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Review Article

Medicinal Plants as a Promising Source of Antiviral Agents Against Influenza a Virus: A Review

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ABSTRACT

Influenza A virus (IAV) causes seasonal epidemics and occasional pandemics. Its continual genetic variation, together with resistance to the available neuraminidase and M2 inhibitors, limits the effectiveness of current vaccines and antiviral drugs, and this has renewed interest in medicinal plants. Traditional medicines contain flavonoids, phenolics, terpenoids, alkaloids, saponins and organosulfur compounds that can act at several stages of the viral life cycle. This review summarises the biology and life cycle of IAV, the clinical burden of influenza, and the experimental evidence for six medicinal plants: Tulsi (*Ocimum tenuiflorum*), Neem (*Azadirachta indica*), Turmeric (*Curcuma longa*), Garlic (*Allium sativum*), Ginger (*Zingiber officinale*) and Licorice (*Glycyrrhiza glabra*). Their principal constituents, reported antiviral activities and proposed mechanisms are discussed. These mechanisms include inhibition of viral attachment and entry, interference with viral replication, inhibition of neuraminidase and modulation of host inflammatory signalling. Most of the evidence comes from in-vitro, animal and computational studies, and direct inhibition of the M2 channel has not been firmly shown for any of these plants. Standardised extracts, pharmacokinetic and toxicity studies and well-designed clinical trials are needed before plant-derived compounds can be considered alternatives to established antivirals.

INTRODUCTION

1.1 Viral Diseases and the Need for New Antivirals

Viruses are major causes of human disease and have also been associated with certain cancers and chronic disorders. The emergence and re-emergence of viral infections, driven by factors such as global travel and urbanisation, pose significant public health challenges. Outbreaks of diseases caused by viruses such as dengue, influenza, measles, SARS and West Nile virus

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highlight the need for effective antiviral strategies. Moreover, limited vaccines and antiviral drugs, along with the development of drug-resistant viral strains, reduce the effectiveness of current treatments. There is therefore a need to identify safe, effective, affordable and novel antiviral agents for the prevention and management of viral infections.[1]

Herbal medicines and purified natural products provide a rich resource for novel antiviral drug development. Identification of the antiviral mechanisms of these natural agents has shown where they interact with the viral life cycle, such as viral entry, replication, assembly and release, and how they target virus-host interactions.[1]

1.2 Limitations of Current Control Measures

Annual vaccination is the most effective preventive measure, but the virus evolves constantly, so the vaccine composition is revised twice a year. Vaccine mismatch, slow production against novel subtypes and incomplete protection in the elderly leave a gap that antivirals must fill.[2] The approved antivirals themselves have limits. Only a few drug classes exist, and resistance has emerged against each of them. Neuraminidase (NA) inhibitors such as zanamivir, oseltamivir and peramivir are the mainstay of treatment, yet the constant emergence of drug-resistant strains and the threat of pandemics have weakened their role.[3]

1.3 Influenza Viruses

Influenza viruses have a global distribution and are both human and animal pathogens. Human influenza viruses cause annual seasonal epidemics, which are highly contagious respiratory infections that can lead to severe illness and life-threatening complications in high-risk groups. Occasionally, a new human influenza virus strain arises from an animal origin and spreads rapidly through human populations that have no pre-existing immunity, causing excessive morbidity and mortality worldwide; this is known as a pandemic.[4]

Humans can also be sporadically infected by animal (zoonotic) influenza viruses, most often avian and swine viruses, although these have not yet established sustained infection in humans. In particular, avian H5 and H7 influenza A viruses have caused hundreds to thousands of human infections with a high case fatality rate (30-50%). The possibility that these viruses acquire efficient human-to-human transmissibility and cause the next pandemic poses a significant public health risk.[5]

1.4 Types of Influenza Viruses

Influenza viruses belong to the family *Orthomyxoviridae* and are classified into four types: influenza A, B, C and D. Influenza A and B are the principal types responsible for seasonal influenza in humans. Influenza A viruses are further classified into subtypes on the basis of the combination of the surface glycoproteins hemagglutinin (HA) and neuraminidase (NA), whereas influenza B viruses are classified into genetic lineages. Influenza C generally causes milder respiratory disease, while influenza D primarily affects cattle and has not been established as a cause of human disease.[6]

1.5 Structure of Influenza A Virus

The influenza A virion is enveloped and carries two major surface glycoproteins, HA and NA, together with the M2 ion channel embedded in the lipid bilayer. Beneath the envelope lie the matrix protein M1 and the nuclear export protein (NEP), and the interior contains the segmented negative-strand RNA genome associated with the nucleoprotein (NP) and the polymerase proteins PB1, PB2 and PA (Fig 1).[7]

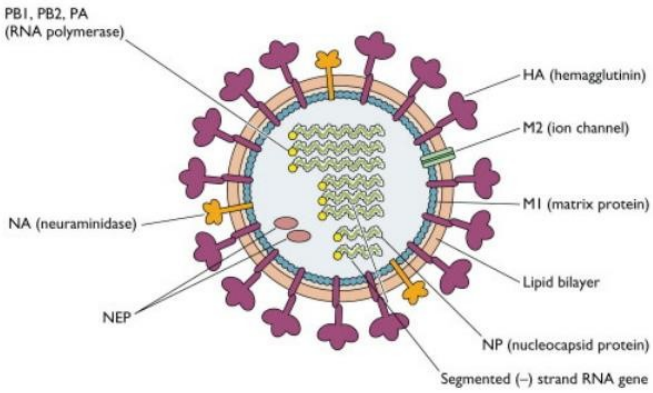

Figure 1: Structure of Influenza a Virus Showing the Two Major Surface Glycoproteins (HA and NA), the Nucleocapsid and Polymerase Proteins (NP, PB1, PB2 and PA), the Matrix Proteins (M1 and M2), the Nuclear Export Protein (NEP), the Lipid Bilayer and the Segmented Negative-Strand RNA Genes.[7]

1.6 Life Cycle and Replication of Influenza A Virus

The life cycle of influenza A virus and the stages at which natural compounds may act against H1N1 are summarised in Fig 2.[8] It can be divided into four broad stages.[9,10]

(1) Attachment and Entry: IAV attaches to sialic acid (SA)-containing receptors on the host-cell surface through HA and enters the cell by receptor-mediated endocytosis, forming an endosome.

