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Emergence of Novel *Shamonda* Virus Lineages: Clinical Impacts, Vectors, and One Health Surveillance

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ABSTRACT

The summer 2026 outbreak of the *Shamonda* virus (SHAV) across Europe serves as a timely warning that the epidemiological boundaries of animal viruses carried by vectors are neither stable nor easily foreseeable. The *Shamonda* virus is an enclosed, negative-sense, single-stranded RNA virus that is a member of the Simbu serogroup, genus *Orthobunyavirus*, and family Peribunyaviridae. This article aimed to cover the Shamonda virus briefly.

Keywords: soybean meal; poultry feed formulation; proximate analysis; protein solubility; ingredient quality control

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Introduction

Shamonda virus (SHAV) is an arthropod-borne *orthobunyavirus*; transmission occurs via hematophagous *Culicoides* biting midges that primarily affect ruminants, causing acute production losses and congenital defects [1,2]. First isolated in West Africa during the 1960s, SHAV gained widespread veterinary concern following historical detections in Asia and an unexpected emergence across Central Europe [3,4]. In adult dairy cattle, acute infection induces a transient febrile syndrome marked by watery diarrhea, lethargy, and milk production losses ranging between 15% and 75% [1]. In neonatal ruminants, vertical transmission during gestation poses a significant teratogenic risk, resulting in abortion, stillbirth, hydranencephaly, and severe arthrogryposis [1,2]. In cases of equine encephalitis and caprine abortion tissues, viral genome identification verifies active neurotropism [1,5].

Virology and Genomic Architecture

The *Shamonda* virus is a member of the *Simbu* serogroup of the species *Orthobunyavirus schmallenbergense* and the family *Peribunyaviridae* [1, 5]. Large (L), Medium (M), and Small (S) segments make up the virus's tri-segmented, negative-sense, single-stranded RNA genome [1,6]. Sequence analyzes reveal that the M segment codes for viral surface glycoproteins with various non-coding terminal topologies and antigenic diversity [7, 8]. High levels of genomic reassortment between SHAV and related *Orthobunyaviruses* frequently occur in mammalian host settings, as shown by in vitro co-infection models [6,9].

Global Emergence and Epidemiology

Historically detected in Nigeria in 1965, SHAV was subsequent observed in sentinel herds and biting midges in Japan and coastal Australia [1,4].

- Transmission relies on hematophagous vectors, predominantly Culicoides biting midges, which carry viral RNA under field conditions [1,5].
- Long-term serological surveillance in New South Wales established sustained transmission expanding across coastal regions since at least 2012 [5].
- Metagenomic profiling identified the emergence of two distinct clades (SHAV-EU1 and SHAV-EU2) across Central and Southern Europe [7,9].
- Pre-existing immunity against SBV provides no cross-protective neutralization due to critical amino acid divergences in the M segment [7,9].

Vector Transmission Dynamics and Reservoir Ecology

According to epidemiological trends, infectious viruses and viral RNA are frequently isolated from hematophagous midges, particularly *Culicoides* species, which are the main source of transmission [1,7]. *Shamonda* virus sequences were also found in larval mosquito populations in India by molecular metagenomics, demonstrating intricate arthropod connections [10]. Transmission extends beyond past endemic zones and follows seasonal progressions linked to temperature-driven midge activity [1].

Clinical Presentations Across Ruminants and Equids

In adult cattle, epizootic incursions of European *Shamonda* virus (SHAV) lineages cause an acute, brief disease characterized by severe diarrhea, pyrexia above 40 °C, and lethargy [7,9]. Across impacted cattle, milk output shows severe declines ranging from 15% to 75% [1,7]. Teratogenic effects are similar to previous *Simbu* serogroup outbreaks, but acute illness in mature cattle is still self-limiting [1]:

- Exposure during pregnancy results in hydranencephaly and arthrogryposis, which can lead to fetal abortion, stillbirth, and severe abnormalities of the central nervous system [1,3].
- Fetal caprine losses occur alongside severe maternal disease, with viral RNA detected directly in the brain and lung tissue of aborted fetuses [1,5].
- Equids manifest fatal encephalitis and ataxia, yielding viral genome detections from cerebral tissue despite negative blood and cerebrospinal fluid assays [1,12].

Surveillance, Prevention, and Zoonotic Assessment

Epidemiological control is challenged by the absence of commercial vaccines or targeted antivirals for livestock [1,9]. Immunoinformatics approaches have modeled multi-epitope subunit candidate vaccines targeting Toll-like receptor-3 to elicit adaptive humoral and cellular responses [13]. Cross-reactivity among *Simbu* group members also complicates standard serological surveillance, emphasizing the necessity of untargeted metagenomic sequencing and three-segment molecular characterization [1,9,14].

Current surveillance shows no direct evidence of *Shamonda* virus infection in humans [1,13]. However, due to the known zoonotic transmission and human neurovirulence of related *Simbu orthobunyaviruses*, such as *Oropouche* and *Shuni* viruses, continuous One Health monitoring is advised [9,15]. To track developing lineages and stop outbreaks of neurological and reproductive diseases, coordinated surveillance across vector populations, animals, and domestic ruminants is still crucial [7,16].

Conclusion

In conclusion, *Shamonda* virus represents a re-emerging veterinary threat whose teratogenic and economic burdens demand integrated global surveillance, updated diagnostic panels, and targeted vaccine development.

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