctional phosphoprotein (P protein) (Wang *et al.,* 2013). The P protein forms a ribonucleoprotein (RNP) complex with the nucleoprotein (N) and the large (L) protein and acts as a crucial non-catalytic cofactor for the large viral polymerase to facilitate transcription and replication (Tudhope *et al.,* 2025). Beyond replication, the P protein mediates retrograde axonal transport of the virus into the CNS via interaction with the cytoplasmic dynein light chain (LC8) (Jacob *et al.,* 2000; Liu *et al.,* 2022). Furthermore, the P protein functions as a potent interferon antagonist to evade host immunity, notably inhibiting the phosphorylation of Interferon Regulatory Factor (IRF)-3/7 and disrupting the Janus Kinase-Signal Transducer and Activator of Transcription (JAK-STAT) pathway by preventing STAT1/2 nuclear translocation (Masatani *et al.,* 2016).

Genomic surveillance conducted over the past two decades has repeatedly demonstrated region-specific, rather

[1]Department of Veterinary Microbiology, Mumbai Veterinary College, Mumbai-400012, MAFSU, Maharashtra, India

[2]Department of Veterinary Microbiology, College of Veterinary and Animal Sciences, Udgir-413517, MAFSU, Maharashtra, India

[3]Department of Veterinary Physiology, Mumbai Veterinary College, Mumbai-400012, MAFSU, Maharashtra, India

[4]Department of Veterinary Public Health and Epidemiology, Mumbai Veterinary College, Mumbai-400012, MAFSU, Maharashtra, India

Corresponding Author: Dr. R. R. Pharande, Department of Veterinary Microbiology, Mumbai Veterinary College, Parel, Mumbai-400012, Maharashtra, India. e-mail: rpharande@gmail.com

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than host-specific, clustering of Indian RABV isolates (Reddy *et al.,* 2011; Varun *et al.,* 2026). To date, the vast majority of molecular epidemiological studies in India have targeted the partial nucleoprotein and glycoprotein genes, consistently reporting the national predominance of the Arctic-like 1a (AL1a) lineage (Varun *et al.,* 2026). However, there is a significant paucity of data regarding the P gene, despite its pivotal roles in viral adaptation (Holtz *et al.,* 2023). To address this gap, the present study aimed to detect RABV in rabies-suspected dogs from Mumbai using P-gene-targeted reverse transcription-polymerase chain reaction (RT-PCR) and to elucidate the evolutionary lineages, genetic diversity, and mutational profile of the complete P gene, coupled with *in silico* 3D protein modelling to assess the conformational impact of these mutations.

Materials And Methods

Approval for the present study was obtained from the Institutional Biosafety Committee (IBSC) of Mumbai Veterinary College, Mumbai (No. MUMB071220222542/ Meeting No. 4/S. No 4/Agenda Item No. 1.4/dated 29-07-2025).

Sample Collection, RNA Extraction, and Complementary DNA Synthesis

Twenty brain tissue samples from stray dogs suspected to have died of rabies were collected from the Mumbai region between February 2025 and December 2025. All samples (n=20) were screened using the direct fluorescent antibody test (dFAT) (WOAH, 2023), which identified 14 positive and 6 negative cases. All samples were subsequently stored at -80°C until further molecular evaluation.

A 10% (w/v) tissue homogenate was prepared in sterile phosphate-buffered saline (PBS). Viral RNA was extracted using the TRIzol™-T reagent (SRL Pvt. Ltd., Mumbai, India), incorporating chloroform for phase separation and glycogen as an RNA carrier, following the manufacturer's guidelines. Complementary DNA (cDNA) was synthesised using the PrimeScript™ 1st strand cDNA Synthesis Kit (Takara Bio Inc., Shiga, Japan) utilising random hexamer primers, according to the standard protocol.

Reverse Transcription-PCR

The full-length P gene (894 bp) was amplified in a single reaction using commercially synthesised (Eurofins Genomics India Pvt. Ltd., Bengaluru, India) specific forward (P for: 5'-GAA CCA TCC CAA ATA TGA G-3') and reverse (P rev: 5'-CTA TCT TGC GCA GAA AAT TCA T-3') oligonucleotide primers (Wang *et al.,* 2013). PCR was carried out in a 25 µL reaction volume containing 12.5 µL of EmeraldAmp® GT PCR Master Mix (Takara Bio Inc.), 1.0 µL of each primer, 1.0 µL of template cDNA, and 9.5 µL of nuclease-free water. Thermal cycling was performed with an initial denaturation at 94°C for 4 min, followed by 31 cycles of denaturation at 94°C for 45 s, annealing at 52.5°C for 45 s, and extension at 72°C for 1 min, with a final extension at 72°C for 5 min. The amplicons were resolved using 1.5% agarose gel electrophoresis supplemented with 0.5 µg/mL ethidium bromide for visualisation of DNA bands.

