[Template:Redirect](/wiki/Template:Redirect" \o "Template:Redirect) [Template:Distinguish2](/wiki/Template:Distinguish2) [Template:Pp-semi-indef](/wiki/Template:Pp-semi-indef) [Template:Use dmy dates](/wiki/Template:Use_dmy_dates) [Template:Infobox medical condition](/wiki/Template:Infobox_medical_condition) **Poliomyelitis**, often called **polio** or **infantile paralysis**, is an [infectious disease](/wiki/Infectious_disease) caused by the [poliovirus](/wiki/Poliovirus). In about 0.5% of cases there is [muscle weakness](/wiki/Muscle_weakness) resulting in an [inability to move](/wiki/Flaccid_paralysis).<ref name = PinkBook2009/> This can occur over a few hours to few days.<ref name = PinkBook2009/><ref name=WHO2014/> The weakness most often involves the legs but may less commonly involve the muscles of the head, neck and [diaphragm](/wiki/Thoracic_diaphragm). Many but not all people fully recover. In those with muscle weakness about 2% to 5% of children and 15% to 30% of adults die.<ref name = PinkBook2009>[Template:Cite book](/wiki/Template:Cite_book)</ref> Another 25% of people have minor symptoms such as fever and a sore throat and up to 5% have [headache](/wiki/Headache), neck stiffness and pains in the arms and legs.<ref name = PinkBook2009/><ref name=WHO2014/> These people are usually back to normal within one or two weeks. In up to 70% of infections [there are no symptoms](/wiki/Asymptomatic).<ref name = PinkBook2009/> Years after recovery [post-polio syndrome](/wiki/Post-polio_syndrome) may occur, with a slow development of muscle weakness similar to that which the person had during the initial infection.[[1]](#cite_note-1) Poliovirus is usually spread from person to person through [infected fecal matter entering the mouth](/wiki/Fecal-oral_route).<ref name = PinkBook2009/> It may also be spread by food or water containing human feces and less commonly from infected [saliva](/wiki/Saliva).<ref name = PinkBook2009/><ref name=WHO2014/> Those who are infected may spread the disease for up to six weeks even if no symptoms are present. The disease may be diagnosed by finding the virus in the [feces](/wiki/Feces) or detecting [antibodies](/wiki/Antibodies) against it in the blood. The disease only occurs naturally in humans.<ref name = PinkBook2009/>

The disease is preventable with the [polio vaccine](/wiki/Polio_vaccine); however, a number of doses are required for it to be effective.<ref name=WHO2014/> The [United States Center for Disease Control](/wiki/United_States_Center_for_Disease_Control) recommends polio vaccination boosters for travelers and those who live in countries where the disease is occurring.[[2]](#cite_note-2) Once infected there is no specific treatment.<ref name=WHO2014/> In 2015 polio affected less than 100 people down from 350,000 cases in 1988.[[3]](#cite_note-3)<ref name=WHO2014>[Template:Cite web](/wiki/Template:Cite_web)</ref> In 2014 the disease was only spreading between people in [Afghanistan](/wiki/Afghanistan), [Nigeria](/wiki/Nigeria), and [Pakistan](/wiki/Pakistan).<ref name=WHO2014/> In 2015 Nigeria had stopped the spread of wild poliovirus.[[4]](#cite_note-4) Poliomyelitis has existed for thousands of years, with depictions of the disease in ancient art.<ref name = PinkBook2009/> The disease was first recognized as a distinct condition by [Michael Underwood](/wiki/Michael_Underwood_(physician)) in 1789<ref name = PinkBook2009/> and the virus that causes it was first identified in 1908 by [Karl Landsteiner](/wiki/Karl_Landsteiner).[[5]](#cite_note-5) Major [outbreaks](/wiki/Epidemic) started to occur in the late 19th century in Europe and the United States.<ref name = PinkBook2009/> In the 20th century it became one of the most worrying [childhood diseases](/wiki/List_of_childhood_diseases) in these areas.[[6]](#cite_note-6) The first polio vaccine was developed in the 1950s by [Jonas Salk](/wiki/Jonas_Salk).<ref name=Aylward\_2006>[Template:Cite journal](/wiki/Template:Cite_journal)</ref> It is hoped that [vaccination](/wiki/Vaccination) efforts and early detection of cases will result in global [eradication](/wiki/Poliomyelitis_eradication) of the disease by 2018.[[7]](#cite_note-7)[Template:TOC limit](/wiki/Template:TOC_limit)

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## Signs and symptoms[[edit](/index.php?title=(none)&action=edit&section=1)]

|  |  |
| --- | --- |
| **Outcomes of poliovirus infection** | |
| **Outcome** | **Proportion of cases<ref name = PinkBook2009/>** |
| No symptoms | 72% |
| Minor illness | 24% |
| Nonparalytic aseptic meningitis | 1–5% |
| Paralytic poliomyelitis | 0.1–0.5% |
| — Spinal polio | 79% of paralytic cases |
| — Bulbospinal polio | 19% of paralytic cases |
| — Bulbar polio | 2% of paralytic cases |

