Only **once** in human history have we witnessed the total eradication of dreaded disease, and that was smallpox more than two decades ago.

Now humanity stands on the brink of second triumph

-- a scourge that at one time killed or crippled half a million people a year, many of them children.
Poliomyelitis
Composition of Term

- *Polios* = grey
- *Myelos* = marrow, spinal cord
- *Myelitis* = inflammation of spinal cord
Wild Poliovirus 1999

- **Wild poliovirus transmission in 1999**
- **Probable wild poliovirus transmission**
- **Poliovirus importations in 1999**
Wild Poliovirus*

*Excludes viruses detected from environmental surveillance and vaccine derived polio viruses.

The boundaries and names shown and the designations used on this map do not imply the expression of any opinion whatsoever on the part of the World Health Organization concerning the legal status of any country, territory, city or area or of its authorities, or concerning the delimitation of its frontiers or boundaries. Dotted lines on maps represent approximate border lines for which there may not yet be full agreement.

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### Wild Poliovirus 2000 – 2006*

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| No. of countries     | 1118 |
| No. of endemic countries | 20 |

*Data in WHO HQ as of 4th Sep 2006

### Percentage of Global Wild Polio Virus Case

- **India**: 20.84%
- **Nigeria**: 66.82%
- **Namibia**: 1.79%
- **Somalia**: 2.68%
- **Angola**: 0.98%
- **Bangladesh**: 0.98%
- **Ethiopia**: 0.09%
- **Nepal**: 0.09%
- **Pakistan**: 1.25%
- **Somalia**: 2.68%
- **Niger**: 0.89%
- **Cambodia**: 0.09%
- **Myanmar**: 0.09%
- **DRC**: 0.72%
- **Indonesia**: 0.09%
- **Nepal**: 0.09%

*Countries highlighted in pale yellow are considered to have active transmission of an imported virus.

Countries highlighted in yellow are currently endemic.
Location of poliovirus, India, 2002 - 2006

2002: 1600 cases in 159 districts
2003: 225 cases in 87 districts
2004: 134 cases in 43 districts
2005: 66 cases in 35 districts
2006*: 297 cases in 47 districts

* data as on 15th September 2006
Districts infected with poliovirus

- 2002: 159 districts
- 2003: 87 districts
- 2004: 43 districts
- 2005: 35 districts
- 2006*: 47 districts

* data as on 15th September 2006
2001
(216 cases)

P1 = 159
P3 = 54
P1+P3 = 3

Wild virus of type P1

2002
(1242 cases)

P1 = 1139
P3 = 100
P1+P3 = 3

Wild virus of type P3

2003
(88 cases)

P1 = 72
P3 = 16

Wild virus of type P1

2004
(42 Cases)

P1 = 40
P3 = 02
contact = 02

Most recent virus – 24th August 2004, Bijnor

* data as on 24th sep-04
Location of poliovirus, 2005

66 Cases*
35 Districts*

- Polio in AFP
  - Bihar: 30
  - Uttar Pradesh: 29
  - Jharkhand: 2
  - Delhi: 1
  - Gujarat: 1
  - Uttranchal: 1
  - Punjab: 1
  - Haryana: 1
Location of poliovirus, 2006*

(297 cases)

Most recent virus – 20th August, 2006
Rampur

* data as on 15th September 2006

Uttar Pradesh 269
Bihar 17
Haryana 5
Chandigarh 1
Madhya Pradesh 1
Jharkhand 1
West Bengal 1
Uttaranchal 1
Maharashtra 1

Polio in AFP

* data as on 15th September 2006
Wild virus positive cases 2005
Uttar Pradesh

29 Cases
14 Districts
24 Blocks

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Total P1 wild cases =25
Total P3 wild cases =04
Wild virus positive cases 2006*
Uttar Pradesh

269 Cases*
29 Districts*
118 Blocks*

Most recent virus – 20th August, 2006 Rampur

Total P1 wild cases =264
Total P3 wild cases =05

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Total P1W = 264
Total P3W = 05

* data as on 15th Sept 2006
Status of WPV Cases by Community – Uttar Pradesh 2003-2006*