Sequencing and Sequence Annotation

Five representative amplicons (GSR-1 to GSR-5) were subjected to commercial bi-directional Sanger sequencing (Eurofins Genomics India Pvt. Ltd.) using gene-specific primers. The raw forward and reverse sequencing reads were assembled into high-quality consensus sequences using SeqTrace software (v0.9.0) (Stucky, 2012) and deposited in the NCBI GenBank database (Accession Nos. PZ273776 to PZ273780). Viral identity was confirmed via Basic Local Alignment Search Tool (BLAST) homology searches (Altschul *et al.,* 1990) against the GenBank database. The consensus sequences were translated into amino acid sequences using the ExPASy Translate tool (Artimo *et al.,* 2012) and MEGA 12 software (Kumar *et al.,* 2024b) to verify open reading frames (ORFs) and confirm the absence of premature stop codons. Pairwise amino acid sequence identities were calculated using the sequence identity matrix function in BioEdit (v7.7.1) (Hall, 1999).

Phylogenetic and Deduced Amino Acid Analyses

All five study sequences were subjected to automated genotyping using the RABV-GLUE pipeline (Singer *et al.,* 2018). For comparative phylogenetic analysis, representative global and Indian RABV reference sequences were retrieved from GenBank (Table 1), and multiple sequence alignment was performed using the MUSCLE algorithm in MEGA 12. A Maximum Likelihood (ML) phylogenetic tree was constructed applying the Kimura 2-parameter model with Gamma distribution and Invariant sites (K2+G+I), evaluated with 1000 bootstrap replicates. Estimates of evolutionary divergence were calculated using the *p*-distance method. The geographic distribution of the Arctic-like 1a (AL1a) lineage was visualised using Simple Mappr (Shorthouse, 2010). Evolutionary pressure was evaluated via a codon-based *Z*-test of selection in MEGA 12, estimating the number of synonymous (d_S) and non-synonymous (d_N) substitutions per site using the Nei-Gojobori method with the Jukes-Cantor correction (Nei and Gojobori, 1986), evaluated with 1000 bootstrap replicates (significance set at $p<0.05$). Deduced amino acid sequence alignments were rendered using the ESPript 3.0 web server (Robert and Gouet, 2014) to visually highlight mutational patterns across the full-length P protein in comparison with the SAD B19 vaccine strain.

Table 1: Information for the RABV isolates available in GenBank and included for comparison of the full-length phosphoprotein (P) gene

Host	Place of origin	Year of isolation/ submit	GenBank Accession No.
Human	Sri Lanka	2008	AB569299
Goat	Bangladesh	2010	AB699220
Wolf	Laos	2011	AB981663
Dog	India	1995	AF369310
Cattle	India	1991	AF369330
Human	India	2007	EF437215
Fox	France	1991	EU293115
Cattle	Pakistan	2010	HE802675
Dog	China	2008	HQ450385
Cattle	South Korea	2008	KC171643
Human	UK (Viral Origin: India)	1987	KF154996
Mongoose	India	2014	KY775604
Dog	Thailand	1985	LC717425
Vaccine strain (PV)*	France	-	M13215
Vaccine strain (SAD-B19)*	USA	-	M31046
Racoon	USA	1990	MK540671
Dog	India	2018	OL422907
Spotted deer	New Delhi	2016	OM891790
Horse	Uttar Pradesh	2020	OM891791
Sloth bear	Madhya Pradesh	2021	OM891792
Tiger	Uttarakhand	2017	ON814541
Leopard	Uttar Pradesh	2021	ON814542
Spotted hyena	Rajasthan	2022	ON986305
Dog	Nepal	2023	PP805693
Cattle	Nepal	2023	PP805694
Goat	Nepal	2023	PP805695
Dog	Bengaluru‡	2023	PQ149197
Dog	Bengaluru	2023	PV289009
Dog	Bengaluru	2024	PV387684
Dog	Bengaluru	2024	PV387690
Dog	Bengaluru	2024	PV387691
Dog	Bengaluru	2024	PV387692
Dog	Bengaluru	2024	PV393131
Dog	Pakistan	2023	PV491557
Dog	India	2025	PV584366
Wolf	Pakistan	2023	PV641715

*PV: Pasteur Virus, SAD-B19: Street-Alabama-Dufferin (Clone 19)

‡**Note:** Reference sequences labelled "Bengaluru" refer to direct submissions from the National Institute of Mental Health and Neurosciences (NIMHANS), Bengaluru, Karnataka, sourced from their NNV RAB repository as part of ongoing rabies surveillance in the South Indian region. Due to the absence of specific district-level metadata, "Bengaluru" is used here strictly to denote the submission site and serves as a geographic proxy for the broader Peninsular India lineage.