The term "poliomyelitis" is used to identify the disease caused by any of the three [serotypes](/wiki/Serovar) of poliovirus. Two basic patterns of polio infection are described: a minor illness which does not involve the [central nervous system](/wiki/Central_nervous_system) (CNS), sometimes called abortive poliomyelitis, and a major illness involving the CNS, which may be paralytic or nonparalytic.[[8]](#cite_note-8) In most people with a [normal immune system](/wiki/Immunocompetent), a poliovirus infection is [asymptomatic](/wiki/Subclinical_infection). Rarely, the infection produces minor symptoms; these may include upper [respiratory tract](/wiki/Respiratory_tract) infection ([sore throat](/wiki/Pharyngitis) and fever), [gastrointestinal](/wiki/Human_gastrointestinal_tract) disturbances (nausea, vomiting, [abdominal pain](/wiki/Abdominal_pain), constipation or, rarely, diarrhea), and [influenza-like illness](/wiki/Influenza-like_illness).[[9]](#cite_note-9) The virus enters the central nervous system in about 1% of infections. Most patients with CNS involvement develop nonparalytic [aseptic meningitis](/wiki/Aseptic_meningitis), with symptoms of headache, neck, back, abdominal and extremity pain, fever, vomiting, [lethargy](/wiki/Lethargy), and irritability.[[10]](#cite_note-10)<ref name=Late>[Template:Cite book](/wiki/Template:Cite_book)</ref> About one to five in 1000 cases progress to [paralytic](/wiki/Paralytic) disease, in which the muscles become weak, floppy and poorly controlled, and, finally, completely paralyzed; this condition is known as [acute flaccid paralysis](/wiki/Flaccid_paralysis).[[11]](#cite_note-11) Depending on the site of paralysis, paralytic poliomyelitis is classified as spinal, [bulbar](/wiki/Medulla_oblongata), or bulbospinal. [Encephalitis](/wiki/Encephalitis), an infection of the brain tissue itself, can occur in rare cases, and is usually restricted to infants. It is characterized by confusion, changes in mental status, headaches, fever, and, less commonly, [seizures](/wiki/Seizure) and [spastic paralysis](/wiki/Spastic_paralysis).<ref name= Encephalitis>[Template:Cite book](/wiki/Template:Cite_book)</ref>

## Cause[[edit](/index.php?title=(none)&action=edit&section=2)]

[Template:Main](/wiki/Template:Main) [thumb|right|A](/wiki/File:Polio_EM_PHIL_1875_lores.PNG) [TEM](/wiki/Transmission_electron_microscopy) [micrograph](/wiki/Micrograph) of poliovirus

Poliomyelitis is caused by infection with a member of the [genus](/wiki/Genus) [*Enterovirus*](/wiki/Enterovirus) known as [poliovirus](/wiki/Poliovirus) (PV). This group of [RNA viruses](/wiki/RNA_virus) colonize the [gastrointestinal tract](/wiki/Human_gastrointestinal_tract)<ref name=Harrison>[Template:Cite book](/wiki/Template:Cite_book)</ref> — specifically the [oropharynx](/wiki/Oropharynx) and the [intestine](/wiki/Intestine). The incubation time (to the first signs and symptoms) ranges from three to 35 days, with a more common span of six to 20 days.[[9]](#cite_note-9) PV [infects and causes disease](/wiki/Pathogen) in humans alone.<ref name=Sherris>[Template:Cite book](/wiki/Template:Cite_book)</ref> Its [structure](/wiki/Virus_structure) is very simple, composed of a single [(+) sense](/wiki/Sense_(molecular_biology)) [RNA](/wiki/RNA) [genome](/wiki/Genome) enclosed in a protein shell called a [capsid](/wiki/Capsid).[[12]](#cite_note-12) In addition to protecting the virus’s genetic material, the capsid proteins enable poliovirus to infect certain types of cells. Three [serotypes](/wiki/Serovar) of poliovirus have been identified—poliovirus type 1 (PV1), type 2 (PV2), and type 3 (PV3)—each with a slightly different capsid protein.[[13]](#cite_note-13) All three are extremely [virulent](/wiki/Virulence) and produce the same disease symptoms.[[12]](#cite_note-12) PV1 is the most commonly encountered form, and the one most closely associated with paralysis.<ref name= Ohri/>

Individuals who are exposed to the virus, either through infection or by [immunization](/wiki/Immunization) with polio vaccine, develop [immunity](/wiki/Immunity_(medical)). In immune individuals, [IgA](/wiki/IgA) [antibodies](/wiki/Antibodies) against poliovirus are present in the [tonsils](/wiki/Tonsil) and gastrointestinal tract, and are able to block virus replication; [IgG](/wiki/IgG) and [IgM](/wiki/IgM) antibodies against PV can prevent the spread of the virus to motor neurons of the [central nervous system](/wiki/Central_nervous_system).<ref name=Kew\_2005/> Infection or vaccination with one serotype of poliovirus does not provide immunity against the other serotypes, and full immunity requires exposure to each serotype.<ref name=Kew\_2005/>

A rare condition with a similar presentation, nonpoliovirus poliomyelitis, may result from infections with nonpoliovirus [enteroviruses](/wiki/Enterovirus).[[14]](#cite_note-14)