(2) Endosomal Acidification and Uncoating: Acidification of the endosome triggers conformational changes in HA that facilitate fusion of the viral envelope with the endosomal membrane. The M2 proton channel promotes acidification inside the virion, allowing the viral ribonucleoprotein complexes (vRNPs) to dissociate from the M1 matrix protein. The released vRNPs enter the cytoplasm and are transported into the nucleus.

(3) Transcription and Genome Replication: In the nucleus, the viral RNA-dependent RNA polymerase complex (PB1, PB2 and PA) transcribes viral mRNA and replicates the genome. Negative-sense viral RNA (vRNA) is copied into complementary RNA (cRNA), which serves as the template for new vRNA. Viral mRNAs are translated into proteins, and HA, NA and M2 are processed through the endoplasmic reticulum (ER) and Golgi apparatus before reaching the host-cell membrane.

(4) Assembly, Budding and Release: Newly synthesised vRNPs associate with viral structural proteins at the plasma membrane, where progeny virions assemble and bud from the infected cell. NA cleaves sialic acid residues, releasing the new virions so that they can infect additional host cells.

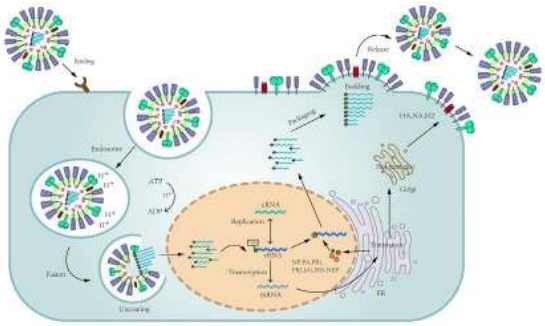

Figure 2: Life Cycle of Influenza Virus and the Targets of Natural Compounds Against Influenza a (H1N1) Viruses.[8]

At a finer resolution, the replication cycle comprises seven discernible phases: attachment, endocytosis, uncoating and membrane fusion, transcription, trans-

lation, replication of the viral RNA and virion budding (Fig 3).[11]

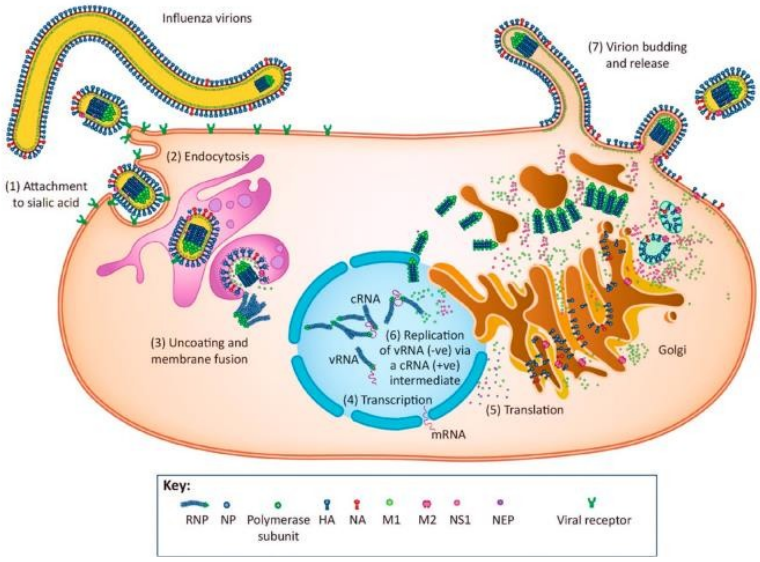

Figure 3: The Replication Cycle of Influenza Virus, Showing Its Seven Phases.[11]

Attachment. Replication begins when HA binds sialic acid on a glycoprotein or glycolipid at the host-cell surface. HA of human-adapted viruses prefers sialic acid joined to galactose through an α-2,6 linkage, which is abundant on human respiratory epithelial cells, whereas avian-virus HA prefers the α-2,3 linkage.[11]

Endocytosis. After attachment, the virion is taken up by clathrin-mediated endocytosis and ends up inside an endosome.[11]

Uncoating and Membrane Fusion. The falling pH of the late endosome (about pH 5) triggers a conformational change in cleaved HA. Its HA2 fusion peptide inserts into the endosomal membrane, the two membranes fuse, and the vRNPs are released into the cytoplasm. At the same time, protons enter the virion through the M2 ion channel; the resulting acidification disrupts the M1-RNP interaction and frees the vRNPs, which are then imported into the nucleus by host importins.[11]

Transcription of the Viral RNA. In the nucleus, the viral polymerase transcribes the negative-sense genome segments into mRNA. Transcription is primed by "cap snatching": PB2 binds the 5′ cap of host mRNA, the PA endonuclease cleaves 10-13 nucleotides downstream, and PB1 extends this capped fragment. Transcription ends with polyadenylation at an oligo-U signal.[11]

Translation of Viral Proteins. Viral mRNAs are exported to the cytoplasm and translated by host ribosomes. HA, NA and M2 are made on the ER and processed in the Golgi apparatus (glycosylation of HA and NA, palmitoylation of HA and M2) before moving to the plasma membrane. Newly made NP and polymerase subunits are imported back into the nucleus.[11]

