IPS Journal of Applied Microbiology and Biotechnology
ISSN: 3115-5685
IPS J Appl Microbiol Biotech, 4(4): 203-209 (2025)
DOI: https://doi.org/10.54117/ijamb.v4i4.91

Human Echovirus Infection: Virology, Pathogenesis, Clinical Manifestations, and Management

Iheukwumere, I. H.[1], Iheukwumere, C. M.[2], Unaeze, B. C.[3], Ike, V. E.[4], Nnadozie, H. C.[1] and Onyema, S. O.[1]

[1]Department of Microbiology, Faculty of Natural Sciences, Chukwuemeka Odumegwu Ojukwu University, Anambra State, Nigeria.
[2]Department of Applied Microbiology & Brewing, Faculty of Biosciences, Nnamdi Azikiwe University, Awka, Anambra State, Nigeria.
[3]Department of Medical Sciences, Nnamdi Azikiwe University, Awka, Anambra State, Nigeria.
[4]Department of Microbiology, University of Agriculture and Environmental Sciences, Umuagwo, Imo State, Nigeria.
*Corresponding author email: ik.iheukwumere@coou.edu.ng / ikpower2007@yahoo.com

Abstract

Human echoviruses (Enteric Cytopathic Human Orphan viruses), members of the *Enterovirus* B species within the *Picornaviridae* family, are significant human pathogens with a global distribution. Initially isolated in the 1950s as "orphan" viruses not linked to disease, they are now recognized as a leading cause of a wide spectrum of illnesses, particularly in neonates, children, and immunocompromised individuals. Echoviruses are non-enveloped, positive-sense single-stranded RNA viruses characterized by a simple yet efficient structure that facilitates robust replication in host cells. Transmission occurs primarily via the fecal-oral route, with additional pathways including respiratory droplets and vertical transmission. The pathogenesis involves initial replication in the oropharyngeal and intestinal mucosa, followed by viremic dissemination to secondary target organs such as the central nervous system (CNS), heart, and liver. Clinical manifestations range from mild febrile illness and rashes to severe, life-threatening conditions including aseptic meningitis, neonatal sepsis-like syndrome, encephalitis, and myocarditis. Diagnosis relies on clinical suspicion confirmed by molecular methods like RT-PCR, while treatment remains largely supportive due to the absence of specific antiviral therapies. Prevention emphasizes stringent hygiene practices and infection control, especially in high-risk settings. This review provides a detailed synthesis of the history, virology, transmission dynamics, molecular pathogenesis, clinical disease spectrum, diagnostic approaches, and current management strategies for human echovirus infections.

Keywords: Echovirus, Enterovirus, Picornaviridae, Aseptic Meningitis, Neonatal Sepsis, Viral Pathogenesis, RT-PCR, Pleconaril, Outbreak Management.

Article History
Received: 09 Sept 2025
Accepted: 04 Oct 2025
Published: 10 Oct 2025

How to cite this paper: Iheukwumere, I. H., Iheukwumere, C. M., Unaeze, B. C., Ike, V. E., Nnadozie, H. C., & Onyema, S. O. (2025). Human Echovirus Infection: Virology, Pathogenesis, Clinical Manifestations, and Management. *IPS Journal of Applied Microbiology and Biotechnology*, *4*(4), 203–209. https://doi.org/10.54117/ijamb.v4i4.91

1. Introduction

The first isolation of echoviruses occurred in the early 1950s from the faeces of asymptomatic children, shortly after the development of cell culture techniques for viral research. The term "ECHO" is an acronym for *Enteric Cytopathic Human Orphan* virus. The designation "orphan" was originally used because these viruses were not initially associated with any known disease. Although echoviruses have since been linked to various clinical conditions, the original nomenclature is still widely used (Virology Online, 2009).

Echoviruses are highly infectious, with a primary target population of children. They are among the leading causes of acute febrile illnesses in infants and young children and represent one of the most common viral causes of aseptic meningitis. Infection in newborns can lead to severe systemic illness and is associated with a high mortality rate in this age group. The clinical symptoms of echovirus infections can resemble those caused by other viral or bacterial pathogens, making diagnosis challenging.

Echoviruses belong to the species *Enterovirus B* and the genus *Enterovirus* within the family *Picornaviridae*. As members of the enterovirus group, echoviruses are typically found in the gastrointestinal tract. Exposure to echoviruses may predispose individuals to secondary opportunistic infections and can complicate clinical outcomes (Committee on Echoviruses, 1955).

1.1 History of Human Echovirus

The history of human echoviruses (enteric cytopathic human orphan viruses) dates back to the early 1950s, during a period of rapid advancement in virology, particularly with the development of cell culture techniques that enabled the isolation and identification of new viral agents. The first echoviruses were isolated from the feces of asymptomatic children, initially regarded as "orphan" viruses because they were not immediately linked to any known disease (Sabin, 1955). The term "ECHO" was coined as an acronym for Enteric Cytopathic Human Orphan virus, reflecting their discovery in the human intestinal tract, their cytopathic effect in cell cultures, and the fact that they were initially unassociated with any clinical syndrome.

