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Therapeutic potential of anti-inflammatory diets in Nipah Virus–related complications

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Abstract
Nipah virus (NiV) is a highly lethal, biosafety level-4 paramyxovirus causing acute respiratory distress syndrome (ARDS) and fulminant encephalitis, with case fatality rates up to 100%. Given the absence of globally licensed vaccines or definitive antiviral therapies, clinical management relies on supportive care to battle the virus's primary driver of mortality: a host-mediated, hyper-inflammatory cytokine storm that causes systemic vasculitis and severe tissue damage. This review explores the therapeutic potential of anti-inflammatory diets rich in immunomodulatory bioactives as an accessible, dual-action adjunctive therapy. Structural in silico docking models indicate that dietary flavonoids and polyphenols, such as naringin and luteolin, successfully bind to critical NiV entry and fusion proteins, outperforming traditional small-molecule options like ribavirin. Concurrently, bioactives like curcumin and 6-gingerol suppress key pro-inflammatory mediators (PGE_2 and TXA_2) and rate-limiting viral enzymes, effectively mitigating neutrophil infiltration in pulmonary tissues and stabilizing the blood–brain barrier against cerebral vasculitis. Ultimately, while nutritional interventions cannot replace standard critical care or biosafety containment, targeted anti-inflammatory diets represent a highly scalable, non-toxic strategy to protect tissue integrity and prevent multi-organ failure, though *in vivo* animal challenges remain necessary to translate these promising dynamics into validated clinical outbreak protocols.

Keywords: Nipah virus, anti-inflammatory diet, cytokine storm, phytochemicals, vasculitis, encephalitis

Introduction

Nipah virus (NiV) is a highly pathogenic, biosafety level-4 zoonotic paramyxovirus that triggers severe, often fatal disease outbreaks in humans (Rathish, 2026; Singh *et al*., 2019) [4, 7]. Characterized by case fatality rates ranging from 40% to 100%, NiV causes acute respiratory distress syndrome (ARDS) and fulminant, necrotizing encephalitis (Enmozhi *et al*., 2024; Scotto, 2026) [1, 6].

The primary pathophysiology of NiV does not merely stem from direct viral replication, but from a hyper-inflammatory immune response. Following entry via host ephrin-B2 and ephrin-B3 receptors, the virus exhibits intense endothelial and neuronal tropism, triggering systemic vasculitis and a catastrophic breakdown of the blood–brain barrier (BBB) (Scotto, 2026) [6]. Because there are currently no globally licensed, definitive antiviral therapies or human vaccines available, treatment remains fundamentally supportive (Rababi & Nag, 2023; Scotto, 2026) [3, 6].

Consequently, manipulating the host's inflammatory cascade through adjunctive therapies has gathered immense scientific interest. In particular, a targeted anti-inflammatory diet rich in immunomodulatory bioactives represents a promising strategy to mitigate the devastating tissue damage and multi-organ failure caused by NiV-induced cytokine storms.

Pathogenic Cascades and the Need for Anti-Inflammatory Intervention

When NiV infects endothelium and central nervous system (CNS) tissues, it deploys specific viral proteins that successfully block host interferon signaling, paralyzing the early innate immune defense (Scotto, 2026) [6]. Deprived of early control, the host system compensates by unleashing a dysregulated, massive wave of pro-inflammatory cytokines and chemokines.

- **In the Lungs:** This hyper-inflammation induces severe alveolar damage, pulmonary edema, and ARDS, which frequently progresses to rapid multi-organ failure (Rathish, 2026; Scotto, 2026) [4, 6].

- **In the Brain:** Microglial activation and endothelial disruption produce widespread cerebral vasculitis. This drives acute, subacute, or delayed-onset relapsing encephalitis, leaving survivors with permanent neurological deficits like epilepsy and personality changes (Scotto, 2026) [6].

Because standard small-molecule options like ribavirin offer limited efficacy and are plagued by severe side effects—such as anemia and teratogenicity (Rababi & Nag, 2023) [3]—dietary phytochemicals are being heavily evaluated for their dual-action safety profile: protecting tissue via anti-inflammatory pathways while simultaneously inhibiting viral machinery (Santhi *et al*., 2021) [5].