Replication of the Viral RNA. The polymerase copies vRNA into a positive-sense cRNA intermediate, which is copied back into new vRNA without needing a capped primer. Progeny vRNPs combine with M1 and nuclear export proteins and travel to the cell surface along microtubules. Packaging of all eight segments is thought to depend on segment-specific signals, and particles lacking segments are not fully infectious.[11]

Virion Budding and Release. Budding takes place in cholesterol- and sphingolipid-rich lipid rafts, initiated by M1 accumulating beneath the plasma membrane, with M2 at the edges of the bud contributing to scission. Release depends on NA, which cleaves sialic acid from the cell and viral glycoproteins so that HA no longer holds the progeny virions to the cell surface. This NA step is the target of the neuraminidase inhibitors described in Section 1.2, whereas the M2

channel in the uncoating phase is the target of amantadine and rimantadine.[11]

1.7 Etiology, Transmission and Symptoms

Influenza results from infection with influenza A, B or C virus. Influenza A is generally more pathogenic than influenza B, which primarily infects humans and is an important cause of seasonal influenza. Epidemics of influenza C have been reported, especially in young children, but it usually causes milder respiratory infection. Influenza D primarily infects cattle and other animals. Human influenza is mainly transmitted through respiratory droplets and aerosols generated when an infected person coughs, sneezes, talks or breathes. Close contact with infected individuals, contaminated hands and surfaces, exposure to infected animals, antigenic drift and shift, and seasonal and environmental factors all contribute to its occurrence. Viral genetic variation, host susceptibility, viral subtype, immune status and exposure influence the severity of disease.[12]

The typical symptoms are fever, chills, body aches, cough, headache, sore throat, a runny or stuffy nose and tiredness. Diarrhoea and vomiting occur usually only in children.[12]

1.8 Pathogenesis of Influenza A Virus

IAV initiates infection by attaching, through HA, to sialic acid-containing receptors on susceptible respiratory epithelial cells. After receptor-mediated endocytosis, acidification of the endosome triggers conformational changes in HA, fusion of the viral envelope with the endosomal membrane and release of the vRNPs into the cytoplasm. The vRNPs are transported into the nucleus, where viral RNA transcription and replication occur. Newly synthesised viral proteins and genomic RNA are then transported to the cell membrane, where assembly and budding take place. NA facilitates the release of new virions by cleaving sialic acid residues, supporting viral dissemination and the establishment of infection in neighbouring cells.[13,14] Certain plant-derived flavonoids and biflavonoids have been investigated for their ability to inhibit influenza neuraminidase, indicating a potential target for antiviral drug development.[15]

1.9 Clinical Effects and Complications

Influenza A virus infection primarily affects the respiratory tract and produces a spectrum of illness ranging from mild, self-limiting disease to severe pneumonia, acute respiratory distress syndrome (ARDS), hospitalisation and death. Severity depends on viral characteristics and on host factors such as age, immunosuppression and underlying respiratory disease. A 2024 study reported that influenza affects approximately 10% of the global population annually and that severe infection can progress to acute respiratory infection requiring intensive care.[16] The major effects are as follows.

Respiratory effects. Influenza commonly causes fever, cough, sore throat, myalgia and respiratory inflammation. Severe infection may result in primary viral pneumonia, secondary bacterial pneumonia, ARDS and respiratory failure.[17]

Cardiovascular effects. Influenza can increase the risk of myocarditis, heart failure, arrhythmias, myocardial infarction and stroke. Systemic inflammation, hypoxaemia, endothelial dysfunction and increased coagulation are proposed mechanisms.[18]

Neurological effects. Although less common, influenza may cause encephalitis, encephalopathy, meningitis, seizures, encephalomyelitis and Guillain-Barré syndrome, which can occasionally be severe or life-threatening.[19]

Systemic and extrapulmonary effects. Influenza is not restricted to the respiratory system. A large scoping review identified 50 different clinical conditions associated with influenza exposure, with most associations indicating increased risk of extrapulmonary outcomes.[20]

Hospitalisation and mortality. Severe influenza can lead to hospitalisation and death, particularly among older adults, young children, immunocompromised individuals and people with chronic disease. A systematic umbrella review found a substantial influenza burden, with particularly high morbidity among children aged 5 years or less and high mortality among adults aged 65 years or more.[21]

1.10 Influenza A Virus as a Global Health Threat

IAV is a major global health threat because it is associated with considerable morbidity and mortality in humans and animals.[22] The continuous antigenic evolution of influenza viruses, particularly antigenic drift, challenges effective vaccination and contributes to their persistent circulation.[2] Although antiviral agents such as amantadine and oseltamivir are used for the management of influenza, their clinical usefulness is limited by adverse effects and the emergence of resistance.[3] These limitations have stimulated the search for novel and effective antiviral agents from natural sources, and medicinal plants have attracted considerable attention as potential sources of antiviral compounds and lead molecules for anti-influenza therapies.[23]

2. Medicinal Plants as a Promising Source against Influenza A Virus

Medicinal plants have long been used in traditional systems of medicine for the management of infectious diseases. In recent years they have gained attention as sources of antiviral agents because they contain a wide variety of biologically active secondary metabolites. The phytoconstituents considered responsible for antiviral activity include flavonoids, phenolic compounds, terpenoids, alkaloids, tannins, saponins, lignans, coumarins and organosulfur compounds, which may act against different stages of the viral life cycle and may also modify host-cell responses.[24-26]

Plant-derived compounds may prevent viral attachment and entry, interfere with viral fusion, inhibit viral enzymes such as NA or other viral proteins, suppress viral nucleic acid synthesis, interfere with viral protein synthesis and assembly, or enhance and modulate host immune responses. Medicinal plants may therefore provide multi-target antiviral effects, which makes them important candidates for the discovery of new antiviral molecules.[27] For IAV, several plant-derived compounds have been investigated against HA, NA and the matrix protein M2. Because HA is involved in attachment to sialic-acid receptors and NA in the release of new virions, inhibition of these proteins can interfere with viral entry and release.[28,29]

However, most of the evidence for medicinal plants is still based on in-vitro, animal or mechanistic studies, and plant extracts vary with species, plant part, extraction method and phytochemical composition. Further standardisation, toxicity studies, pharmacokinetic studies and well-designed clinical trials are required before medicinal plants can be considered substitutes for established antiviral medicines.[24,30] The six plants considered in this review are summarised in Table 1.