In silico Three-Dimensional Structural Modelling

To assess the structural impact of the identified amino acid (aa) substitutions, *in silico* three-dimensional (3D) protein modelling was performed using the AlphaFold 3 server (Abramson *et al.,* 2024). Due to the intrinsically disordered nature of the N-terminal and central regions, modelling was specifically targeted at the structured dimerisation and carboxy (C)-terminal domains, which were modelled as a homodimer and monomer, respectively (Mavrakis *et al.,* 2004; Ivanov *et al.,* 2010). Corresponding reference models for the SAD B19 vaccine strain domains were similarly generated. The predicted structures were superimposed, and the aa substitutions were spatially mapped using UCSF Chimera X software (v1.11) (Pettersen *et al.,* 2021). Structural divergence between the study sequences and the SAD B19 reference models was quantified using Root Mean Square Deviation (RMSD).

Results And Discussion

Lineage Assignment and Phylogenetic Analysis

Targeted amplification of the full-length P gene yielded an 894 bp specific amplicon in 14/20 samples (70% positivity) Fig. 1 (Gel image), demonstrating complete concordance with dFAT results and validating the high sensitivity and diagnostic utility of the P gene RT-PCR assay. Automated genotyping (RABV-GLUE) (Table 2) and ML phylogenetic reconstruction (Fig. 3) classified all five study sequences into the well-established Arctic-like 1a (AL1a) lineage. Specifically, the study sequences (Mumbai) formed a strongly supported monophyletic cluster (95-98% bootstrap) within a distinct "West and Peninsular" subclade, grouping closely with reference sequences from Bengaluru and other peninsular regions.

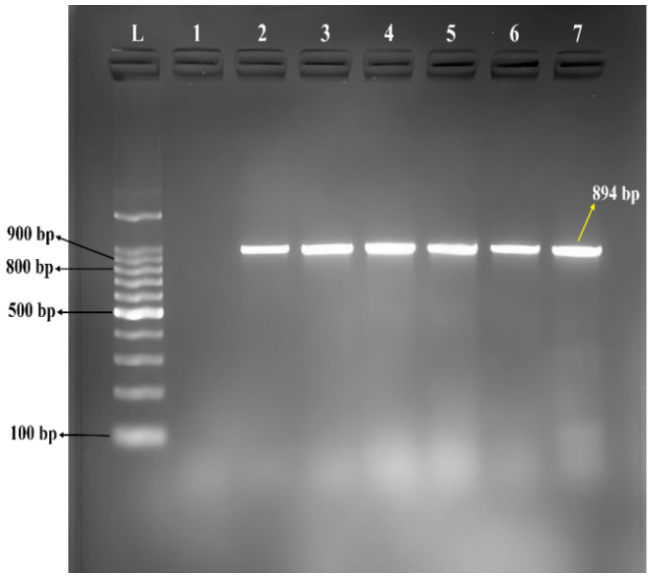

Fig. 1: RT-PCR Amplicons of the Phosphoprotein (P) Gene in Canine Brain Samples. Lane L: 100 bp DNA Ladder; Lane 1: Negative Control; Lane 2: Positive Control; Lanes 3-7: GSR-1, GSR-2, GSR-3, GSR-4, GSR-5.