Echoviruses are members of the genus Enterovirus within the family Picornaviridae, and are closely related to polioviruses and coxsackieviruses. As research progressed, many echoviruses were later found to be associated with human disease, including aseptic meningitis, encephalitis, myocarditis, neonatal sepsis-like illness, rash illnesses, and gastrointestinal disorders (Pallansch and Roos, 2017). This led to the recognition that these viruses were far from benign and could cause significant morbidity, particularly in infants, young children, and immunocompromised individuals.

By the mid-20th century, advances in molecular virology, serotyping, and sequencing had enabled the classification of over 30 serotypes of echoviruses. Some previously classified echoviruses, such as echovirus 10 and 28, were reclassified as reoviruses and rhinoviruses, respectively, as understanding of viral taxonomy improved (Racaniello, 2001). The continuing surveillance and study of echoviruses through public health laboratories have remained essential, especially given their ability to cause seasonal outbreaks of viral meningitis and neonatal infections worldwide.

2. Viral Structure and Genome Organization

Echovirus (Fig. 1) measures approximately 24 to 30 nanometers (nm) in diameter and shares structural similarities with other RNA viruses. It possesses a naked icosahedral protein capsid, which comprises about 75% of the viral particle, enclosing a dense central core of single-stranded RNA (ssRNA). The RNA genome is approximately 7.5 kilobases (kb) in length and encodes an RNA-dependent RNA polymerase (replicase) along with a single polyprotein (Fig. 2). This polyprotein is subsequently cleaved to form both structural and non-structural proteins essential for viral replication and assembly within host cells. The structural proteins are crucial for host specificity and facilitate the delivery of the viral RNA genome into the host cell cytoplasm.

Upon entry, the virus disseminates to secondary sites throughout the body, including the central nervous system (CNS), liver, spleen, bone marrow, heart, and lungs. Following further replication in these tissues, clinical symptoms typically appear 4 to 6 days after infection. The most severe manifestations of echovirus infection are often delayed, emerging as neurological symptoms when the virus affects the central nervous system, which can result in life-threatening complications (Pallansch and Roos, 2017).

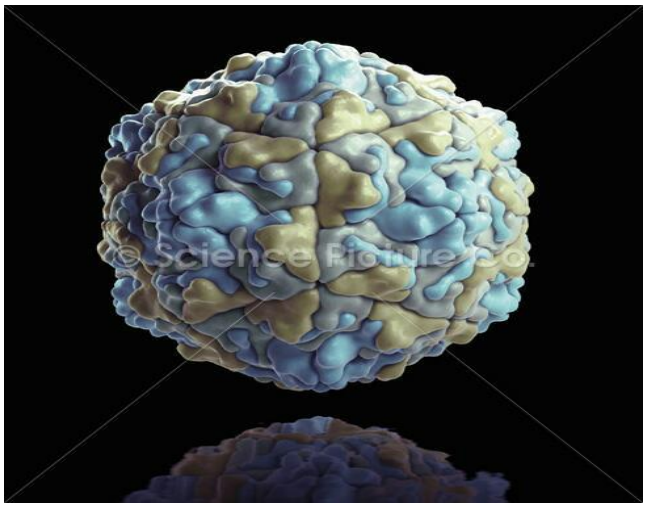

Figure 1: Viral structure of Human echovirus
Source: Pallansch and Roos, (2017).

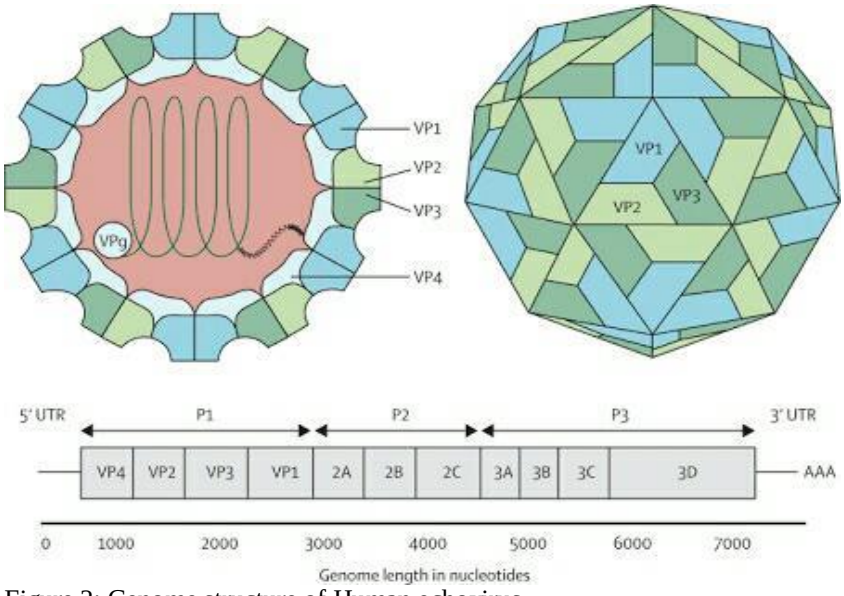

Figure 2: Genome structure of Human echovirus
Source: Pallansch and Roos, (2017).