Table 1: Profile of the Six Medicinal Plants Reviewed

Sr. No.	Plant (common name)	Family	Part used	Major constituents	Influenza-related evidence
1	*Ocimum tenuiflorum* L. (syn. *O. sanctum* L.); Tulsi, holy basil	Lamiaceae	Leaves (whole plant also used)	Eugenol, ursolic acid, oleanolic acid, rosmarinic acid, luteolin, apigenin	Antiviral activity against H1N1 in embryonated-egg and in-vitro models.[31,32]
2	*Azadirachta indica* A. Juss.; Neem	Meliaceae	Leaves (also bark, seeds, fruits, flowers, roots)	Azadirachtin, nimbin, nimbolide, gedunin, quercetin, rutin, nimbaflavone	Predicted interaction with influenza NP and NS1 in docking studies.[33,34]
3	*Curcuma longa* L.; Turmeric, haldi	Zingiberaceae	Rhizome	Curcumin, demethoxycurcumin, bisdemethoxycurcumin, turmerones, zingiberene	Inhibits IAV infectivity, adsorption, replication and inflammation.[35-37]
4	*Allium sativum* L.; Garlic, lahsun	Amaryllidaceae	Bulb (cloves)	Alliin, allicin, ajoene, DADS, DATS, SAC, SAMC, quercetin	Extracts inhibited influenza A in cell models.[38]

Sr. No.	Plant (common name)	Family	Part used	Major constituents	Influenza-related evidence
5	*Zingiber officinale* Roscoe; Ginger, adrak	Zingiberaceae	Rhizome	6-, 8- and 10-Gingerol, 6-, 8- and 10-shogaol, zingerone, zingiberene	Extract inhibited growth of A/Aichi/2/68; 6-gingerol inhibited H1N1 and NA.[39,40]
6	*Glycyrrhiza glabra* L.; Licorice, mulethi	Fabaceae	Root and rhizome	Glycyrrhizin, glycyrrhetinic acid, liquiritin, isoliquiritin, glabridin	Glycyrrhizin reduced viral uptake and H5N1-induced inflammation.[41,42]

DADS, diallyl disulfide; DATS, diallyl trisulfide; SAC, S-allylcysteine; SAMC, S-allylmercaptocysteine; NP, nucleoprotein; NS1, non-structural protein 1.

2.1 Tulsi (*Ocimum tenuiflorum* L.)

Tulsi (family Lamiaceae; synonym *Ocimum sanctum* L.), also known as holy basil or sacred basil, is used mainly as the leaf, although the whole plant is also used traditionally (Fig 4). Its major constituents include eugenol, ursolic acid, oleanolic acid, rosmarinic acid, luteolin, apigenin, quercetin, orientin, vicenin, β-sitosterol and limonene. These belong to the flavonoid, phenolic acid, terpenoid, triterpenoid, tannin, alkaloid, saponin and glycoside classes, and the plant shows antioxidant, anti-inflammatory, antimicrobial, immunomodulatory, antidiabetic, hepatoprotective, anticancer and analgesic activities.[43]



Figure 4: Tulsi (*Ocimum tenuiflorum* L.).

A phospholipid-complexed methanol extract of *O. sanctum*, standardised against ursolic acid, was assessed against H1N1 in embryonated eggs,[31] and *in-vitro* studies against pandemic swine-flu H1N1 reported reduced viral replication and protein synthesis.[32] The exact mechanism of the individual Tulsi constituents against influenza requires further validation (Table 2, Fig 5).[43]

Table 2: Bioactive Constituents of Tulsi and Relevance to Influenza

Sr. No.	Constituent	Chemical class	Reported activity	Relevance to influenza
1	Eugenol	Phenylpropanoid (volatile oil constituent)	Antioxidant, anti-inflammatory, antimicrobial	Important bioactive constituent that contributes to the pharmacological activity of Tulsi.[44]
2	Ursolic acid	Triterpenoid	Antioxidant, anti-inflammatory, antimicrobial	Used as a marker (standardising) constituent in an H1N1 investigation of *O. sanctum*.[31]
3	Rosmarinic acid	Phenolic acid	Antioxidant and protective effects	May contribute to antioxidant and anti-inflammatory effects during viral infection.[43]

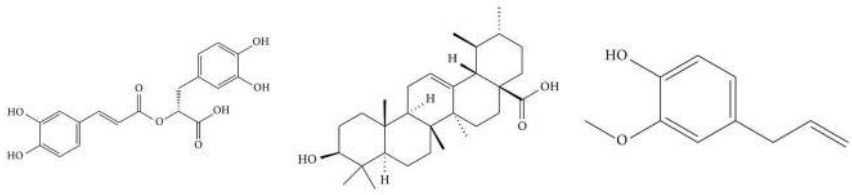

Figure 5: Chemical Structures of Selected Tulsi Constituents: (A) Rosmarinic Acid, (B) Ursolic Acid and (C) Eugenol.