Dietary Phyto-Nutrients as Dual Antiviral & Anti-Inflammatory Agents

Anti-inflammatory diets capitalize on dense concentrations of plant-derived bioactive molecules (flavonoids, polyphenols, and terpenoids) that possess distinct structural binding affinities for Nipah virus target proteins while suppressing host inflammatory pathways (Enmozhi *et al*., 2024; Rababi & Nag, 2023) [1, 3].

Curcumin (Turmeric)
Curcumin is a premier polyphenol widely recognized for suppressing cytokine gene expression at both the mRNA and translational levels (Vahid & Rahmani, 2021) [8]. It serves as an inosine-5′-monophosphate dehydrogenase (IMPDH) inhibitor, which cuts off a rate-limiting enzyme required for viral nucleotide synthesis (Vahid & Rahmani, 2021) [8]. Clinically, curcumin reduces cellular inflammatory infiltration, improves tissue repair, and dampens the prostaglandin synthesis that drives systemic vasculitis (Vahid & Rahmani, 2021) [8].

6-Gingerol (Ginger)
Derived from the rhizome of *Zingiber officinale*, 6-gingerol is highly pharmacologically active in managing respiratory inflammation. Research demonstrates that ginger bioactives selectively downregulate the production of inflammatory mediators like prostaglandin E2 (PGE_2) and thromboxane A2 (TXA_2), reducing myeloperoxidase activity and cellular infiltration in lung tissue (Vahid & Rahmani, 2021) [8]. Concurrently, structure-based molecular docking models show that 6-gingerol binds natively to the Nipah Virus Fusion (NiV-F) outer membrane protein, obstructing the viral entry mechanism into host cells (Verma *et al*., 2021) [9].

Flavonoids (Naringin, Luteolin, and Quercetin)
Flavonoids heavily prominent in anti-inflammatory dietary profiles have demonstrated significant therapeutic potential against Henipaviruses:

- **Naringin:** Advanced in silico molecular docking models revealed that naringin achieves exceptionally high binding affinity ($-9.19 \text{ kcal mol}^{-1}$) against the Nipah virus attachment glycoprotein (NiV G), significantly outperforming the standard control drug ribavirin (Rababi & Nag, 2023) [3].

- **Luteolin:** Luteolin acts as a heavy biochemical antioxidant and anti-inflammatory agent, showing stable negative free-binding energies when targeted against the NiV-F protein to block cell-to-cell fusion (Verma *et al*., 2021) [9].

Mechanisms of Dietary Mitigation in NiV Complications
The therapeutic efficacy of an anti-inflammatory diet during an active NiV infection operates through three primary physiological axes:

Table 1: Dietary Bioactive Compounds and Their Therapeutic Targets in Nipah Virus–Associated Pathophysiology

Therapeutic Target	Dietary Bioactive Component	Primary Mechanism of Action
Endothelial Vasculitis	Polyphenols & Flavonoids	Stabilizes the blood–brain barrier; limits endothelial cell activation and adhesion molecule expression (Scotto, 2026).
Pulmonary ARDS	Gingerols & Curcuminoids	Suppresses PGE_2 and TXA_2; limits neutrophil infiltration into pulmonary alveoli (Vahid & Rahmani, 2021).
Glycoprotein Entry Blockade	Naringin & Quercetin	Competitively binds to host cell entry receptors (ephrin-B2/B3) and viral attachment G proteins (Rababi & Nag, 2023; Scotto, 2026).

By modifying these pathways, dietary compounds help ensure that the host immune response remains localized and controlled, rather than escalating into a deadly, systemic cytokine storm that leads to tissue necrosis and coma.