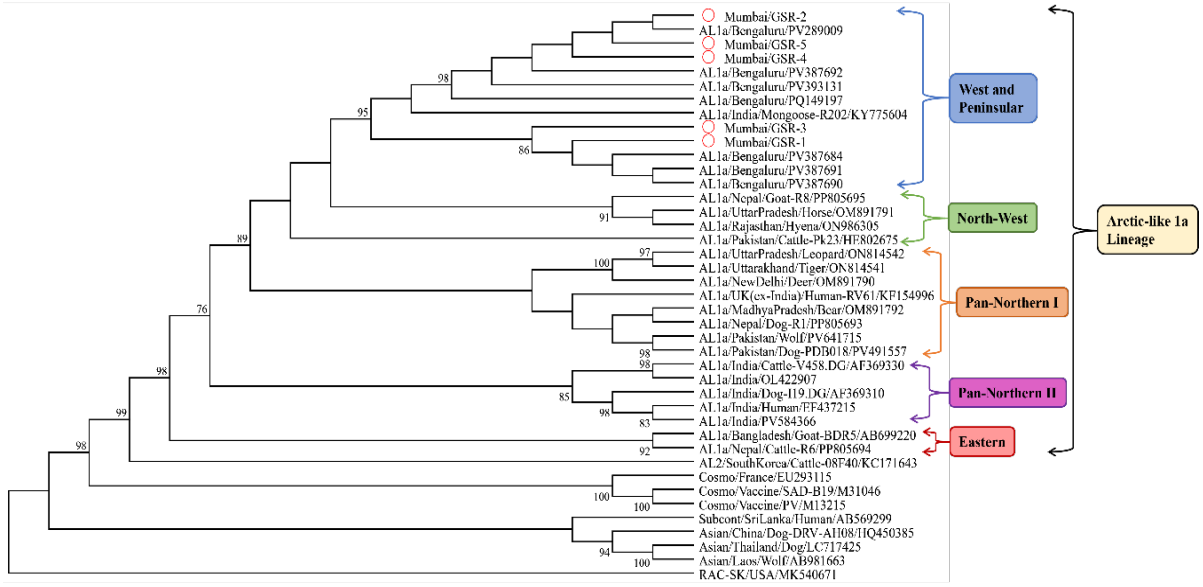

Fig. 2: Phylogeographic distribution of the Arctic-like 1a (AL1a) lineage in the Indian subcontinent based on full-length P gene analysis of study and reference sequences. Blue circles indicate sequence origins; the dashed line represents the phylogenetic North-South divide segregating the West and Peninsular subclade.

Table 2: Lineage assignment of the full-length RABV P gene study sequences using the RABV-GLUE pipeline

Sequence ID	Assigned Clade	Closest full genome reference sequence	P Gene coverage (%)
Mumbai/GSR-1	Arctic AL1a	KF154996	100.00%
Mumbai/GSR-2	Arctic AL1a	HE802675	100.00%
Mumbai/GSR-3	Arctic AL1a	KF154996	100.00%
Mumbai/GSR-4	Arctic AL1a	HE802675	100.00%
Mumbai/GSR-5	Arctic AL1a	HE802675	100.00%

The marked phylogeographic segregation of the "West and Peninsular" subclade from the "North-West", "Pan-Northern", and "Eastern" subclades indicates that ecogeographic barriers likely restrict viral gene flow, driving the independent evolution of localised variants through sustained local transmission (Nagarajan *et al.,* 2006). This result in a broad North-South bifurcation, a phenomenon consistently reported across national surveillance data (Cherian *et al.,* 2015; Varun *et al.,* 2026) and visually delineated in the present study (Fig. 2) (Map) . The interspersed clustering of sequences from domestic dogs, wildlife, and livestock reinforces the hypothesis that rabies epidemiology in India is defined by region-specificity rather than host-specificity (Reddy *et al.,* 2011; Varun *et al.,* 2026); this mixed host distribution highlights the role of the domestic dog as the primary reservoir responsible for viral spillover into other populations (Cherian *et al.,* 2015). Phylogeographic mapping (Fig. 2) (Map) revealed a trans-regional circulation pattern of the AL1a lineage, spanning western, northern, central, and southern India, as well as neighbouring nations (Pakistan, Nepal, and Bangladesh). This broad dispersal confirms that the Mumbai sequences belong to a dominant, genetically stable viral population actively maintained across the Indian subcontinent.

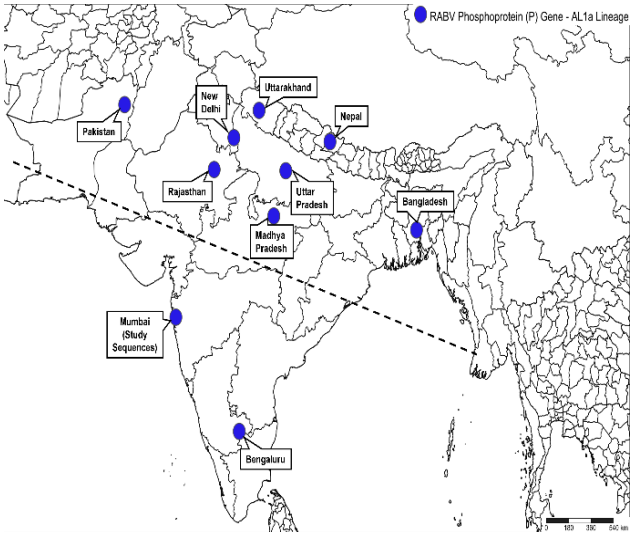

Fig. 3: Maximum Likelihood phylogenetic tree of the full-length RABV Phosphoprotein (P) gene sequences. Bootstrap values > 70% are included. Branch lengths are not to scale. Study sequences GSR-1 to GSR-5 are marked with red circles. Distinct geographical sub-clades within the Indian subcontinent are colour-coded for clarity.