2.2 Neem (*Azadirachta indica* A. Juss.)

Neem (family Meliaceae; also known as Indian lilac or margosa) yields leaves, bark, seeds, fruits, flowers and roots, of which the leaves are particularly important for antiviral studies (Fig 6). Its major phytochemical classes are limonoids, tetranortriterpenoids, triterpenoids, flavonoids, phenolic compounds, tannins, saponins and glycosides. The principal constituents include azadirachtin, nimbin, nimbidin, nimbolide, salannin, gedunin, quercetin, rutin, hyperoside and nimbaflavone. Neem is reported to be antiviral, antimicrobial, antioxidant, anti-inflammatory, immunomodulatory, antidiabetic, hepatoprotective and anticancer.



Figure 6: Neem (*Azadirachta indica* a. Juss.).

For influenza, the evidence for neem is computational. Neem-derived compounds showed potential interaction with the conserved viral proteins NP and NS1 in molecular-docking studies, with rutin and nimbaflavone identified as potential binders of NP and quercitrin among the compounds interacting with NS1 (Table 3, Fig 7).[33,34] These predictions have not yet been confirmed in cell-based or animal infection models.

Table 3: Neem Bioactive Constituents Relevant to Antiviral Activity

Sr. No.	Constituent	Chemical class	Reported activity	Relevance to influenza
1	Rutin	Flavonoid glycoside	Antioxidant, anti-inflammatory	Docking identified rutin as a potential binder of influenza viral proteins.[33]
2	Nimbaflavone	Flavonoid	Antioxidant	One of the neem compounds predicted to bind influenza nucleoprotein.[33]
3	Quercitrin	Flavonoid glycoside	Antioxidant, anti-inflammatory	Among the neem compounds with potential interaction with the influenza NS1 protein.[34]

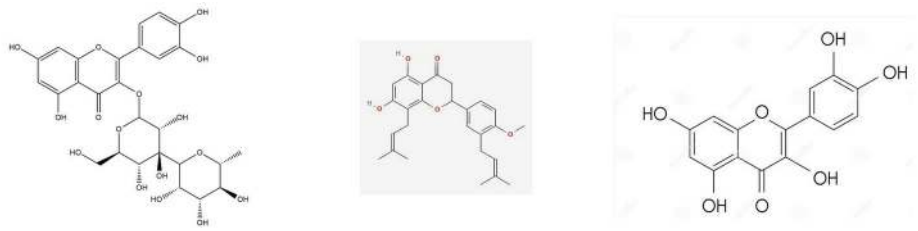

Figure 7: Chemical Structures of Neem Constituents: (A) Rutin, (B) Nimbaflavone and (C) Quercetin (the Aglycone of Quercitrin).

2.3 Turmeric (*Curcuma longa* L.)

Turmeric (family Zingiberaceae; haldi) is used mainly as the rhizome (Fig 8). Its phytochemical classes include curcuminoids, diarylheptanoids, sesquiterpenes, essential oils and phenolics, and the major constituents are curcumin, demethoxycurcumin, bisdemethoxycurcumin, ar-turmerone, α-turmerone, β-turmerone and zingiberene. Turmeric is antiviral, anti-inflammatory, antioxidant, antimicrobial, anticancer, hepatoprotective and immunomodulatory. Curcumin is the most studied constituent, and the strains studied are mainly influenza A/H1N1 and H9N2.



Figure 8: Turmeric (*Curcuma longa* L.) Rhizomes and Powder.

Curcumin reduced IAV infection and influenza-associated pneumonia, acting on viral infectivity and adsorption, on replication and on host signalling through oxidative stress, TLR2/4, p38/JNK MAPK and NF-κB pathways.[35] Curcuminoids from *C. longa* have also been reported to inhibit influenza A neuraminidases,[29] and curcumin derivatives reduced H1N1 neuraminidase activity and viral infectivity in MDCK cells.[36] In addition, bisabolane-type sesquiterpenoids isolated from turmeric inhibited H1N1 replication and inflammatory signalling in vitro (Table 4, Fig 9).[37]

Table 4: Bioactive Constituents of Turmeric Relevant to Influenza

Sr. No.	Constituent	Chemical class	Reported activity	Relevance to influenza
1	Curcumin	Curcuminoid (polyphenol)	Antiviral, antioxidant, anti-inflammatory	Strongest evidence: inhibits IAV infectivity, adsorption and replication and reduces influenza-associated inflammation.[35]
2	Demethoxycurcumin	Curcuminoid	Antioxidant, anti-inflammatory	Contributes to the activity of turmeric; curcuminoid derivatives have shown anti-IAV potential.[36]
3	Bisabolane-type compounds	Sesquiterpenoids	Antiviral, anti-inflammatory	Several isolated compounds inhibited H1N1 replication and inflammatory signalling in vitro.[37]

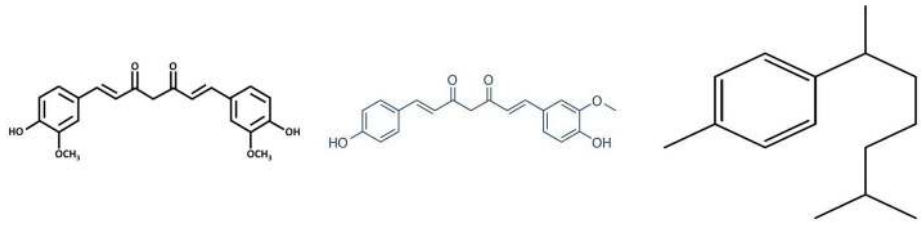

Figure 9: Chemical Structures of Turmeric Constituents: (A) Curcumin, (B) Demethoxycurcumin and (C) the Bisabolane Skeleton, the Core Structure of the Bisabolane-Type Sesquiterpenoids.