2.1 Structural Proteins

Picornaviruses share common structural and compositional characteristics across all members of the family. These viruses are small, non-enveloped virions measuring approximately 27 to 30 nanometers in diameter and possess a single-stranded RNA (ssRNA) genome. Structurally, picornaviruses exhibit an icosahedral symmetry, which provides stability and facilitates efficient packaging of the viral genome.

The viral RNA core is enclosed within a protein shell (capsid) composed of four major structural proteins: VP1, VP2, VP3, and VP4. Additionally, a precursor protein, VP0, is present in trace amounts and is cleaved during virus assembly to form VP2 and VP4, completing the mature capsid structure.

These structural proteins, through their geometrical arrangement on the virion surface, form critical antigenic sites that are targets for neutralizing antibodies and also include receptor-binding domains necessary for host cell attachment and entry (Coffin, 1997).

2.2 Transmission Mechanisms of Human Echovirus

Human echoviruses, members of the *Enterovirus* genus within the *Picornaviridae* family, are primarily transmitted through the **fecal-oral route**, although other transmission pathways can also contribute to their spread. These viruses are highly contagious and capable of persisting in the environment, which facilitates their transmission, especially in settings with poor sanitation and hygiene.

1. **Fecal-Oral Transmission**
   The principal mode of echovirus transmission is via the fecal-oral route. Infected individuals shed the virus in their feces, and transmission occurs when contaminated hands, water, food, or surfaces are ingested by a susceptible host. This is particularly common in young children and in institutions such as daycare centers and schools (Pallansch and Roos, 2017).

2. **Respiratory Droplets**
   Although less common, echoviruses can also spread through **respiratory secretions**, including droplets from coughing or sneezing. This route may contribute to person-to-person spread, particularly in close-contact environments (Abzug, 2004).

3. **Vertical Transmission (Mother to Child)**
   Vertical transmission of echovirus can occur from **mother to infant during childbirth**, especially if the mother is viremic at the time of delivery. This mode of transmission is associated with severe systemic illness in neonates, including hepatitis, myocarditis, and meningoencephalitis (Modlin, 1995)

4. **Nosocomial Transmission**
   In healthcare settings, echovirus outbreaks have been linked to **nosocomial transmission**, often involving contaminated medical equipment or inadequate hand hygiene practices among healthcare workers (WHO, 2003).

5. **Waterborne and Fomite Transmission**
   Echoviruses can survive for extended periods in **untreated water** and on contaminated surfaces (fomites). Ingestion of or contact with contaminated water (e.g., recreational or drinking water) is a recognized pathway for outbreaks, particularly in areas with inadequate sanitation infrastructure (CDC, 2020).

2.3 Viral replication of human Echovirus

Human echovirus, a member of the *Enterovirus B* species in the *Picornaviridae* family, follows a typical replication cycle characteristic of non-enveloped, positive-sense single-stranded RNA (+ssRNA) viruses. The replication process occurs entirely in the cytoplasm of the host cell and involves several well-defined stages:

1. **Attachment and Entry**
   The echovirus attaches to specific receptors on the surface of the host cell, such as the decay-accelerating factor (DAF/CD55). Following receptor binding, the virus enters the cell via **receptor-mediated endocytosis** (Oberste et al., 2004; Iheukwumere *et al*., 2025a).

2. **Uncoating**
   Once internalized, the viral capsid undergoes conformational changes triggered by the endosomal environment, leading to the release of the viral RNA genome into the host cell cytoplasm (Iheukwumere *et al.*, 2025b).

3. **Translation of Viral RNA**
   The positive-sense RNA acts directly as messenger RNA (mRNA) and is translated by host ribosomes into a large **single polyprotein**. This polyprotein is then cleaved by viral proteases (2A and 3C) into structural proteins (VP1–VP4) and nonstructural proteins (e.g., RNA-dependent RNA polymerase 3D^pol, proteases, and replication factors) (Iheukwumere *et al*., 2025c).

4. **RNA Replication**
   The viral RNA-dependent RNA polymerase uses the genomic (+)RNA as a template to synthesize a complementary (–)RNA strand. This (–)RNA serves as a template for producing numerous new (+)RNA genomes, which are used for both translation and packaging into new virions (Iheukwumere *et al*., 2025d).