Quantitative estimates of evolutionary divergence corroborated these phylogenetic relationships (Table 3). The lowest genetic divergence (0.47% ± 0.14%) was observed between the study sequences and the Peninsular India subclade, confirming high genetic homogeneity. In contrast, divergence from other AL1a subclades (North-West, Pan-Northern, and Eastern) ranged from 2.19% to 3.82%, reflecting the phylogeography of the virus within the country. Notably, the study sequences demonstrated a substantial 13.43% ± 1.04% divergence (~86.6% nucleotide identity) from the Cosmopolitan lineage, which includes the SAD B19 vaccine strain. This marked genetic distance highlights the

significant evolutionary drift of circulating field viruses from their vaccine ancestors.

Table 3: Estimates of Evolutionary Divergence (%) between the P gene sequences of the study samples and global reference lineages

Sr. No.	Groups	Divergence ± SE (%)*
1	AL1a Lineage- Peninsular Subclade	0.47 ± 0.14
2	AL1a Lineage- North–West Subclade	2.19 ± 0.36
3	AL1a Lineage- Pan–Northern Subclade I	2.91 ± 0.40
4	AL1a Lineage- Pan–Northern Subclade II	3.38 ± 0.52
5	AL1a Lineage- Eastern Subclade	3.82 ± 0.56
6	AL2 Lineage	7.95 ± 0.88
7	Cosmopolitan Lineage	13.43 ± 1.04
8	Asian Lineage	15.80 ± 1.08
9	Indian Subcontinent Lineage	17.48 ± 1.25
10	Raccoon–Skunk Lineage	20.92 ± 1.30

*Values represent the mean number of base differences per site between groups.

Deduced Amino Acid Analysis and Evolutionary Pressure

The deduced amino acid (aa) sequences confirmed a conserved P protein length of 297 residues (Conzelmann *et al.,* 1990) (Fig. 4), sharing 91.2-91.5% aa identity with the SAD B19 reference strain and a 99.6%-100% aa identity amongst themselves. This high degree of sequence conservation is consistent with previous global and national RABV P gene characterisations, which record >80% to 99% aa identities among closely related isolates (Nadin-Davis *et al.,* 2002; Wang *et al.,* 2013). Compared to SAD B19, the study samples exhibited 26 conserved non-synonymous aa substitutions. Despite this genetic drift, a codon-based *Z*-test of selection yielded a statistically significant *Z*-score of 10.99 ($p<0.001$), rejecting neutral evolution in favour of strong purifying (negative) selection (Kumar *et al.,* 2024a; Varun *et al.,* 2026).

Due to this purifying selection, the observed mutations were largely restricted to the intrinsically disordered N-terminal and central flexible regions, corresponding to Variable Domains 1 (VD1, residues 61-80) and 2 (VD2, residues 134-180) (Fig. 4) (Kim *et al.,* 2012). Conversely, critical functional motifs remained strictly conserved. These included the Large (L) protein binding site (residues 1-19), the short lysine-rich Nucleoprotein (N) binding segment (FSKKYKFP, residues 209-216), and the cytoplasmic dynein (LC8) binding motif (KSTQT, residues 144-148) implicated in retrograde axonal transport (Fig. 4) (Kim *et al.,* 2012; Wang *et al.,* 2013).