2.4 Garlic (*Allium sativum* L.)

Garlic (family Amaryllidaceae; lahsun) is used mainly as the bulb (cloves) (Fig 10). Its phytochemical classes include organosulfur compounds, flavonoids, phenolics, saponins and polysaccharides, and the major constituents are alliin, allicin, ajoene, diallyl disulfide (DADS), diallyl trisulfide (DATS), S-allylcysteine (SAC), S-allylmercaptocysteine (SAMC), vinyldithiins and quercetin. Allicin is the most important constituent; it is formed when crushing or damaging garlic brings alliin into contact with the enzyme alliinase.[45] Garlic is antiviral, antibacterial, antifungal, antioxidant, anti-inflammatory, immunomodulatory and cardioprotective.



Figure 10: Garlic (*Allium sativum* L.).

Garlic extracts inhibited influenza A infection and replication in experimental cell models, with studies mainly on influenza A/H1N1.[38] Allicin is the major candidate for the antiviral activity and has been proposed to interfere with thiol-containing viral enzymes and possibly with viral RNA polymerase, while quercetin and DATS may add independent antiviral and immunomodulating effects (Table 5, Fig 11).[46]

Table 5: Bioactive Constituents of Garlic Relevant to Influenza

Sr. No.	Constituent	Chemical class	Reported activity	Relevance to influenza
1	Allicin	Thiosulfinate (organosulfur compound) from freshly crushed bulb	Antimicrobial, antioxidant, anti-inflammatory, antiviral potential	Major candidate for the antiviral activity; proposed to interfere with thiol-containing viral enzymes and possibly viral RNA polymerase.[45]
2	Quercetin	Flavonoid	Antioxidant, anti-inflammatory	Has independent antiviral activity against some respiratory viruses and contributes to the phytochemical profile of garlic.[38]
3	Diallyl trisulfide (DATS)	Organosulfur compound (garlic oil)	Antimicrobial, antioxidant, immunomodulatory	Potential antiviral and immune-modulating activity.[46]

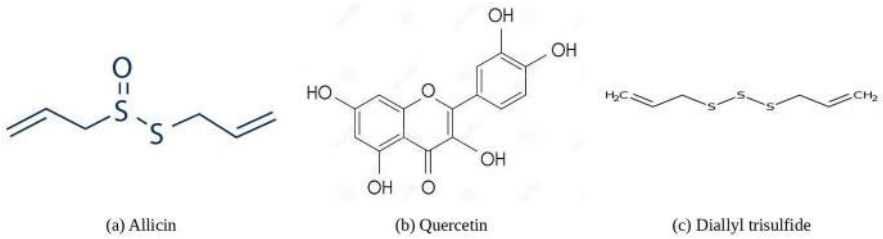

Figure 11: Chemical Structures of Garlic Constituents: (A) Allicin, (B) Quercetin and (C) Diallyl Trisulfide.

2.5 Ginger (*Zingiber officinale* Roscoe)

Ginger (family Zingiberaceae; adrak) is used as the rhizome (Fig 12). Its phytochemical classes are gingerols, shogaols, paradols, diarylheptanoids, phenolics and volatile terpenes, and the major constituents are 6-, 8- and 10-gingerol, 6-, 8- and 10-shogaol, zingerone and zingiberene, of which 6-gingerol is the most characteristic. Ginger is antioxidant, anti-inflammatory, antimicrobial, analgesic, gastroprotective and immunomodulatory.



Figure 12: Ginger (*Zingiber officinale* Roscoe) Plant and Rhizome.

Ginger extract inhibited the growth of influenza A/Aichi/2/68 virus in an MDCK-cell model, and the effect was reported to be macrophage-mediated.[39] More recently, [6]-gingerol reduced H1N1 (PR8) infection in vitro, inhibited viral neuraminidase activity, lowered the viral load in the lungs of infected mice and enhanced hemagglutinin-specific T-cell responses.[40] For 6-shogaol, antiviral activity has been shown mainly against other viruses; a recent study of the related 10-shogaol reported broad antiviral and anti-inflammatory activity against dengue and Zika viruses, and influenza-specific evidence for shogaols remains limited (Table 6, Fig 13).[47]

Table 6: Bioactive Constituents of Ginger Relevant to Antiviral Activity

Sr. No.	Constituent	Chemical class	Reported activity	Relevance to influenza
1	6-Gingerol	Phenolic ketone (fresh rhizome)	Antioxidant, anti-inflammatory, antimicrobial, antiviral potential	Inhibited H1N1 infection and NA activity in vitro and reduced lung viral load in mice; evidence comes mainly from a single H1N1 (PR8) study.[40]
2	6-Shogaol	Phenolic ketone (dried or heated ginger)	Antioxidant, anti-inflammatory	Related shogaols show antiviral activity against other viruses; influenza-specific evidence is limited.[47]

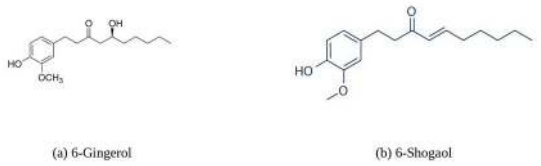

Figure 13: Chemical Structures of Ginger Constituents: (A) 6-Gingerol and (B) 6-Shogaol.

2.6 Licorice (*Glycyrrhiza glabra* L.)

Licorice (family Fabaceae; yashtimadhu or mulethi) is used as the root and rhizome (Fig 14). Its most important active constituent is glycyrrhizin (glycyrrhizic acid); others include glycyrrhetinic acid, liquiritin, isoliquiritin, liquiritigenin, glabridin, licoflavone B, other flavonoids and saponins. Licorice is antiviral, anti-inflammatory, antioxidant, immunomodulatory, hepatoprotective and expectorant, and activity has been reported against influenza A, influenza B and several other respiratory viruses.



Figure 14: Licorice (*Glycyrrhiza glabra* L.).