Year – 2003
N-88
Muslim 68%
Others 32%

Year – 2004
N-82
Muslim 78%
Others 22%

Year – 2005
N-29
Muslim 59%
Others 41%

Year – 2006*
N-269
Muslim 67%
Others 33%

*Data as on 15th Sept, 2006
Prevalence

• Lameness (of leg) Surveys – number of cases

• Rough Prevalence = cases x 1.25 (includes all other sites)

• Prevalence of all cases = RP x 1.33 (to account for attrition)
The Agent
Classification of Poliovirus

- Family – **Picornaviridae**
- Genera –
  1. **Enteroviruses** –
     (a) **Poliovirus**
     (b) Non-Polio Enteroviruses or NPEV - Coxsackie, Echo
  2. **Rhinoviruses** – Influenza virus
  3. **Aphthoviruses**
  4. **Cardioviruses**
The Polio virus

- Single strand of positive sense RNA
- Stable at Acid pH, not affected by lipid solvents
- Looses infectivity in 30 minutes at 56°C
- Period of Communicability – 7-10 days before & after the onset
One of the smallest RNA viruses, measuring about 25 nm in diameter

Viral particles seen by TEM
Epidemiology of Poliovirus

• 3 types
  Type 1 (Brunhilde/Mahoney): mostly causes outbreaks
  Type 2 (Lansing): easiest to eradicate
  Type 3 (Leon): often last to be eradicated

• Highly contagious (usually infects 100% of all susceptibles)

• Occurs worldwide and is seasonal

• Inapparent to apparent infection ratio = 200-1000:1
Polioivirus type 1 - Mahoney
Poliovirus Type 2 Lansing

X-ray Structure determination:


Structure (London) 5, 961–978

STRUCTURE OF POLIOMYRUS TYPE 2 LANING COMPLEXED WITH ANTIVIRAL AGENT SCH-48173: COMPARISON OF THE STRUCTURAL AND BIOLOGICAL PROPERTIES OF THREE POLIOMYRUS SEROTYPES

(PDB ENTRY: 1EAH)

Radial Depth Cue Rendering with grasp (A. NICHOLLS) on Silicon Graphics:

J-Y. SGRO
**Polioviruses:** Are human enteroviruses that exist as three well defined serotypes, which infect cells via a specific receptor [PVR: CD 155]

**Wild polioviruses:** field isolates and reference strains of polioviruses known to have circulated persistently in the community.

**Oral poliovirus vaccine strains:** attenuated polioviruses approved for use in oral vaccines by national control authorities.

**Vaccine derived polioviruses:** mutated progeny of approved poliovirus vaccine strain.
Epidemiology of Poliovirus

Reservoir

• infects only humans

• no animal reservoir

• does not survive long in environment

• no long term carrier state

• Survival outside human – very brief
The Host
• Age – most vulnerable 6 months to 3 years

• Sex – 3 males:1 female

• Risk Factors – Fatigue, trauma, IM injections, tonsillectomy, alum containing DPT

• Immunity – No cross immunity, by natural/immunization
Immunity

• Active
  – through immunization / natural infection
  – immunity believed to be lifelong
  – immunity to one type not protective against infection with other types
  – two types of immunity: intestinal and humoral

• Passive
  – infants born to mothers with high antibody protected for first several weeks
The Environment
Rate of inactivation of this virus varies with immediate environment. *Infectivity decreases by 90%*

- In soil - every 20 days in winter.
  - every 1.5 days in summer.
- In water at ambient temperature –
  » sewage water every 26 days.
  » freshwater every 5.5 days.
  » sea water every 2.5 days.

- It can survive at –
  Freezing temperature for many years,
  Under refrigeration for many months,
  At room temperature for many days.