Within the conserved domains (CD1 and CD2) (Wang *et al.,* 2013), two clade-defining mutations, E165D and A267G (Fig. 4), were observed across all study sequences, aligning with recent large-scale surveillance data defining the molecular signatures of the Arctic clade (Kumar *et al.,* 2024a). The strict

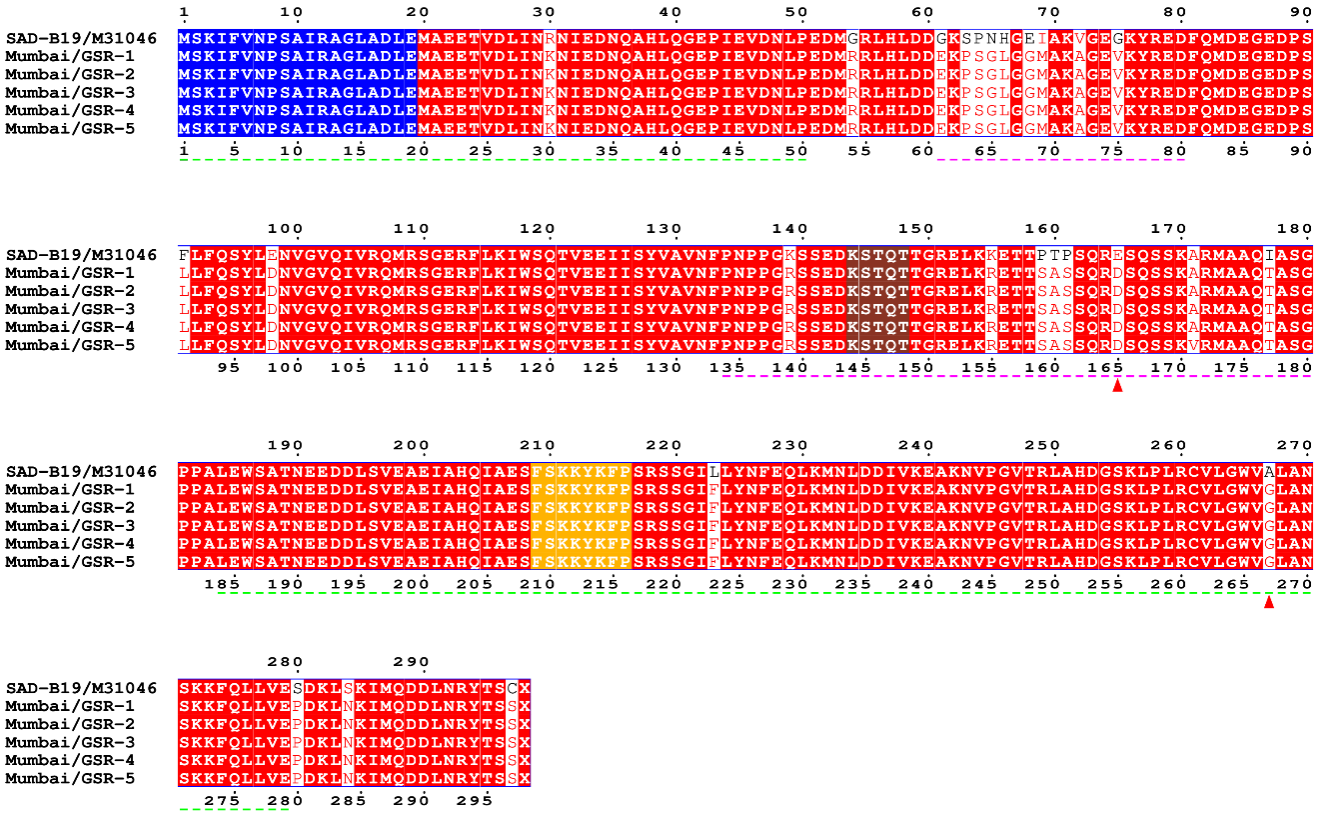

Fig. 4: P protein amino acid alignment of study sequences (GSR-1 to 5) versus the SAD-B19 reference. Annotated features include: strictly conserved residues (red background), L-binding domain (blue), dynein-binding motif (brown), lysine-rich segment (orange), conserved/variable domains (green/pink dashed lines), and clade-defining mutations E165D and A267G (red triangles).

conservation of A267G within the C-terminal domain, a region critical for N-RNA template binding (Mavrakis *et al.,* 2004), highlights its importance to the structural stability of the Arctic-like lineages. Additionally, mutations such as the G54R substitution in the Nuclear Export Sequence (NES), which may influence nucleocytoplasmic trafficking of the P protein (Tudhope *et al.,* 2025), and the A171V variation (GSR-5), underscore the ongoing, localised microevolution of the RABV P gene in Western India.