The evidence for glycyrrhizin against influenza is among the strongest of the six plants. In cell studies, glycyrrhizin reduced the number of influenza-infected human lung cells and produced approximately a 90% reduction in $CCID_{50}$ titre; the effect was associated with an early stage of infection, particularly reduced viral uptake into cells.[41] In H5N1-infected A549 human lung epithelial cells, glycyrrhizin:[42]

- reduced H5N1-induced inflammatory responses and decreased CXCL10, IL-6, CCL2 and CCL5 expression;
- reduced reactive oxygen species and inhibited activation of NF-κB, JNK and p38; and
- showed inhibitory effects on H5N1 replication at higher concentrations (Fig 15).

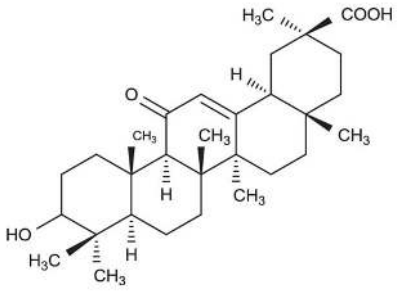

Figure 15: Chemical Structure of Glycyrrhetinic Acid, the Aglycone of Glycyrrhizin.

3. Mechanisms of Action of Medicinal Plants against Influenza Virus

The reported antiviral mechanisms can be grouped into seven categories: (1) inhibition of viral attachment through HA; (2) inhibition of viral entry and membrane fusion; (3) inhibition of M2 ion-channel activity and viral uncoating; (4) inhibition of viral RNA replication and transcription; (5) inhibition of neuraminidase; (6) inhibition of viral replication through host signalling pathways; and (7) modulation of the inflammatory response. The plants for which each mechanism has been reported are listed in Table 7.

Table 7: Mechanisms of Action of the Reviewed Plants Against Influenza Virus

Sr. No.	Mechanism	General basis	Plants and evidence
1	Inhibition of viral attachment	Compounds may interfere with binding of HA to sialic acid receptors, reducing viral adsorption.	*Curcuma longa*: curcumin inhibited IAV adsorption in experimental studies.[35]
2	Inhibition of viral entry and membrane fusion	Phytochemicals may affect viral-envelope integrity or host-cell endocytosis, reducing viral entry.	*C. longa*.[35] *Glycyrrhiza glabra*: glycyrrhizin reduced virus uptake by reducing endocytotic activity.[41]

Sr. No.	Mechanism	General basis	Plants and evidence
3	Inhibition of M2 ion-channel activity and uncoating	Blocking the M2 proton channel prevents acidification of the virion and release of vRNPs.	Direct M2-channel inhibition has not been firmly established for the six plants in the literature reviewed here.
4	Inhibition of viral RNA replication and transcription	Phytochemicals may reduce viral replication or protein production, or interfere with cellular processes required for multiplication.	*Ocimum sanctum*: reduced viral replication and protein synthesis.[32] *C. longa*: inhibited virus proliferation.[35] *Azadirachta indica*: interaction with NP predicted computationally.[33]
5	Inhibition of neuraminidase	Inhibition of NA prevents cleavage of sialic acid, reducing release and spread of new virions.	*Zingiber officinale*: [6]-gingerol inhibited NA activity.[40] *C. longa*: curcuminoids and derivatives reduced NA activity.[29,36]
6	Modulation of host signalling pathways	Compounds may influence oxidative stress and signalling through NF-κB and MAPK, altering viral replication and host responses.	*C. longa*: modulated Nrf2-associated responses and TLR/MAPK/NF-κB pathways.[35] *G. glabra*: glycyrrhizin modulated ROS-associated NF-κB, JNK and p38 signalling.[42]
7	Modulation of the inflammatory response	Compounds may regulate pro-inflammatory cytokines and chemokines, limiting excessive inflammation and tissue injury.	*C. longa*: reduced inflammatory responses in experimental models.[35] *G. glabra*: reduced pro-inflammatory gene expression in H5N1-infected cells.[42] *Z. officinale*: macrophage-mediated antiviral effects.[39]

4. Summary of Key Studies

The principal studies and reviews that support the preceding sections are summarised in Table 8. Of particular note, a systematic review and meta-analysis of randomised controlled trials of herbal medicines for influenza, including 25 trials and 3,044 patients, found that herbal medicine could reduce the duration of fever compared with placebo, and some trials evaluated herbal medicines combined with oseltamivir. The authors stressed the need for better-designed trials, and this evidence does not establish the efficacy of any individual plant discussed here.[48]

Table 8: Summary of Key Studies on Plant-Derived Anti-Influenza Activity

Sr. No.	Authors (year)	Subject	Principal findings
1	Arumugam et al. (2025)	Review of plant extracts against IAV	Extracts of *Silybum marianum*, *Scutellaria baicalensis*, *Angelica dahurica*, *Peganum harmala*, *Sambucus nigra*, *Echinacea purpurea*, *Panax ginseng* and *Camellia sinensis* act by inhibiting viral RNA synthesis, attachment and entry, by direct virucidal activity and by modulating immunity.[30]
2	Li et al. (2024)	Phytochemicals against H1N1	Flavonoids, alkaloids, terpenoids, phenolics and polysaccharides interfere with viral entry, replication and other stages; toxicity and druggability were discussed.[9]
3	Bhattarai et al. (2024)	Phytochemistry of *Ocimum tenuiflorum*	Rosmarinic acid, oleanolic acid, ursolic acid, luteolin and limonene with antioxidant, antimicrobial and anti-inflammatory properties.[43]
4	Dutta et al. (2023)	[6]-Gingerol against H1N1	Reduced infection, inhibited NA, lowered lung viral load in mice and enhanced hemagglutinin-specific T-cell responses.[40]
5	Mehrbod et al. (2021)	Iranian medicinal plant extracts	Certain extracts inhibited influenza in cell culture; activity depended on phytochemical composition and extraction method.[26]
6	Ti et al. (2021)	Bisabolane sesquiterpenoids from turmeric	Inhibited H1N1 in MDCK and A549 cells, reduced TNF-α, IL-6, IL-8 and IP-10, involving NF-κB/MAPK and RIG-I/STAT1/2 signalling.[37]
7	Brindha Devi and Sarala (2021)	Review of about 50 medicinal herbs	Mechanisms include inhibition of attachment, entry, replication and release; clinical investigation is still required.[27]