### Transmission[[edit](/index.php?title=(none)&action=edit&section=3)]

Poliomyelitis is highly contagious via the fecal-oral (intestinal source) and the oral-oral (oropharyngeal source) routes.<ref name=Kew\_2005>[Template:Cite journal](/wiki/Template:Cite_journal)</ref> In endemic areas, wild polioviruses can infect virtually the entire human population.<ref name=McGraw>[Template:Cite book](/wiki/Template:Cite_book)</ref> It is seasonal in [temperate climates](/wiki/Temperate_climate), with peak transmission occurring in summer and autumn.[[15]](#cite_note-15) These seasonal differences are far less pronounced in [tropical](/wiki/Tropical_climate) areas.[[16]](#cite_note-16) The time between first exposure and first symptoms, known as the [incubation period](/wiki/Incubation_period), is usually 6 to 20 days, with a maximum range of three to 35 days.<ref name=Racaniello>[Template:Cite journal](/wiki/Template:Cite_journal)</ref> Virus particles are excreted in the [feces](/wiki/Feces) for several weeks following initial infection.[[17]](#cite_note-17) The disease is [transmitted](/wiki/Transmission_(medicine)) primarily via the [fecal-oral route](/wiki/Fecal-oral_route), by ingesting contaminated food or water. It is occasionally transmitted via the oral-oral route,<ref name= Ohri>[Template:Cite journal](/wiki/Template:Cite_journal) (Available free on [Medscape](/wiki/Medscape); registration required.)</ref> a mode especially visible in areas with good sanitation and hygiene.[[15]](#cite_note-15) Polio is most infectious between seven and 10 days before and after the appearance of symptoms, but transmission is possible as long as the virus remains in the saliva or feces.<ref name= Ohri/>

Factors that increase the risk of polio infection or affect the severity of the disease include [immune deficiency](/wiki/Immune_deficiency),[[18]](#cite_note-18) [malnutrition](/wiki/Malnutrition),[[19]](#cite_note-19) physical activity immediately following the onset of paralysis,[[20]](#cite_note-20) skeletal muscle injury due to [injection](/wiki/Intramuscular_injection) of vaccines or therapeutic agents,[[21]](#cite_note-21) and [pregnancy](/wiki/Pregnancy).<ref name= Evans\_1960>[Template:Cite journal](/wiki/Template:Cite_journal)</ref> Although the virus can cross the [maternal-fetal barrier](/wiki/Placenta) during pregnancy, the fetus does not appear to be affected by either maternal infection or polio vaccination.<ref name=UK>[Template:Cite book](/wiki/Template:Cite_book)</ref> Maternal antibodies also cross the [placenta](/wiki/Placenta), providing [passive immunity](/wiki/Passive_immunity) that protects the infant from polio infection during the first few months of life.[[22]](#cite_note-22) As a precaution against infection, public [swimming pools](/wiki/Swimming_pool) were often closed in affected areas during poliomyelitis epidemics.

## Pathophysiology[[edit](/index.php?title=(none)&action=edit&section=4)]

[thumb|left|A blockage of the](/wiki/File:Polio_spine.png) [lumbar](/wiki/Lumbar) anterior spinal cord [artery](/wiki/Artery) due to polio (PV3)

Poliovirus enters the body through the mouth, infecting the first cells with which it comes in contact — the [pharynx](/wiki/Human_pharynx) and [intestinal mucosa](/wiki/Intestinal_mucosa). It gains entry by binding to an [immunoglobulin-like](/wiki/Immunoglobulin) receptor, known as the poliovirus receptor or [CD155](/wiki/CD155), on the cell membrane.<ref name=He>[Template:Cite journal](/wiki/Template:Cite_journal)</ref> The virus then hijacks the [host cell's](/wiki/Host_(biology)) own machinery, and begins to [replicate](/wiki/Viral_replication). Poliovirus divides within gastrointestinal cells for about a week, from where it spreads to the [tonsils](/wiki/Tonsils) (specifically the [follicular dendritic cells](/wiki/Follicular_dendritic_cell) residing within the tonsilar [germinal centers](/wiki/Germinal_center)), the intestinal [lymphoid tissue](/wiki/Lymphoid_tissue) including the [M cells](/wiki/Microfold_cell) of [Peyer's patches](/wiki/Peyer's_patches), and the deep [cervical](/wiki/Cervical_lymph_nodes) and [mesenteric lymph nodes](/wiki/Inferior_mesenteric_lymph_nodes), where it multiplies abundantly. The virus is subsequently absorbed into the bloodstream.<ref name=Baron>[Template:Cite book](/wiki/Template:Cite_book)</ref>

Known as [viremia](/wiki/Viremia), the presence of a virus in the bloodstream enables it to be widely distributed throughout the body. Poliovirus can survive and multiply within the blood and lymphatics for long periods of time, sometimes as long as 17 weeks.[[23]](#cite_note-23) In a small percentage of cases, it can spread and replicate in other sites, such as [brown fat](/wiki/Brown_fat), the [reticuloendothelial](/wiki/Reticuloendothelial) tissues, and muscle.[[24]](#cite_note-24) This sustained replication causes a major viremia, and leads to the development of minor influenza-like symptoms. Rarely, this may progress and the virus may invade the central nervous system, provoking a local [inflammatory response](/wiki/Inflammatory_response). In most cases, this causes a self-limiting inflammation of the [meninges](/wiki/Meninges), the layers of tissue surrounding the [brain](/wiki/Human_brain), which is known as nonparalytic aseptic meningitis.[[10]](#cite_note-10) Penetration of the CNS provides no known benefit to the virus, and is quite possibly an incidental deviation of a normal gastrointestinal infection.<ref name= Mueller>[Template:Cite journal](/wiki/Template:Cite_journal)</ref> The mechanisms by which poliovirus spreads to the CNS are poorly understood, but it appears to be primarily a chance event—largely independent of the age, gender, or [socioeconomic](/wiki/Socioeconomics) position of the individual.[[25]](#cite_note-25)

### Paralytic polio[[edit](/index.php?title=(none)&action=edit&section=5)]

[thumb|right|Denervation of](/wiki/File:PHIL_2767_Poliovirus_Myotonic_dystrophic_changes.jpg) [skeletal muscle](/wiki/Skeletal_muscle) tissue secondary to poliovirus infection can lead to paralysis.