- Rate of inactivation - slows by presence of org matter.
Place and time

- Place
  - occurrence, not randomly distributed

- Incidence highest in -
  - low immunization coverage
  - poor sanitation
  - crowded conditions
  - urban slums

- Time - seasonal
  - rainy season in warm climate countries
  - summer / early autumn in temperate climate countries
Transmission

- Rapid widespread transmission to non-immune children
- Clusters of susceptibles needed to maintain circulation
- Silent transmission - >99% of cases are sub clinical
- Highly communicable
  - One infected individual will infect all non-immune persons in a household
- Faeco-oral route predominates
Virus Excretion

- Virus *intermittently* excreted for 6-8 weeks after infection
- Most heavy excretion
  - just prior to paralysis onset
  - up to first two weeks
  - dramatically tapers off after 4 weeks
SPREAD OF POLIO IN THE COMMUNITY

CHILD INFECTED WITH POLIOVIRUS PASSING STOOL

POLIOVIRUS ENTERS THE MOUTH OF ANOTHER CHILD

DIRTY HANDS NOT WASHED PROPERLY AFTER BEING IN CONTACT WITH STOOL

CONTAMINATED WATER/FOOD

CHILD GETS POLIO
Incubation period

Short incubation period
usually 7-14 days,
but may be as short as 4 days (range 3-35 days)
Pathogenesis

- Virus enters oral cavity
- Local replication in tissues expressing receptor (tonsils, intestinal M cells, Peyer patches of ileum, and lymph nodes)
- Viremia with hematologic spread to CNS
- Retrograde spread along neurons to spinal cord
- Motor neurons destroyed by viral replication
- Paralysis extent depends on proportion of motor neurons lost
Cross-section of the poliovirus showing the RNA, capsid, and nerve cell receptors
1. A poliovirus approaches a nerve cell via the bloodstream.

2. Nerve cell receptors attach to the virus.

3. The capsid (protein shell) of the virus breaks to release its RNA (genetic material) into the cell.

4. Polio RNA moves toward a ribosome, the cell’s protein assembly station.

5. Polio RNA takes over the ribosome and forces it to make more polio RNA and more capsids.

6. The new polio capsids and new polio RNA unite to form more polioviruses.

7. The host cell swells and bursts, releasing thousands of new viruses back into the bloodstream.
Clinical Outcome of Poliovirus Infections

Paralysis is unusual manifestation of infection

- Paralytic poliomyelitis
- Clinical illness, no paralysis
- Asymptomatic infection

- Spinal: 85-90%
- Bulbospinal: 10-15%
- Bulbar: <1%
- Encephalitic: Rare
Clinical aspects

A. Inapparent (asymptomatic) in 90-95%

B. Abortive in 4-8%: non-specific symptoms

- low grade fever
- malaise
- sore throat
- Anorexia, nausea, vomiting
- Unlocalized abdominal pain, constipation
- recovery rapid and complete
- no paralysis
- indistinguishable from other mild viral infections
C. Non paralytic
- Symptoms similar to abortive
- Headache nausea vomiting more intense
- Stiffness and soreness of muscles in neck, trunk & limbs

D. Paralytic Poliomyelitis - in 0.5% of infections
- symptoms in 2 phases – minor and major CNS phase
- sometimes separated by several days without symptoms
• **Minor phase** – indistinguishable with abortive polio

• **Major phase**
  – muscle pain, spasms
  – return of fever
  – rapid onset of flaccid paralysis
  – progression usually complete within 72 hours
  – sensation remains normal
  – reflexes (DTRs) diminished
  – asymmetric paralysis (legs>arms)
  – proximal more than distal
  – residual flaccid paralysis at 60 days
Clinical aspects ....

• Rarely "bulbar polio"
  – affects motor neurons of cranial nerves
  – may develop
    • respiratory insufficiency
    • difficulty in swallowing, eating and speaking
    • risk of death high

• Very rarely poliovirus may cause meningitis or encephalitis
  – clinically indistinguishable from other causes
Prognosis

Among children who are paralyzed by polio:

• 30% make a full recovery
• 30% are left with mild paralysis
• 30% have medium to severe paralysis
• 10% die
Distinguishing Clinical aspects....