5. **Assembly**
   Structural proteins form the viral capsid, and new viral genomes are encapsidated into icosahedral particles within the cytoplasm. VP0, VP1, and VP3 assemble into pentamers, which then form the complete capsid. VP0 is subsequently cleaved into

VP2 and VP4 to mature the virion (Iheukwumere *et al*., 2025e).

6. **Release**
   Newly formed virions are released from the host cell mainly through **cell lysis**, contributing to tissue damage and inflammation (Iheukwumere *et al*., 2025f).

3. Viral Pathogenesis of Human Echovirus

The pathogenesis of human echoviruses is a complex process that unfolds through a series of defined steps, beginning with viral entry into the host and culminating in a spectrum of clinical manifestations. Echoviruses belong to the Picornaviridae family and are notable for causing a wide range of illnesses, particularly affecting children and immunocompromised individuals.

Entry into Host

The initial stage of infection involves the entry of the virus into the host, primarily through the fecal-oral route or, less commonly, via respiratory droplets. Following ingestion or inhalation, echoviruses target the epithelial cells lining the oropharynx and the intestinal tract. This localized infection serves as the gateway for subsequent viral replication (Iheukwumere *et al*., 2024a).

Primary Replication

Primary replication of the virus occurs within the mucosa-associated lymphoid tissues, including the tonsils and Peyer's patches. Here, the virus multiplies and establishes a localized infection, which eventually leads to a primary viremia. This early presence of virus in the bloodstream allows for dissemination to secondary target organs, marking the next phase of the pathogenesis (Iheukwumere *et al*., 2024b).

Secondary Viremia and Dissemination

During secondary viremia, echoviruses spread through the bloodstream to infect various organs. The central nervous system (CNS) is a significant target, where viral invasion can cause meningitis or encephalitis. The heart may also be affected, resulting in myocarditis or pericarditis. Additionally, organs such as the liver, spleen, and pancreas can be involved, contributing to systemic inflammatory responses that exacerbate the disease process (Iheukwumere *et al*., 2024c).

Cellular Tropism and Targeting

A critical aspect of echovirus pathogenesis is their cellular tropism—the preference for infecting specific cell types. Echoviruses exhibit affinity for neurons and glial cells within the CNS, cardiac myocytes, as well as endothelial and epithelial cells in various tissues. This tropism is mediated by the virus's ability to bind to specific cellular receptors, notably the decay-accelerating factor (DAF or CD55) and the neonatal Fc receptor (FcRn). Attachment to these receptors facilitates viral entry into host cells through receptor-mediated endocytosis (Iheukwumere *et al*., 2024d).

Viral Replication

Once inside the host cell, the viral RNA genome is released into the cytoplasm, where it undergoes direct translation into a single polyprotein. This polyprotein is subsequently cleaved into functional structural and non-structural proteins necessary for viral replication and assembly of new virions. The efficient replication cycle enables rapid viral proliferation and spread within the host (Iheukwumere *et al*., 2024e).

Host Immune Response

The host immune system responds to echovirus infection through both innate and adaptive mechanisms. Pattern recognition receptors (PRRs), such as Toll-like receptors (TLRs) and RIG-I-like receptors, detect viral components and trigger the production of interferons and other proinflammatory cytokines. Despite these defenses, echoviruses have evolved strategies to evade immune detection, including suppressing interferon signaling pathways and inducing apoptosis in immune cells. The adaptive immune response, particularly the generation of neutralizing antibodies, is crucial for clearing the virus and achieving recovery (Iheukwumere *et al*., 2024f).

Tissue Damage and Clinical Manifestations

Tissue damage during echovirus infection arises from multiple sources: the direct cytopathic effects of viral replication, the host's inflammatory response, and immune-mediated injury. These mechanisms are particularly damaging in the CNS and cardiac tissues, where they manifest as aseptic meningitis, myocarditis, neonatal sepsis-like syndromes, and encephalitis. The combination of viral-induced cell destruction and immune response leads to the clinical symptoms observed in echovirus infections.

3.1 Diseases Associated with Human Echovirus and Clinical Manifestations

Human echoviruses are associated with a broad spectrum of diseases, particularly affecting infants, children, and immunocompromised individuals. While many infections are asymptomatic or present with mild symptoms, echoviruses can also cause severe and potentially life-threatening illnesses, highlighting their clinical significance.

Aseptic (Viral) Meningitis

One of the most common conditions caused by echoviruses is aseptic (viral) meningitis, especially prevalent among children. This disease is characterized by symptoms such as headache, fever, neck stiffness, and sensitivity to light (photophobia). Echoviruses are recognized as leading viral agents responsible for this inflammation of the protective membranes surrounding the brain and spinal cord.

Neonatal Sepsis-like Illness

In newborns, echovirus infections can result in neonatal sepsis-like illness, which mimics bacterial sepsis but is caused by a viral pathogen. This condition is severe and carries a high mortality risk, particularly with echovirus serotypes 11 and 30, making it a critical concern in neonatal care.