In around 1% of infections, poliovirus spreads along certain nerve fiber pathways, preferentially replicating in and destroying [motor neurons](/wiki/Motor_neuron) within the [spinal cord](/wiki/Spinal_cord), [brain stem](/wiki/Brain_stem), or [motor cortex](/wiki/Motor_cortex). This leads to the development of paralytic poliomyelitis, the various forms of which (spinal, bulbar, and bulbospinal) vary only with the amount of neuronal damage and inflammation that occurs, and the region of the CNS affected.

The destruction of neuronal cells produces [lesions](/wiki/Lesion) within the [spinal ganglia](/wiki/Dorsal_root_ganglion); these may also occur in the [reticular formation](/wiki/Reticular_formation), [vestibular nuclei](/wiki/Vestibular_nuclei), [cerebellar vermis](/wiki/Cerebellar_vermis), and deep [cerebellar nuclei](/wiki/Cerebellar_nuclei).[[25]](#cite_note-25) Inflammation associated with [nerve cell](/wiki/Neuron) destruction often alters the color and appearance of the gray matter in the [spinal column](/wiki/Spinal_column), causing it to appear reddish and swollen.<ref name=Chamberlin\_2005/> Other destructive changes associated with paralytic disease occur in the [forebrain](/wiki/Forebrain) region, specifically the [hypothalamus](/wiki/Hypothalamus) and [thalamus](/wiki/Thalamus).[[25]](#cite_note-25) The molecular mechanisms by which poliovirus causes paralytic disease are poorly understood.

Early symptoms of paralytic polio include high fever, headache, stiffness in the back and neck, asymmetrical weakness of various muscles, sensitivity to touch, [difficulty swallowing](/wiki/Dysphagia), [muscle pain](/wiki/Myalgia), loss of superficial and deep [reflexes](/wiki/Reflex), [paresthesia](/wiki/Paresthesia) (pins and needles), irritability, constipation, or difficulty urinating. Paralysis generally develops one to ten days after early symptoms begin, progresses for two to three days, and is usually complete by the time the fever breaks.<ref name= Silverstein>[Template:Cite book](/wiki/Template:Cite_book)</ref>

The likelihood of developing paralytic polio increases with age, as does the extent of paralysis. In children, nonparalytic meningitis is the most likely consequence of CNS involvement, and paralysis occurs in only one in 1000 cases. In adults, paralysis occurs in one in 75 cases.[[26]](#cite_note-26) In children under five years of age, paralysis of one leg is most common; in adults, extensive paralysis of the [chest](/wiki/Chest) and [abdomen](/wiki/Abdomen) also affecting all four limbs—[quadriplegia](/wiki/Quadriplegia)—is more likely.<ref name= Young>[Template:Cite journal](/wiki/Template:Cite_journal)</ref> Paralysis rates also vary depending on the serotype of the infecting poliovirus; the highest rates of paralysis (one in 200) are associated with poliovirus type 1, the lowest rates (one in 2,000) are associated with type 2.<ref name=Nathanson>[Template:Cite journal](/wiki/Template:Cite_journal)</ref>

#### Spinal polio[[edit](/index.php?title=(none)&action=edit&section=6)]

[thumb|left|The location of](/wiki/File:Polio_spinal_diagram.PNG) [motor neurons](/wiki/Motor_neuron) in the [anterior horn cells](/wiki/Anterior_horn_(spinal_cord)) of the [spinal column](/wiki/Spinal_column)

Spinal polio, the most common form of paralytic poliomyelitis, results from viral invasion of the motor neurons of the [anterior horn cells](/wiki/Anterior_horn_(spinal_cord)), or the [ventral](/wiki/Ventral) (front) [grey matter](/wiki/Grey_matter) section in the [spinal column](/wiki/Spinal_column), which are responsible for movement of the muscles, including those of the [trunk](/wiki/Torso), [limbs](/wiki/Limb_(anatomy)), and the [intercostal muscles](/wiki/Intercostal_muscle).<ref name= Henry1>[Template:Cite book](/wiki/Template:Cite_book)</ref> Virus invasion causes inflammation of the nerve cells, leading to damage or destruction of motor neuron [ganglia](/wiki/Ganglion). When spinal neurons die, [Wallerian degeneration](/wiki/Wallerian_degeneration) takes place, leading to weakness of those muscles formerly [innervated](/wiki/Innervate) by the now-dead neurons.[[27]](#cite_note-27) With the destruction of nerve cells, the muscles no longer receive signals from the brain or spinal cord; without nerve stimulation, the muscles [atrophy](/wiki/Atrophy), becoming weak, floppy and poorly controlled, and finally completely paralyzed.[[11]](#cite_note-11) Maximum paralysis progresses rapidly (two to four days), and usually involves fever and muscle pain. Deep [tendon](/wiki/Stretch_reflex) [reflexes](/wiki/Reflex) are also affected, and are typically absent or diminished; [sensation](/wiki/Sense) (the ability to feel) in the paralyzed limbs, however, is not affected.[[28]](#cite_note-28) The extent of spinal paralysis depends on the region of the cord affected, which may be [cervical](/wiki/Neck), [thoracic](/wiki/Human_thorax), or [lumbar](/wiki/Lumbar).<ref name=Guide>[Template:Cite book](/wiki/Template:Cite_book)</ref> The virus may affect muscles on both sides of the body, but more often the paralysis is [asymmetrical](/wiki/Asymmetry).[[29]](#cite_note-29) Any [limb](/wiki/Limb_(anatomy)) or combination of limbs may be affected—one leg, one arm, or both legs and both arms. Paralysis is often more severe [proximally](/wiki/Proximally) (where the limb joins the body) than [distally](/wiki/Distally) (the [fingertips](/wiki/Fingertip) and [toes](/wiki/Toe)).[[29]](#cite_note-29)