Sr. No.	Authors (year)	Subject	Principal findings
8	Lai et al. (2020)	Curcumin derivatives against H1N1 NA	Modelling, QSAR and cell studies showed reduced NA activity and viral infectivity in MDCK cells.[36]
9	Denaro et al. (2020)	Review of antiviral plants	Flavonoids, phenolics and terpenoids are important in antiviral discovery; interactions with conventional antivirals possible.[24]
10	Choi et al. (2020)	Meta-analysis of 25 RCTs (3,044 patients)	Herbal medicine reduced the duration of fever compared with placebo; better-designed trials needed.[48]
11	Dai et al. (2018)	Curcumin against IAV and pneumonia	Reduced viral infection and inflammation via oxidative stress, TLR2/4, p38/JNK MAPK and NF-κB pathways.[35]

5. Research Gap

Despite promising results, the use of medicinal plants against influenza still has several limitations. Most studies have used in-vitro models, animal experiments, molecular docking or non-standardised plant extracts, while well-designed human studies remain comparatively limited.[24,30] Differences in plant species, geographical origin, plant part, extraction method and phytochemical composition also make results difficult to reproduce and compare.[30] Although several phytochemicals can interfere with different stages of the influenza life cycle, their exact molecular targets and mechanisms are not completely understood.[27,48] In addition, limited information on bioavailability, pharmacokinetics, effective dose, toxicity and drug interactions remains a major barrier to clinical development.[30,48] Further standardised and clinical studies are therefore required to establish safe and effective plant-based anti-influenza therapies.

6. Future Perspectives

Medicinal plants represent a promising source of new antiviral compounds against IAV, but further research and clinical evaluation are required to convert their experimental properties into effective therapeutic agents. The main directions are as follows.

1. **Identification of active phytochemicals.** Isolation, purification and identification of active compounds from Tulsi, Neem, Turmeric, Garlic, Ginger and Licorice, and further investigation of curcumin, glycyrrhizin, allicin, gingerol and ursolic acid.[24,30]
2. **Advanced molecular docking studies.** Docking and molecular dynamics simulations can identify interactions between plant compounds and viral proteins such as HA, NA, NP and NS1, and help to find new lead molecules.[33,34]
3. **Standardised herbal extracts.** Appropriate extraction methods, phytochemical markers and quality-control parameters are needed to ensure consistent quality, safety and reproducible results.[24,30]
4. **Pharmacokinetic and bioavailability studies.** Many plant compounds are poorly absorbed, have low bioavailability and are metabolised rapidly, so their absorption, distribution, metabolism and excretion (ADME) properties should be studied.[24,30]
5. **Safety and toxicity evaluation.** Acute and chronic toxicity, adverse effects, drug interactions and safe therapeutic doses should be established before human application.[24]
6. **Novel drug delivery systems.** Nanoparticles, liposomes, nanoemulsions and phytosomal formulations may improve the solubility, stability and bioavailability of plant-derived antivirals.
7. **Clinical trials and human studies.** Well-designed trials with adequate sample sizes, standardised formulations and suitable controls are necessary to establish safety and efficacy in humans.[48,49]
8. **Combination therapy.** Combining plant compounds with established antivirals should be studied for synergy, toxicity and drug interactions, rather than assuming that combinations improve outcomes.
9. **Multiple viral targets.** Compounds acting on several stages of the life cycle (attachment, fusion, RNA replication and NA-mediated release) may open new directions for antiviral discovery.[27,28]

10. **Integration of traditional medicine with modern research.** Combining traditional knowledge with pharmacological, molecular and clinical research may help to develop affordable, safe and effective antiviral candidates from natural sources.[1,24,30]

CONCLUSION

Influenza remains an important public health problem because of its rapid genetic variation, the emergence of antiviral resistance and the limitations of the available preventive and therapeutic approaches. This has increased interest in medicinal plants as natural sources of antiviral compounds. The literature reviewed shows that Tulsi, Neem, Turmeric, Garlic, Ginger and Licorice contain diverse phytochemicals, including flavonoids, phenolics, terpenoids, alkaloids, tannins and saponins, which may interfere with viral attachment and entry, membrane fusion, uncoating, RNA replication, assembly and neuraminidase-mediated release. Some compounds may also help to control the excessive inflammatory response associated with influenza.

The current evidence must nevertheless be interpreted carefully. Much of it is based on laboratory experiments, animal studies, crude extracts and computational investigations, and the activity varies with plant species, plant part, extraction procedure and phytochemical composition. Data on bioavailability, pharmacokinetics, toxicity, standardised dose and clinical efficacy are still lacking for many compounds. Medicinal plants therefore cannot yet be regarded as direct replacements for established antiviral medicines. They should instead be viewed as a promising source for the identification and development of new antiviral candidates.

Future work should focus on identifying the most active phytoconstituents and their precise molecular targets, developing standardised extracts, evaluating safety and pharmacokinetics, and conducting well-designed clinical studies. Combining traditional knowledge with modern pharmacological, molecular and clinical research could open a valuable avenue for the discovery of safer, cheaper and effective plant-derived approaches against influenza.

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Conflict of Interest

The authors declare that there is no conflict of interest regarding the publication of this manuscript.

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