Poliovirus

Monday, November 3, 2025 1:47 PM

Disease

Poliovirus (Enterovirus C) causes Poliomyelitis, a disease primarily characterized by flaccid paralysis due to the destruction of motor neurons in the spinal cord and brainstem.

Important Properties

Property	Description
Family	*Picornaviridae*
Genus	*Enterovirus*
Genome	Single-stranded, positive-sense RNA (nonsegmented)
Envelope	Absent (non-enveloped virus)
Capsid Symmetry	Icosahedral
Polymerase in Virion	None

Number of Serotypes	Three (Type 1, 2, and 3)
Host Range	Primates only (humans, apes, monkeys)
Transmission Route	Fecal-oral route 💩➡🧍

💡 *Note:* The viral capsid protein binds to a specific receptor found only on primate cells — this explains the virus's host restriction.

However, purified viral RNA (without capsid) is "infectious RNA" and can replicate in non-primate cells since it bypasses receptor entry requirements.

🧬 Serotypes and Immunity

There are three distinct serotypes of poliovirus:

- Type 1 – Most commonly associated with epidemics
- Type 2 – Rare, but causes paralytic disease occasionally
- Type 3 – Sporadic and less virulent

Because no cross-immunity exists between them,

protection requires antibodies against all three types 💧.

⚙️ Summary of Replicative Cycle

Replication takes place in the cytoplasm and is rapid (6–8 hours). The virus does not bud from the cell — it lyses the host cell on release.

Replication Flowchart:

Attachment to specific cell receptor (on primate cell membrane)

Entry → Uncoating of capsid proteins

Viral RNA acts directly as mRNA (positive-sense RNA)

Translation → Large polyprotein formed (Noncapsid viral protein 00)

Cleavage by viral protease into:

↳ Structural (capsid) proteins
↳ Nonstructural proteins, including RNA-dependent RNA polymerase

Synthesis of negative-sense RNA strand (as template)

Formation of new positive-sense RNAs
↳ Some used as mRNA
↳ Others packaged as genome RNA

Assembly of virions in cytoplasm

Cell lysis → Virus release 💥

🧠 *Exam Tip:* Unlike many RNA viruses, Poliovirus RNA itself is infectious, because it can act directly as mRNA upon entering the cell.

🌍 Transmission & Epidemiology

Feature	Details
Mode of Transmission	Fecal-oral route
Initial Site of Replication	Oropharynx and intestinal mucosa (esp. Peyer's patches)
Natural Host	Humans only
Seasonal Pattern	Peaks in summer and fall (pre-vaccine era)
Virus Shedding	Virus excreted in feces for several weeks post-infection

🦠 Sources of Infection: Contaminated water, food, and hands are common vectors for spread.

🌍 Global Epidemiology

- Before vaccination: Frequent summer epidemics in children.
- After vaccine introduction: *Wild-type poliovirus* eradicated from the Western Hemisphere.
- Current endemic regions (as of 2017):
  - Afghanistan
  - Pakistan
  - Nigeria

📊 Global Eradication Progress:

Year	Cases of Paralytic Polio
1988	~388,000
2005	< 2,000
2017	< 100

✅ *Smallpox* remains the only completely eradicated human infectious disease so far, but polio is close behind thanks to vaccination efforts.

Pathogenesis & Immunity

1 Initial Infection:

Virus enters via mouth → Replicates in oropharyngeal and intestinal lymphoid tissues.

2 Spread:

Local replication → Viremia (bloodstream spread) → Reaches central nervous system (CNS).

3 Target Site:

Within CNS, the virus preferentially infects motor neurons in:

- Anterior horn of spinal cord
- Brainstem motor nuclei

4 Pathologic Effects:

- Destruction of motor neurons → Flaccid paralysis
- Respiratory paralysis may occur in *bulbar poliomyelitis* (brainstem involvement).
- No muscle infection: Paralysis is neurogenic, not muscular.

5 Immunity:

- Secretory IgA in the intestine → Prevents reinfection.
- Serum IgG → Provides long-term protection.
- Lifelong, type-specific immunity after infection.

Pathogenesis Flowchart:

Ingestion of virus (contaminated food/water)

↓

Replication in oropharynx and intestine

↓

Spread to lymphoid tissue (tonsils, Peyer's patches)

↓

Primary viremia → Dissemination

↓

Invasion of CNS via blood or retrograde axonal transport

↓

Infection of anterior horn motor neurons

↓

Neuronal destruction → Flaccid paralysis ⚡

Exam Points

Feature	Key Point
Type of Virus	Positive-sense ssRNA (Picornavirus)
Envelope	Absent
Replication Site	Cytoplasm
Pathogenesis	Motor neuron destruction in anterior horn

Major Clinical Form	Paralytic poliomyelitis
Immunity	Lifelong, type-specific
Transmission	Fecal-oral
Prevention	Vaccination (discussed in next section)

📓 *Summary Insight:*

Poliovirus represents a classic enterovirus that travels from gut to neurons, teaching a key concept in virology:

👉 An enteric infection can manifest as a neurological disease.