#### Bulbar polio[[edit](/index.php?title=(none)&action=edit&section=7)]

[thumb|right|The location and anatomy of the bulbar region (in orange)](/wiki/File:Brain_bulbar_region.svg)

Making up about 2% of cases of paralytic polio, bulbar polio occurs when poliovirus invades and destroys nerves within the [bulbar](/wiki/Bulbar) region of the [brain stem](/wiki/Brain_stem).[[9]](#cite_note-9) The bulbar region is a [white matter](/wiki/White_matter) pathway that connects the [cerebral cortex](/wiki/Cerebral_cortex) to the brain stem. The destruction of these nerves weakens the muscles supplied by the [cranial nerves](/wiki/Cranial_nerve), producing symptoms of [encephalitis](/wiki/Encephalitis), and causes [difficulty breathing](/wiki/Dyspnea), speaking and swallowing.[[30]](#cite_note-30) Critical nerves affected are the [glossopharyngeal nerve](/wiki/Glossopharyngeal_nerve) (which partially controls swallowing and functions in the throat, tongue movement, and taste), the [vagus nerve](/wiki/Vagus_nerve) (which sends signals to the heart, intestines, and lungs), and the [accessory nerve](/wiki/Accessory_nerve) (which controls upper neck movement). Due to the effect on swallowing, secretions of [mucus](/wiki/Mucus) may build up in the airway, causing suffocation.[[31]](#cite_note-31) Other signs and symptoms include [facial weakness](/wiki/Facial_weakness) (caused by destruction of the [trigeminal nerve](/wiki/Trigeminal_nerve) and [facial nerve](/wiki/Facial_nerve), which innervate the cheeks, [tear ducts](/wiki/Tear_duct), gums, and muscles of the face, among other structures), [double vision](/wiki/Diplopia), difficulty in chewing, and abnormal [respiratory rate](/wiki/Respiratory_rate), depth, and rhythm (which may lead to [respiratory arrest](/wiki/Respiratory_arrest)). [Pulmonary edema](/wiki/Pulmonary_edema) and [shock](/wiki/Shock_(circulatory)) are also possible and may be fatal.<ref name=Guide/>

#### Bulbospinal polio[[edit](/index.php?title=(none)&action=edit&section=8)]

Approximately 19% of all paralytic polio cases have both bulbar and spinal symptoms; this subtype is called respiratory or bulbospinal polio.[[9]](#cite_note-9) Here, the virus affects the upper part of the cervical spinal cord ([cervical vertebrae](/wiki/Cervical_vertebrae) C3 through C5), and paralysis of the [diaphragm](/wiki/Thoracic_diaphragm) occurs. The critical nerves affected are the [phrenic nerve](/wiki/Phrenic_nerve) (which drives the diaphragm to inflate the [lungs](/wiki/Human_lung)) and those that drive the muscles needed for swallowing. By destroying these nerves, this form of polio affects breathing, making it difficult or impossible for the patient to breathe without the support of a [ventilator](/wiki/Medical_ventilator). It can lead to paralysis of the arms and legs and may also affect swallowing and heart functions.<ref name= Hoyt/>

## Diagnosis[[edit](/index.php?title=(none)&action=edit&section=9)]

Paralytic poliomyelitis may be clinically suspected in individuals experiencing acute onset of flaccid paralysis in one or more limbs with decreased or absent tendon reflexes in the affected limbs that cannot be attributed to another apparent cause, and without sensory or [cognitive](/wiki/Cognitive) loss.[[32]](#cite_note-32) A laboratory diagnosis is usually made based on recovery of poliovirus from a stool sample or a swab of the [pharynx](/wiki/Human_pharynx). [Antibodies](/wiki/Antibody) to poliovirus can be diagnostic, and are generally detected in the blood of infected patients early in the course of infection.[[9]](#cite_note-9) Analysis of the patient's [cerebrospinal fluid](/wiki/Cerebrospinal_fluid) (CSF), which is collected by a [lumbar puncture](/wiki/Lumbar_puncture) ("spinal tap"), reveals an increased number of [white blood cells](/wiki/White_blood_cell) (primarily [lymphocytes](/wiki/Lymphocyte)) and a mildly elevated protein level. Detection of virus in the CSF is diagnostic of paralytic polio, but rarely occurs.<ref name = PinkBook2009/>

If poliovirus is isolated from a patient experiencing acute flaccid paralysis, it is further tested through [oligonucleotide](/wiki/Oligonucleotide) mapping ([genetic fingerprinting](/wiki/Genetic_fingerprint)), or more recently by [PCR](/wiki/Polymerase_chain_reaction) amplification, to determine whether it is "[wild type](/wiki/Wild_type)" (that is, the virus encountered in nature) or "vaccine type" (derived from a strain of poliovirus used to produce polio vaccine).[[33]](#cite_note-33) It is important to determine the source of the virus because for each reported case of paralytic polio caused by wild poliovirus, an estimated 200 to 3,000 other contagious [asymptomatic carriers](/wiki/Asymptomatic_carrier) exist.[[34]](#cite_note-34)