Encephalitis
Encephalitis, or inflammation of the brain itself, is another serious complication of echovirus infection. Affected individuals may experience seizures, altered levels of consciousness, and neurological damage, which can have long-term consequences depending on the severity of the infection.

Myocarditis and Pericarditis
Echoviruses can also target the heart, causing myocarditis (inflammation of the heart muscle) and pericarditis (inflammation of the surrounding heart sac). Symptoms of cardiac involvement can include chest pain, irregular heart rhythms (arrhythmias), and in severe cases, sudden cardiac failure.

Respiratory Illness
Respiratory illnesses caused by echoviruses tend to be mild to moderate, especially in children, presenting with symptoms such as cough, sore throat, and nasal congestion. Although less severe than other complications, these symptoms contribute to the overall disease burden.

Hand, Foot, and Mouth Disease (HFMD)
While hand, foot, and mouth disease (HFMD) is more commonly linked to coxsackieviruses, certain echovirus strains have also been identified as causative agents. This disease manifests with characteristic vesicular rashes on the hands, feet, and oral mucosa.

Hepatitis
Echoviruses have been implicated in hepatitis-like illnesses, particularly in infants and neonates, with symptoms resembling liver inflammation.

Exanthems and Rashes
Echovirus infections may cause exanthems, presenting as nonspecific maculopapular rashes or urticaria, contributing to the diagnostic challenges.

Gastrointestinal Illness
Gastrointestinal illness is another clinical presentation, where patients may experience vomiting, diarrhea, and abdominal pain. These symptoms often resemble viral gastroenteritis and can lead to dehydration, especially in young children.

4. Diagnosis of Echovirus Infection

Echovirus infection diagnosis presents a clinical challenge due to the nonspecific nature of symptoms and the absence of a single definitive test. As a member of the enterovirus genus, echoviruses cause a range of diseases with overlapping clinical presentations, such as aseptic meningitis, neonatal sepsis, and febrile illnesses. Therefore, accurate diagnosis relies on a combination of clinical evaluation, laboratory tests, and exclusion of other potential causes (Khetsuriani et al., 2006).

Clinical Evaluation
The diagnostic process begins with a comprehensive medical history and physical examination. Physicians assess symptom onset, duration, and associated clinical features such as fever, rash, neurological signs, or gastrointestinal symptoms. This initial step is critical to suspect echovirus infection and decide on appropriate laboratory testing (Schmidt et al., 2019).

Laboratory Testing
Because echoviruses can infect multiple body systems, various specimens are collected depending on the clinical presentation. Key diagnostic methods include:

- **Culture-Based Methods:** Viral isolation remains a traditional and reliable diagnostic tool. Echoviruses can be cultured from stool, throat swabs, cerebrospinal fluid (CSF), blood, or pericardial fluid using susceptible cell lines. Cultures allow viral identification but may take several days to weeks, limiting their utility in acute clinical management (Stapleton et al., 1999; Iheukwumere *et al*., 2025g).
- **Molecular Diagnostics:** Reverse Transcriptase Polymerase Chain Reaction (RT-PCR) is currently the gold standard for rapid and sensitive detection of echoviral RNA. This method can be performed on CSF, serum, stool, or throat swab samples and significantly reduces diagnostic turnaround time compared to culture methods. RT-PCR assays have improved the ability to confirm echovirus infection during outbreaks or severe cases (Nix et al., 2006; Iheukwumere *et al*., 2025h).
- **Serological Tests:** Serology is used to detect antibodies produced in response to echovirus exposure. While helpful for epidemiological studies, serological assays have limited clinical utility due to cross-reactivity with other enteroviruses and the delay in antibody production after infection (Rotbart, 1995; Iheukwumere *et al*., 2025i).

Specialized Diagnostic Procedures
In cases where echovirus infection affects specific organs, targeted diagnostic tests are performed:

- **Cerebrospinal Fluid Analysis:** For suspected aseptic meningitis, lumbar puncture and CSF analysis are critical. CSF cultures and RT-PCR help confirm viral involvement, differentiating it from bacterial meningitis (Nigro et al., 2018).
- **Cardiac Evaluation:** Echovirus-induced myocarditis or pericarditis necessitates echocardiography to assess cardiac function and electrocardiography (ECG) to detect arrhythmias or conduction abnormalities. These tests help identify complications early and guide treatment (Bowles et al., 2003).
- **Radiographic Imaging:** Chest X-rays and other imaging studies may be employed when respiratory symptoms or systemic involvement is suspected. Radiographs assist in ruling out differential diagnoses and assessing organ damage (Miller et al., 2010).

Differential Diagnosis and Confirmatory Testing

Due to overlapping clinical features with other viral and bacterial infections, healthcare providers often order additional tests to exclude alternative causes. Differential diagnosis includes infections caused by other enteroviruses, herpesviruses, bacterial meningitis, and systemic sepsis. Confirmatory diagnosis of echovirus infection hinges on positive viral isolation or molecular detection coupled with clinical correlation (Weinstein et al., 2012; Iheukwumere *et al*., 2025j).