Table 2: Therapeutic Potential of Anti-Inflammatory Diets in Nipah Virus–Related Complications (Ang *et al*., 2018; Clayton, 2017)

Nipah Virus–Related Complication	Pathophysiological Features	Potential Dietary Components	Proposed Mechanisms	Expected Clinical Benefits
Neuroinflammation and encephalitis	Cytokine storm, neuronal damage, blood-brain barrier disruption	Omega-3 fatty acids (fish oil, flaxseed), polyphenols (berries, green tea)	Suppression of NF-κB signaling, reduction of pro-inflammatory cytokines (IL-6, TNF-α)	Reduced neuroinflammation and improved neurological recovery
Oxidative stress	Excess reactive oxygen species (ROS) production	Vitamins C and E, selenium, carotenoids, colorful fruits and vegetables	Antioxidant scavenging of ROS and enhancement of endogenous antioxidant enzymes	Protection against cellular and tissue damage
Endothelial dysfunction	Vascular inflammation and increased permeability	Mediterranean diet, olive oil, nuts	Improvement of endothelial function and reduction of inflammatory mediators	Enhanced vascular integrity and reduced edema
Immune dysregulation	Hyperactivation or impaired immune response	Vitamin D, zinc, probiotics, fermented foods	Modulation of innate and adaptive immune responses	Improved immune balance and host defense
Respiratory complications	Pulmonary inflammation and acute respiratory distress	Omega-3 fatty acids, fruits, vegetables, whole grains	Reduction of inflammatory mediators and support of lung tissue repair	Improved respiratory outcomes
Secondary metabolic disturbances	Increased catabolism and nutritional deficiencies	High-quality proteins, legumes, nuts, whole grains	Preservation of muscle mass and support of recovery processes	Faster recovery and improved nutritional status
Gastrointestinal dysfunction	Gut microbiota imbalance and inflammation	Prebiotics, probiotics, dietary fiber	Restoration of gut microbial diversity and barrier function	Improved gut health and immune regulation

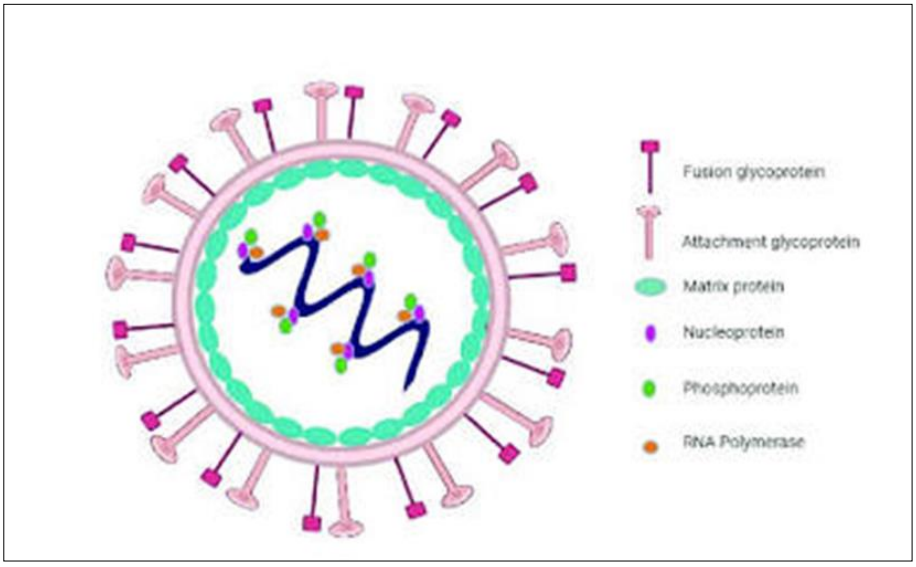

Fig 1: Structural Diagram of the Nipah Virus

Conclusion and Future Clinical Outlook

While nutritional modifications and anti-inflammatory diets cannot replace full-scale biosafety containment and intensive care supportive measures, they offer a highly scalable, non-toxic, and accessible adjunctive therapy. Dietary agents like curcumin, gingerols, and structural flavonoids exhibit a critical dual-therapeutic nature: they actively inhibit viral fusion and attachment proteins while protecting critical brain and lung tissue from the host's own hyper-inflammatory responses (Rababi & Nag, 2023; Vahid & Rahmani, 2021; Verma *et al*., 2021) [3, 8].

Moving forward, *in vivo* animal model challenges are necessary to accurately translate these promising in silico binding dynamics and traditional anti-inflammatory models into validated clinical protocols for managing Henipavirus outbreaks.

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