## Prevention[[edit](/index.php?title=(none)&action=edit&section=10)]

### Passive immunization[[edit](/index.php?title=(none)&action=edit&section=11)]

In 1950, [William Hammon](/wiki/William_Hammon) at the [University of Pittsburgh](/wiki/University_of_Pittsburgh) purified the [gamma globulin](/wiki/Gamma_globulin) component of the [blood plasma](/wiki/Blood_plasma) of polio survivors.<ref name=Hammon\_1955>[Template:Cite journal](/wiki/Template:Cite_journal)</ref> Hammon proposed the gamma globulin, which contained antibodies to poliovirus, could be used to halt poliovirus infection, prevent disease, and reduce the severity of disease in other patients who had contracted polio. The results of a large [clinical trial](/wiki/Clinical_trial) were promising; the gamma globulin was shown to be about 80% effective in preventing the development of paralytic poliomyelitis.[[35]](#cite_note-35) It was also shown to reduce the severity of the disease in patients who developed polio.[[36]](#cite_note-36) Due to the limited supply of blood plasma gamma globulin was later deemed impractical for widespread use and the medical community focused on the development of a polio vaccine.<ref name=Rinaldo>[Template:Cite journal](/wiki/Template:Cite_journal)</ref>

### Vaccine[[edit](/index.php?title=(none)&action=edit&section=12)]

[Template:Main](/wiki/Template:Main) [thumb|right|A child receiving an oral polio vaccine](/wiki/File:Poliodrops.jpg)

Two types of vaccine are used throughout the world to combat polio. Both types induce immunity to polio, efficiently blocking person-to-person transmission of wild poliovirus, thereby protecting both individual vaccine recipients and the wider community (so-called [herd immunity](/wiki/Herd_immunity)).<ref name=Fine>[Template:Cite journal](/wiki/Template:Cite_journal)</ref>

The first candidate [polio vaccine](/wiki/Polio_vaccine), based on one serotype of a live but [attenuated (weakened) virus](/wiki/Attenuated_vaccine), was developed by the [virologist](/wiki/Virologist) [Hilary Koprowski](/wiki/Hilary_Koprowski). Koprowski's prototype vaccine was given to an eight-year-old boy on 27 February 1950.[[37]](#cite_note-37) Koprowski continued to work on the vaccine throughout the 1950s, leading to large-scale trials in the then [Belgian Congo](/wiki/Belgian_Congo) and the vaccination of seven million children in Poland against serotypes PV1 and PV3 between 1958 and 1960.<ref name=Sanofi>[Template:Wayback](/wiki/Template:Wayback) Accessed 16 December 2009.</ref>

The second inactivated virus vaccine was developed in 1952 by [Jonas Salk](/wiki/Jonas_Salk) at the University of Pittsburgh, and announced to the world on 12 April 1955.<ref name= Spice>[Template:Cite news](/wiki/Template:Cite_news)</ref> The Salk vaccine, or inactivated poliovirus vaccine (IPV), is based on poliovirus grown in a type of monkey kidney [tissue culture](/wiki/Tissue_culture) ([vero cell](/wiki/Vero_cell) [line](/wiki/Cell_culture)), which is chemically inactivated with [formalin](/wiki/Formalin).[[15]](#cite_note-15) After two doses of IPV (given by [injection](/wiki/Injection_(medicine))), 90% or more of individuals develop protective antibody to all three [serotypes](/wiki/Serotype) of poliovirus, and at least 99% are immune to poliovirus following three doses.[[9]](#cite_note-9) Subsequently, [Albert Sabin](/wiki/Albert_Sabin) developed another live, oral polio vaccine (OPV). It was produced by the repeated passage of the virus through nonhuman cells at sub[physiological](/wiki/Physiological) temperatures.<ref name=Sabin\_1973>[Template:Cite journal](/wiki/Template:Cite_journal)</ref> The attenuated poliovirus in the Sabin vaccine replicates very efficiently in the gut, the primary site of wild poliovirus infection and replication, but the vaccine strain is unable to replicate efficiently within [nervous system](/wiki/Nervous_system) tissue.[[38]](#cite_note-38) A single dose of Sabin's oral polio vaccine produces immunity to all three poliovirus serotypes in about 50% of recipients. Three doses of live-attenuated OPV produce protective antibody to all three poliovirus types in more than 95% of recipients.[[9]](#cite_note-9) [Human trials](/wiki/Clinical_trial) of Sabin's vaccine began in 1957,<ref name=ScienceOdyssey>[Template:Cite web](/wiki/Template:Cite_web)</ref> and in 1958 it was selected, in competition with the live vaccines of Koprowski and other researchers, by the US National Institutes of Health.<ref name=Sanofi/> Licensed in 1962,<ref name=ScienceOdyssey/> it rapidly became the only polio vaccine used worldwide.<ref name=Sanofi/>

Because OPV is inexpensive, easy to administer, and produces excellent immunity in the intestine (which helps prevent infection with wild virus in areas where it is [endemic](/wiki/Endemic_(epidemiology))), it has been the vaccine of choice for controlling poliomyelitis in many countries.<ref name=Peds>[Template:Cite journal](/wiki/Template:Cite_journal)</ref> On very rare occasions (about one case per 750,000 vaccine recipients), the attenuated virus in OPV reverts into a form that can paralyze.<ref name=Racaniello/> Most [industrialized countries](/wiki/Developed_country) have switched to IPV, which cannot revert, either as the sole vaccine against poliomyelitis or in combination with oral polio vaccine.[[39]](#cite_note-39)