4.1 Treatment of Human Echovirus

No specific treatment for echovirus infection is currently available. Care is directed at relieving symptoms. The antiviral drug pleconaril interferes with the binding of echovirus particles to the cell membrane (Walters, 2007)

4.2 Prevention of Human Echovirus

Currently, there are no definitive preventative methods for echovirus infection. However, the following measures may be helpful. Frequent hand washing may be useful, especially after coming in contact with people who I'll clean and disinfect surfaces (floors, walls etc).

Especially in places like child care centers, hospitals and other institutional settings is important for effective prevention of any infection (World Health Organization, 2015).

References

Abzug, M. J. (2004). The enteroviruses: Problems in need of treatments. *The Journal of Infection, 49*(2), 104–112.

Bowles, N. E., Ni, J., Kearney, D. L., Pauschinger, M., Schultheiss, H. P., McCarthy, R. E., Hare, J. M., and Towbin, J. A. (2003). Detection of viral genomes in myocardial tissues of patients with myocarditis and dilated cardiomyopathy by polymerase chain reaction. *Journal of the American College of Cardiology, 42*(3), 466–472.

Centers for Disease Control and Prevention. (2020). Non-polio enterovirus infections. https://www.cdc.gov/non-polio-enterovirus/about/index.html

Coffin, J. M., and Hughes, S. H. (1997). Picornaviruses and accessory proteins. In H. E. Editors (Ed.), *Cold Spring Harbor* (pp. 30–38). United States of America.

Committee of Echo Virus Infection. (1995). Enteric cytopathogenic human orphan virus sciences. *Journal of Biotechnology, 122*, 118–119.

Iheukwumere, C.M., Iheukwumere, I.H. Obianom, A.O., Unaeze, B.C., Ejike, C.E., Igiri, V.C. and Okereke, F.O. (2024b). Supersizing the neutralizing activities of *Curcuma longa* and *Baphia nitida* extracts against Newcastle disease virus using Vitamin C. *Tropical Journal of Applied Natural Sciences* 2(1): 1 – 15.

Iheukwumere, C.M., Iheukwumere, I.H. Obianom, A.O., Unaeze, B.C., Ejike, C.E., Igiri, V.C. and Okereke, F.O. (2024c). Boosting the antiviral activity *Baphia nitida* leaves extract in broiler chicks using chicks Vitamin C. *Tropical Journal of Applied Natural Sciences* 2(1): 1 – 10.

Iheukwumere, C.M., Iheukwumere, I.H. Obianom, A.O., Unaeze, B.C., Ejike, C.E., Igiri, V.C. and Okereke, F.O. (2024e). Supersizing the neutralizing activities of *Curcuma longa* and *Baphia nitida* extracts against Newcastle disease virus using Vitamin C. *Tropical Journal of Applied Natural Sciences* 2(1): 1 – 15.

Iheukwumere, C.M., Iheukwumere, I.H. Obianom, A.O., Unaeze, B.C., Ejike, C.E., Igiri, V.C. and Okereke, F.O. (2024f). Boosting the antiviral activity *Baphia nitida* leaves extract in broiler chicks using chicks Vitamin C. *Tropical Journal of Applied Natural Sciences* 2(1): 1 – 10.

Iheukwumere, I.H., Iheukwumere, C.M., Obianom, A.O., Nnadozie, C.H., Onwusoanya, U.F., Oduoye, O.T., Ike, V.E., Obiefuna, O.H., Igboanugo, E.U., Ejike, C.E., Udeagbara, O.E., Ochibulu, S.C., Onyemekara, N.N., Iheunatuoha, U.A., Nwakoby, N.E. and Ilechukwu, C.C. (2025a). Enhancement of the antiviral potency of *Curcuma longa* and *Azadiratcha indica* using Vitamin C in embryonated chicken eggs. *IPS Journal of Phytochemistry and Chemistry and Medicinal Plant Research* 1(1): 9 – 14.

Iheukwumere, I.H., Iheukwumere, C.M., Obianom, A.O., Nnadozie, C.H., Onwusoanya, U.F., Oduoye, O.T., Ike, V.E., Obiefuna, O.H., Igboanugo, E.U., Ejike, C.E., Udeagbara, O.E., Ochibulu, S.C., Onyemekara, N.N., Iheunatuoha, U.A., Nwakoby, N.E., Ilechukwu, C.C. and Destiny, C.C. (2025b). Mitigating Newcastle Disease Virus induced damage in chicken embryos using extracts of *Curcuma longa* and *Baphia nitida*. *IPS Journal of Basic and Clinical Medicine* 2(2): 58 – 63.