🩺 Clinical Findings

Poliovirus infection produces a wide spectrum of responses depending on the extent of viral spread and host immunity.

Forms of Poliovirus Infection:

1 Inapparent / Asymptomatic infection

- Most common form. The virus replicates in the gut and oropharynx without causing noticeable illness.

2 Abortive Poliomyelitis (Minor Illness)

- *Most common clinically apparent form* (≈ 1%)

- A mild febrile illness lasting 1–2 days

- Symptoms:

  - Headache 😣
  - Sore throat
  - Nausea, vomiting
  - General malaise and fatigue

- *Recovery is spontaneous and complete.*

3 Non-paralytic Poliomyelitis (Aseptic Meningitis)

- Virus spreads to meninges but not to motor neurons

- Symptoms: Fever, headache, stiff neck (signs of meningeal irritation)

- *Usually resolves completely.*

4 Paralytic Poliomyelitis (Major Illness)

- *Most severe form (occurs in < 1% of infections)*

- Flaccid, asymmetric paralysis due to destruction of motor neurons in the anterior horn of spinal cord

- Clinical features:

  - Muscle weakness → flaccid paralysis
  - Painful muscle spasms
  - No sensory loss (only motor neurons affected)
  - If brainstem is involved → Bulbar Poliomyelitis → respiratory paralysis → may be fatal

- *Some muscle recovery may occur as surviving motor neurons reinnervate fibers.*

🌀 Meningoencephalitis → both meninges and brain parenchyma affected.

🌀 Meningomyeloencephalitis → also involves spinal cord.

Post-Polio Syndrome:

- Occurs decades after initial illness
- Gradual muscle weakness in previously affected muscles
- Mechanism unknown (likely neuronal exhaustion).

Incubation Period: 10–14 days ⏳

Carrier State: None permanent, but fecal shedding can continue for months.

🧪 Laboratory Diagnosis

Specimens: Throat swab, stool, or CSF.

Methods:

- Virus Isolation: Inoculation into cell culture → *cytopathic effect (CPE)* → identified by neutralization with type-specific antisera 🧫

- PCR Assay: Detects poliovirus RNA (highly specific).
- Serology: Rising antibody titer between acute & convalescent sera confirms infection.

💊 Treatment

❌ No antiviral therapy available.

☑ Management is supportive:

- Maintain respiration (may require ventilator)
- Physiotherapy for muscle rehabilitation 🧍

🛡 Prevention

Poliomyelitis is preventable by vaccination.

Two vaccines are used:

Attribute	Killed (Salk, IPV)	Live (Sabin, OPV)

Prevents disease	✅	✅
Interrupts fecal–oral transmission	❌	✅
Induces humoral IgG	✅	✅
Induces intestinal IgA	❌	✅
Secondary protection (spread to others)	❌	✅
Interferes with gut replication of wild virus	❌	✅
Reverts to virulence	❌	⚠ Rarely
Coinfection with other enteroviruses affects efficacy	❌	⚠ Yes
Safe in immunocompromised	✅	❌
Route	Injection 💉	Oral 💊
Refrigeration required	❌	✅
Duration of immunity	Shorter	Longer

🧬 Types & Use

- Inactivated Vaccine (IPV / eIPV):
    - Used currently in the U.S.
    - Higher seroconversion & antibody titers than old IPV
    - Induces partial mucosal IgA → some reduction in transmission
    - Safe (no reversion risk).

- Live Attenuated Vaccine (OPV):
    - Induces strong mucosal (IgA) immunity in GIT → blocks fecal-oral spread
    - Orally administered — easier for mass vaccination

⚠ Disadvantages of Live Attenuated Vaccine (OPV):

1. Rare reversion to virulence (esp. type 3 strain)
2. Dangerous in immunodeficient individuals
3. Interference from other enteroviruses
4. Requires refrigeration

📊 Flowchart — Comparison of Vaccine

Strategies

Poliovirus Exposure

If vaccinated with IPV → strong serum IgG → virus neutralized in blood → prevents CNS infection

If vaccinated with OPV → intestinal IgA formed → virus blocked at entry → prevents both infection & transmission

---

🌍 Vaccine-Derived Poliovirus (VDPV)

- Caused by mutation or recombination of Sabin strains with wild enteroviruses
- Leads to outbreaks in under-immunized populations
- Controlled by emergency OPV immunization campaigns

- WHO now recommends only inactivated trivalent vaccine (2016 onwards) for global use 🌐

⚠ Historical Note: SV40 Contamination

- Early polio vaccines (1950s–60s) grown in monkey kidney cells were contaminated with SV40 virus (a papovavirus).

- SV40 causes tumors in rodents but no proven human cancer link.

- Modern cell cultures are rigorously screened 🧫✅.

👶 Passive Immunization

- Immune Serum Globulin: For exposed, unimmunized individuals.

- Maternal IgG: Provides transient protection in newborns.

Quarantine

- Not effective due to pre-symptomatic fecal shedding and asymptomatic carriers.