## Treatment[[edit](/index.php?title=(none)&action=edit&section=13)]

There is no [cure](/wiki/Cure) for polio. The focus of modern treatment has been on providing relief of symptoms, speeding recovery and preventing complications. Supportive measures include [antibiotics](/wiki/Antibiotics) to prevent infections in weakened muscles, [analgesics](/wiki/Analgesics) for pain, moderate exercise and a nutritious diet.<ref name=Daniel>[Template:Cite book](/wiki/Template:Cite_book)</ref> Treatment of polio often requires long-term rehabilitation, including [occupational therapy](/wiki/Occupational_therapy), [physical therapy](/wiki/Physical_therapy), braces, corrective shoes and, in some cases, [orthopedic surgery](/wiki/Orthopedic_surgery).[[40]](#cite_note-40) Portable [ventilators](/wiki/Ventilator) may be required to support breathing. Historically, a noninvasive, negative-pressure ventilator, more commonly called an [iron lung](/wiki/Negative_pressure_ventilator), was used to artificially maintain respiration during an acute polio infection until a person could breathe independently (generally about one to two weeks). Today, many polio survivors with permanent respiratory paralysis use modern [jacket-type](/wiki/Biphasic_Cuirass_Ventilation) negative-pressure ventilators worn over the chest and abdomen.[[41]](#cite_note-41) Other [historical treatments for polio](/wiki/History_of_poliomyelitis#Historical_treatments) include [hydrotherapy](/wiki/Hydrotherapy), [electrotherapy](/wiki/Electrotherapy), massage and passive motion exercises, and surgical treatments, such as tendon lengthening and nerve grafting.[[11]](#cite_note-11)

## Prognosis[[edit](/index.php?title=(none)&action=edit&section=14)]

[thumb|A child with a deformity of her right leg due to polio](/wiki/File:Polio_sequelle.jpg) Patients with abortive polio infections recover completely. In those who develop only aseptic meningitis, the symptoms can be expected to persist for two to ten days, followed by complete recovery.<ref name=Neumann/> In cases of spinal polio, if the affected nerve cells are completely destroyed, paralysis will be permanent; cells that are not destroyed, but lose function temporarily, may recover within four to six weeks after onset.<ref name=Neumann>[Template:Cite journal](/wiki/Template:Cite_journal) Reproduced online with permission by Post-Polio Health International; retrieved on 10 November 2007.</ref> Half the patients with spinal polio recover fully; one-quarter recover with mild disability, and the remaining quarter are left with severe disability.[[42]](#cite_note-42) The degree of both acute paralysis and residual paralysis is likely to be proportional to the degree of [viremia](/wiki/Viremia), and [inversely proportional](/wiki/Inversely_proportional) to the degree of [immunity](/wiki/Immunity_(medical)).<ref name= Mueller/> Spinal polio is rarely fatal.[[31]](#cite_note-31) Without respiratory support, consequences of poliomyelitis with [respiratory](/wiki/Respiration_(physiology)) involvement include [suffocation](/wiki/Suffocation) or [pneumonia from aspiration of secretions](/wiki/Aspiration_pneumonia).<ref name= Goldberg>[Template:Cite journal](/wiki/Template:Cite_journal)</ref> Overall, 5–10% of patients with paralytic polio die due to the paralysis of muscles used for breathing. The [case fatality rate](/wiki/Case_fatality_rate) (CFR) varies by age: 2–5% of children and up to 15–30% of adults die.[[9]](#cite_note-9) Bulbar polio often causes death if respiratory support is not provided;[[43]](#cite_note-43) with support, its CFR ranges from 25 to 75%, depending on the age of the patient.[[9]](#cite_note-9)[[44]](#cite_note-44) When intermittent positive pressure ventilation is available, the fatalities can be reduced to 15%.<ref name=Wackers>[Template:Cite journal](/wiki/Template:Cite_journal)</ref>

### Recovery[[edit](/index.php?title=(none)&action=edit&section=15)]

Many cases of poliomyelitis result in only temporary paralysis.[[11]](#cite_note-11) Nerve impulses return to the formerly paralyzed muscle within a month, and recovery is usually complete in six to eight months.[[45]](#cite_note-45) The [neurophysiological](/wiki/Neurophysiology) processes involved in recovery following acute paralytic poliomyelitis are quite effective; muscles are able to retain normal strength even if half the original motor neurons have been lost.[[46]](#cite_note-46) Paralysis remaining after one year is likely to be permanent, although modest recoveries of muscle strength are possible 12 to 18 months after infection.[[45]](#cite_note-45) One mechanism involved in recovery is nerve terminal sprouting, in which remaining brainstem and spinal cord motor neurons develop new branches, or axonal sprouts.[[47]](#cite_note-47) These sprouts can [reinnervate](/wiki/Reinnervate) orphaned muscle fibers that have been denervated by acute polio infection,<ref name=Agre>[Template:Cite journal](/wiki/Template:Cite_journal)</ref> restoring the fibers' capacity to contract and improving strength.[[48]](#cite_note-48) Terminal sprouting may generate a few significantly enlarged motor neurons doing work previously performed by as many as four or five units:[[26]](#cite_note-26) a single motor neuron that once controlled 200 muscle cells might control 800 to 1000 cells. Other mechanisms that occur during the rehabilitation phase, and contribute to muscle strength restoration, include [myofiber hypertrophy](/wiki/Muscle_hypertrophy)—enlargement of muscle fibers through exercise and activity—and transformation of [type II muscle fibers](/wiki/Muscle_fiber#Type_II) to [type I muscle fibers](/wiki/Muscle_fiber#Type_I).[[49]](#cite_note-49)<ref name = Grimby\_1989>[Template:Cite journal](/wiki/Template:Cite_journal)</ref>