Iheukwumere, I.H., Iheukwumere, C.M., Obianom, A.O., Nnadozie, C.H., Onwusoanya, U.F., Oduoye, O.T., Ike, V.E., Obiefuna, O.H., Igboanugo, E.U., Ejike, C.E., Udeagbara, O.E., Ochibulu, S.C., Onyemekara, N.N., Ihenatuoha, U.A., Nwakoby, N.E., Ilechukwu, C.C. and Destiny, E.C (2025d). *IPS Journal of Toxicology* 3(2): 55 – 59.

Iheukwumere, I.H., Iheukwumere, C.M., Obianom, A.O., Nnadozie, C.H., Onwusoanya, U.F., Oduoye, O.T., Ike, V.E., Obiefuna, O.H., Igboanugo, E.U., Ejike, C.E., Udeagbara, O.E., Ochibulu, S.C., Onyemekara, N.N., Ihenatuoha, U.A., Nwakoby, N.E. and Ilechukwu, C.C. (2025e). Minifying the effects of Newcastle Disease Virus on Structural development of chicken embryo using *Curcuma longa* and *Baphia nitida* extracts. *IPS Journal of Basic and Clinical Medicine* 2(2): 58 – 63.

Iheukwumere, I.H., Iheukwumere, C.M., Obianom, A.O., Nnadozie, C.H., Onwusoanya, U.F., Oduoye, O.T., Ike, V.E., Obiefuna, O.H., Igboanugo, E.U., Ejike, C.E., Udeagbara, O.E., Ochibulu, S.C., Onyemekara, N.N., Iheunatuoha, U.A., Nwakoby, N.E. and Ilechukwu, C.C. (2025f). Enhancement of the antiviral potency of *Curcuma longa* and *Azadiratcha indica* using Vitamin C in embryonated chicken eggs. *IPS Journal of Phytochemistry and Chemistry and Medicinal Plant Research* 1(1): 9 – 14.

Iheukwumere, I.H., Iheukwumere, C.M., Obianom, A.O., Nnadozie, C.H., Onwusoanya, U.F., Oduoye, O.T., Ike, V.E., Obiefuna, O.H., Igboanugo, E.U., Ejike, C.E., Udeagbara, O.E., Ochibulu, S.C., Onyemekara, N.N., Iheunatuoha, U.A., Nwakoby, N.E., Ilechukwu, C.C. and Destiny, C.C. (2025g). Mitigating Newcastle Disease Virus induced damage in chicken embryos using extracts of *Curcuma longa* and *Baphia nitida*. *IPS Journal of Basic and Clinical Medicine* 2(2): 58 – 63.

Iheukwumere, I.H., Iheukwumere, C.M., Obianom, A.O., Nnadozie, C.H., Onwusoanya, U.F., Oduoye, O.T., Ike, V.E., Obiefuna, O.H., Igboanugo, E.U., Ejike, C.E., Udeagbara, O.E., Ochibulu, S.C., Onyemekara, N.N., Ihenatuoha, U.A., Nwakoby, N.E., Ilechukwu, C.C. and Destiny, E.C (2025i). *IPS Journal of Toxicology* 3(2): 55 – 59.

Iheukwumere, I.H., Iheukwumere, C.M., Obianom, A.O., Nnadozie, C.H., Onwusoanya, U.F., Oduoye, O.T., Ike, V.E., Obiefuna, O.H., Igboanugo, E.U., Ejike, C.E., Udeagbara, O.E., Ochibulu, S.C., Onyemekara, N.N., Ihenatuoha, U.A., Nwakoby, N.E. and Ilechukwu, C.C. (2025j). Minifying the effects of Newcastle Disease Virus on Structural development of chicken embryo using *Curcuma longa* and *Baphia nitida* extracts. *IPS Journal of Basic and Clinical Medicine* 2(2): 58 – 63.

Iheukwumere, I.H., Iheukwumere, C.M., Obianom, A.O., Unaeze, B.C., Ejike, C.E., Igiri, V.C. and Okereke, F.O. (2024a).

Augmenting the antiviral potency of *Baphia nitida* extract against Newcastle disease virus using Vitamin C using embryonated chicken eggs. *Tropical Journal of Applied Natural Scences.* 2(1): 1 – 12.

Iheukwumere, I.H., Iheukwumere, C.M., Obianom, A.O., Unaeze, B.C., Ejike, C.E., Igiri, V.C. and Okereke, F.O. (2024d). Augmenting the antiviral potency of *Baphia nitida* extract against Newcastle disease virus using Vitamin C using embryonated chicken eggs. *Tropical Journal of Applied Natural Scences.* 2(1): 1 – 12.