In silico Structural Modelling and Conformational Stability

Given the high degree of amino acid sequence conservation among study sequences, a representative sequence (GSR-3) was selected for *in silico* analysis. 3D models of the GSR-3 dimerisation and C-terminal domain were superimposed onto the respective SAD B19 reference models via UCSF Chimera X. In the dimerisation domain (residues 91-133) (Fig. 5), GSR-3 exhibited two mutations (F91L and E98D). Structural alignment with the vaccine strain revealed high topological conservation with an RMSD value of 0.154 Å. Hydrophobic surface mapping revealed conservation of physicochemical properties (F91L-hydrophobic to hydrophobic change; E98D-acidic to acidic change), and no alteration in the coiled-coil folding and the hydrophobic interface essential for stable homodimerisation and polymerase interaction (Ivanov *et al.,* 2010).

Within the globular C-terminal domain (CTD), GSR-3 exhibited five substitutions (L223F, A267G, S280P, S284N, and C297S). Superposition confirmed strong conformational stability (RMSD = 0.414 Å; Fig. 6). Although hydrophobic mapping revealed minor localised shifts, such as increased aromatic bulk (L223F) and backbone rigidity (S280P), overall topological conservation validated the structural and functional stability of the CTD nucleocapsid interaction surface (Mavrakis *et al.,* 2004).

Conclusion

In conclusion, the present study provides the first comprehensive genetic and structural analysis of the complete RABV P gene in Mumbai, confirming the endemic circulation of the AL1a lineage within a "West and Peninsular" subclade. While these field variants exhibit significant evolutionary divergence from standard vaccine strains, strong purifying selection and computational modelling confirm the strict conservation of functionally critical motifs and 3D spatial architecture. Ultimately, these findings validate the diagnostic reliability of P gene-based

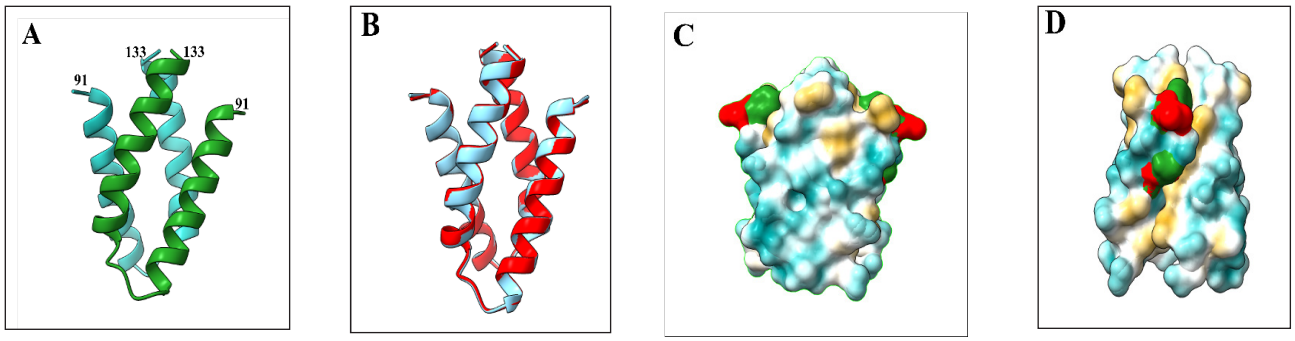

Fig. 5: *In silico* 3D structural modelling of the RABV P protein dimerisation domain (sample GSR-3). (A) Ribbon representation of the predicted homodimer chains (blue and green). (B) Superposition of GSR-3 (red) and SAD B19 (cyan) demonstrating topological conservation (RMSD = 0.154 Å). (C, D) Front and side views of the superimposed models with hydrophobic surface mapping. Substituted residues in GSR-3 (red) and corresponding original residues in SAD B19 (forest green) are highlighted to illustrate spatial and physicochemical alterations.