• asymmetric flaccid paralysis (GBS)
• proximal > distal
• rapid progression to paralysis 2-3 days
• deep tendon reflexes diminished or absent (UMN Paralysis)
• fever at onset, muscle pain
• preservation of sensory nerve function (Transverse Myelitis)
• residual paralysis after 60 days
Differential Diagnosis

• AIDP (Acute Infectious Demyelinating Polyneuropathy) or Gullian-Barre Syndrome of Infectious Polyneuropathy
• Traumatic Neuritis
• Transverse Myelitis
• Post-dipthehric Polyneuritis
• Hemiparesis/Hemiplegia
• Transient Paralysis – Unproven hypokalemia
• Poisonings
# Differential Diagnosis

<table>
<thead>
<tr>
<th>Signs &amp; Symptoms</th>
<th>Polio</th>
<th>GBS</th>
<th>Transverse Myelitis</th>
<th>Traumatic Neuritis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fever Onset</td>
<td>High, always present</td>
<td>Not Common</td>
<td>Rare</td>
<td>Common</td>
</tr>
<tr>
<td>Flaccidity</td>
<td>Acute, asymmetrical, proximal</td>
<td>Acute, asymmetrical, distal</td>
<td>Acute, symmetrical, both lower limbs</td>
<td>Acute, asymmetric limbs</td>
</tr>
<tr>
<td>M. Tone</td>
<td>Diminished</td>
<td>Diminished</td>
<td>Diminished in LL</td>
<td>Diminished</td>
</tr>
<tr>
<td>DTRs</td>
<td>Decreased or absent</td>
<td>Absent</td>
<td>Absent in LL early, hyper-reflexia later</td>
<td>Decreased or absent</td>
</tr>
<tr>
<td>Sensation</td>
<td>Severe Myalgia &amp; backache, NO sensory changes</td>
<td>Cramps, tingling, hypoanesthesia</td>
<td>Anesthesia</td>
<td>Pain in gluteal region</td>
</tr>
<tr>
<td>Cranial Nerve</td>
<td>Only in Bulbar and Bulbospinal</td>
<td>Often</td>
<td>Absent</td>
<td>Absent</td>
</tr>
<tr>
<td>Bladder dysfunction</td>
<td>Absent</td>
<td>Transient</td>
<td>Present</td>
<td>Never</td>
</tr>
<tr>
<td>NCV</td>
<td>Abnormal – AHC disease</td>
<td>Abnormal - demyelination</td>
<td>Normal/abnormal</td>
<td>Abnormal in affected nerve</td>
</tr>
<tr>
<td>EMG</td>
<td>Abnormal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
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</tbody>
</table>
Molecular Epidemiology - Tracking the virus

- Possible through genetic mapping & matching
- Origin of virus determined
- Identification of persistent reservoirs
- Detecting gaps in AFP surveillance
- Detect importations
- When and where and was the transmission of various lineages interrupted
Biodiversity of type P1 wild, 2002

Lineage A

Lineage B

Lineage C
Importations - 2003
**P1 poliovirus**

Genetic lineages over the years

<table>
<thead>
<tr>
<th>Year</th>
<th>Lineage</th>
<th>Chains</th>
<th>Evolution</th>
</tr>
</thead>
<tbody>
<tr>
<td>2000</td>
<td>A</td>
<td>2 Chains</td>
<td>B1 → 2 Chains</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>2 Chains</td>
<td>B2 → 4 Chains</td>
</tr>
<tr>
<td></td>
<td>C</td>
<td>2 Chains</td>
<td>B3 → 4 Chains</td>
</tr>
<tr>
<td>2001</td>
<td>A</td>
<td>6 Chains</td>
<td>A → 6 Chains</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>3 Chains</td>
<td>B1 → 2 Chains</td>
</tr>
<tr>
<td></td>
<td>C</td>
<td>4 Chains</td>
<td>C → 10 Chains</td>
</tr>
<tr>
<td>2002</td>
<td>A</td>
<td>3 Chains</td>
<td>A → 3 Chains</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>1 Chains</td>
<td>B2 → 3 Chains</td>
</tr>
<tr>
<td></td>
<td>C</td>
<td>2 Chains</td>
<td>C → 10 Chains</td>
</tr>
<tr>
<td>2003</td>
<td>4 Chains</td>
<td>1 Chains</td>
<td>C → 1 Chains</td>
</tr>
</tbody>
</table>