Iheukwumere, I.H., Mmaduagha, C.P., Nwike, M.I., Iheukwumere, C.M., Ike, V.E., Obianom, A.O., Ihenatuoha, U.A., Igboanugo, E.U., Okereke, F.O., Obiefuna, O.H., Nwakoby, N.E., Ilechukwu, C.C., Ochibulu, S.C. and Ejike, C.E. (2025c). Mitigating Newcastle Disease Virus Pathogenesis with Allicumin: A patenting approach. *IPS Journal of Advanced and Applied Biochemistry* 1(1): 11 – 18.

Iheukwumere, I.H., Mmaduagha, C.P., Nwike, M.I., Iheukwumere, C.M., Ike, V.E., Obianom, A.O., Ihenatuoha, U.A., Igboanugo, E.U., Okereke, F.O., Obiefuna, O.H., Nwakoby, N.E., Ilechukwu, C.C., Ochibulu, S.C. and Ejike, C.E. (2025h). Mitigating Newcastle Disease Virus Pathogenesis with Allicumin: A patenting approach. *IPS Journal of Advanced and Applied Biochemistry* 1(1): 11 – 18.

Khetsuriani, N., Lamonte-Fowlkes, A., Oberst, S., and Pallansch, M. A. (2006). Enterovirus surveillance — United States, 1970–2005. *MMWR Surveillance Summaries, 55*(8), 1–20.

Miller, J. M., Binnicker, M. J., Campbell, S., Carroll, K. C., Chapin, K. C., Gilligan, P. H., Cookson, B. T., Ferraro, M. J., Gandra, S., Hecht, D. W., Humphries, R. M., Karchmer, T., Patel, R., Procop, G. W., Reimer, L. G., Richter, S. S., Rothman, R. E., Salfinger, M., Tan, J., and Weinstein, M. P. (2018). A guide to utilization of the microbiology laboratory for diagnosis of infectious diseases: 2018 update. *Clinical Infectious Diseases, 67*(6), e1–e94.

Mirand, A., and Peigue-Lafeuille, H. (2015). Acute flaccid myelitis and enteroviruses: An ongoing story. *The Lancet, 385*(9973), 1601–1606.

Modlin, J. F. (1995). Perinatal echovirus and group B coxsackievirus infections. *Clinics in Perinatology, 22*(3), 761–776.

Nigro, G., Adriaenssens, N., and Giangaspero, A. (2018). Viral meningitis in children: A comprehensive review. *Clinical Microbiology Reviews, 31*(2), e00019-17.

Nix, W. A., Oberste, M. S., and Pallansch, M. A. (2006). Sensitive, seminested PCR amplification of VP1 sequences for direct identification of all enterovirus serotypes from clinical specimens. *Journal of Clinical Microbiology, 44*(8), 2698–2704.

Oberste, M. S., Maher, K., and Pallansch, M. A. (2004). Molecular phylogeny and proposed classification of the simian picornaviruses. *Journal of Virology, 78*(19), 4912–4922.

Pallansch, M. A., and Roos, R. P. (2017). Enteroviruses: Polioviruses, coxsackieviruses, echoviruses, and newer enteroviruses. In D. M. Knipe and P. M. Howley (Eds.), *Fields Virology* (6th ed.). Lippincott Williams and Wilkins.

Pallansch, M., and Roos, R. (2007). Enteroviruses: Polioviruses, echoviruses, and newer enteroviruses. In D. M. Knipe and P. M. Howley (Eds.), *Fields Virology* (5th ed., pp. 839–884). Lippincott Williams and Wilkins.

Racaniello, V. R. (2001). Picornaviridae: The viruses and their replication. In D. M. Knipe and P. M. Howley (Eds.), *Fields Virology* (4th ed.). Lippincott Williams and Wilkins.

Rotbart, H. A. (1995). Viral meningitis. *Seminars in Neurology, 15*(2), 141–146.

Ryan, K. J., and Ray, C. G. (Eds.). (2004). Viral structure. In *Sherris Medical Microbiology* (4th ed., pp. 537–539). McGraw-Hill.

Sabin, A. B. (1955). Pathogenesis of poliomyelitis; reappraisal in the light of new data. *Science, 121*(3133), 121–122.

Schmidt, N. J., Emmons, R. W., and Lennette, E. H. (2019). *Diagnostic procedures for viral, rickettsial, and chlamydial infections* (7th ed.). American Public Health Association.

Stapleton, J. T., Cleveland, D. M., and Courouce, A. M. (1999). *Clinical Virology* (3rd ed.). ASM Press.

Weinstein, M. P., Patel, J. B., and DesJarlais, S. L. (2012). *Clinical Microbiology Procedures Handbook* (3rd ed.). ASM Press.

World Health Organization. (2003). Enteroviruses: WHO guidelines for environmental surveillance of poliovirus circulation.

Yur Murphy, M., and Almond, J. W. (1996). Picornoviruses. In G. L. Mandell, J. E. Bennett, and R. Dolin (Eds.), *Principles and Practice of Infectious Diseases* (4th ed., pp. 527–528). University of Texas.

•*Thank you for publishing with us.*