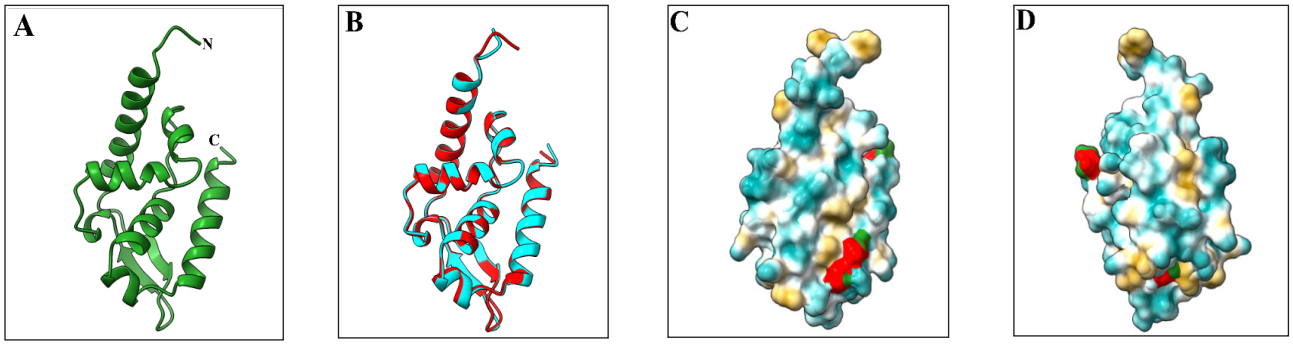

Fig. 6: *In silico* 3D structural modelling of the RABV P protein C-terminal domain (CTD) from sample GSR-3. (A) Ribbon representation of the predicted CTD (green) indicating amino- and carboxy-termini. (B) Superposition of GSR-3 (red) and the SAD B19 reference (cyan) showing high structural conservation (RMSD = 0.414 Å). (C, D) Hydrophobic surface representations (front and 180° views) highlighting localised topography alterations between substituted GSR-3 residues (red) and original SAD B19 residues (forest green).

RT-PCR and highlight a delicate evolutionary balance; RABV undergoes regional genetic drift to adapt to local ecologies while preserving its core structural machinery, underscoring the necessity for sustained, comprehensive molecular surveillance across diverse geographic zones to fully decipher the microevolutionary dynamics of RABV in India.

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ANNOUNCEMENT - III
FELLOWS AND ASSOCIATE FELLOWS AWARDS: SVSBT-2026

(For details please visit society's website: https://www.svsbt.com)

Applications in the prescribed formats are invited from the **Life Members** of the Society for the **Fellows and Associate Fellows Awards of SVSBT-2026.** The awards will be honoured to the deserving candidates during the inaugural ceremony of ***XIII Annual Convention*** and ***National Conference*** to be held **during October 6-7, 2026** at College of Veterinary Science, **BASU, Patna-800014,** Bihar, India.

The application format can be had on request from the President, SVSBT by e-mail at ajdhami59@gmail.com or WhatsApp No. 9898262498. The **application must be submitted only as soft copy in 2 PDFs** to the President, SVSBT, Anand, Gujarat at ajdhami59@gmail.com. **Part-1:** Application in detail along with self-assessed scorecard, and **Part-2:** All other supporting documents for each claim, including only the first page of published articles/books/manuals etc. **on or before 31st July, 2026**. **It is mandatory that the applicant must be LM of the Society**, or else he/she should become life member by due date of application through paying prescribed fees online in the UCO Bank A/C as detailed below. Incomplete applications and those received after the due date will not be entertained. SVSBT reserves the rights for the final selection of the awardees.

Eligibility Criteria & Rules for SVSBT FELLOW AWARD

- The applicant must be a life member of the SVSBT, If not, must register before due date of application.
- Applicant must have a minimum experience of 25 years in the field of teaching research and extension, and should be above 50 years of age, as on the last date of application.
- He/She should be Indian national, who is working in the field of Veterinary Science & Animal Biotechnology & its Allied/Corporate sector in India.
- Awardees may also be nominated by the executive committee from the corporate/ entrepreneurs, on the basis of distinguished contribution in scientific arena of Veterinary Science & Animal Biotechnology towards development of animal sciences.
- Application must include Full details as per the format that can be had from the President SVSBT on request and submitted online in PDF format with all necessary documents/evidences of each claim made to: ajdhami59@gmail.com **on or before 31st July, 2026**.
- Processing fee Rs. 2000/- is to be paid online in SVSBT A/C by the applicant before submission of application.
- Selected candidate shall pay Rs. 7000/- online within 15 days of intimation of results

Eligibility Criteria & Rules for SVSBT ASSOCIATE FELLOW AWARD

- The applicant must be having MVSc/PhD degree in Veterinary Science or Animal Biotech, and life membership of the SVSBT
- Applicant should be between 40 and 50 years of age on the last date of application.
- He/She should be Indian national, who is working in the field of Veterinary Science & Animal Biotechnology in India
- Application must include Full details as per the format that can be had from the President SVSBT on request, and submitted online in PDF format with all necessary documents/evidences of each claim made to: ajdhami59@gmail.com **on or before 31st July, 2026**.
- Processing fee Rs. 1000/- is to be paid online in SVSBT A/C by the applicant before submission of application.
- Selected candidate shall pay Rs. 5000/- online within 15 days of intimation of results.

***Bank Details for Processing Fee*: Online** Payment **in favour of SVSBT**, **payable at UCO Bank, Anand Branch, Account No. 18400110008843, IFSC code: UCBA0000082.**