In addition to these physiological processes, the body possesses a number of compensatory mechanisms to maintain function in the presence of residual paralysis. These include the use of weaker muscles at a higher than usual intensity relative to the [muscle's maximal capacity](/wiki/Muscle_contraction#Contractions,_by_muscle_type), enhancing athletic development of previously little-used muscles, and using [ligaments](/wiki/Ligament) for stability, which enables greater mobility.[[50]](#cite_note-50)

### Complications[[edit](/index.php?title=(none)&action=edit&section=16)]

Residual complications of paralytic polio often occur following the initial recovery process.[[30]](#cite_note-30) Muscle [paresis](/wiki/Paresis) and paralysis can sometimes result in [skeletal](/wiki/Skeletal) deformities, tightening of the joints and movement disability. Once the muscles in the limb become flaccid, they may interfere with the function of other muscles. A typical manifestation of this problem is [equinus foot](/wiki/Equinus_foot) (similar to [club foot](/wiki/Club_foot)). This deformity develops when the muscles that pull the toes downward are working, but those that pull it upward are not, and the foot naturally tends to drop toward the ground. If the problem is left untreated, the [Achilles tendons](/wiki/Achilles_tendon) at the back of the foot retract and the foot cannot take on a normal position. Polio victims that develop equinus foot cannot walk properly because they cannot put their heel on the ground. A similar situation can develop if the arms become paralyzed.<ref name= Aftereffects>[Template:Cite web](/wiki/Template:Cite_web)</ref> In some cases the growth of an affected leg is slowed by polio, while the other leg continues to grow normally. The result is that one leg is shorter than the other and the person limps and leans to one side, in turn leading to deformities of the spine (such as [scoliosis](/wiki/Scoliosis)).[[51]](#cite_note-51) [Osteoporosis](/wiki/Osteoporosis) and increased likelihood of [bone fractures](/wiki/Bone_fracture) may occur. An intervention to prevent or lessen length disparity can be to perform an [epiphysiodesis](/wiki/Epiphysiodesis) on the distal femoral and proximal tibial/fibular condyles, so that limb's growth is artificially stunted, and by the time of [epiphyseal (growth) plate](/wiki/Epiphyseal_plate) closure, the legs are more equal in length. Alternatively, a person can be fitted with custom made footwear which corrects the difference in leg lengths. Other surgery to re-balance muscular agonist/antagonist imbalances may also be helpful. Extended use of braces or wheelchairs may cause compression [neuropathy](/wiki/Neuropathy), as well as a loss of proper function of the [veins](/wiki/Vein) in the legs, due to pooling of blood in paralyzed lower limbs.<ref name= Hoyt>[Template:Cite book](/wiki/Template:Cite_book)</ref><ref name=MayoComps>[Template:Cite web](/wiki/Template:Cite_web)</ref> Complications from prolonged immobility involving the [lungs](/wiki/Lungs), [kidneys](/wiki/Kidney) and [heart](/wiki/Heart) include [pulmonary edema](/wiki/Pulmonary_edema), [aspiration pneumonia](/wiki/Aspiration_pneumonia), [urinary tract infections](/wiki/Urinary_tract_infection), [kidney stones](/wiki/Kidney_stone), [paralytic ileus](/wiki/Paralytic_ileus), [myocarditis](/wiki/Myocarditis) and [cor pulmonale](/wiki/Cor_pulmonale).[[52]](#cite_note-52)

### Post-polio syndrome[[edit](/index.php?title=(none)&action=edit&section=17)]

[Template:Main](/wiki/Template:Main) Between 25% and 50% of individuals who have recovered from paralytic polio in childhood can develop additional symptoms decades after recovering from the acute infection,[[53]](#cite_note-53) notably new muscle weakness and extreme fatigue. This condition is known as [post-polio syndrome](/wiki/Post-polio_syndrome) (PPS) or post-polio sequelae.<ref name=Cashman>[Template:Cite journal](/wiki/Template:Cite_journal)</ref> The symptoms of PPS are thought to involve a failure of the over-sized [motor units](/wiki/Motor_unit) created during the recovery phase of the paralytic disease.<ref name=Ramlow\_1992>[Template:Cite journal](/wiki/Template:Cite_journal)</ref><ref name= Annals>[Template:Cite journal](/wiki/Template:Cite_journal)</ref> Contributing factors that increase the risk of PPS include aging with loss of neuron units, the presence of a permanent residual impairment after recovery from the acute illness, and both overuse and disuse of neurons. PPS is a slow, progressive disease, and there is no specific treatment for it.[[54]](#cite_note-54) Post-polio syndrome is not an infectious process, and persons experiencing the syndrome do not shed poliovirus.[[9]](#cite_note-9)

## Epidemiology[[edit](/index.php?title=(none)&action=edit&section=18)]

|  |  |  |  |
| --- | --- | --- | --- |
| **Reported polio cases in 2015**[[3]](#cite_note-3)  [thumb|center](/wiki/File:Polio_worldwide_2015.svg) | | | |
| **Country** | **Wild cases** | **Circulating vaccine- derived cases** | **Transmission status** |
| [Template:Flag](/wiki/Template:Flag) | 52 | 2 | endemic |
| [Template:Flag](/wiki/Template:Flag) | 19 | 0 | endemic |
| [Template:Flag](/wiki/Template:Flag) | 0 | 10 | circulating vaccine- derived only |
| [Template:Flag](/wiki/Template:Flag) | 0 | 6 | circulating vaccine- derived only |
| [Template:Flag](/wiki/Template:Flag) | 0 | 4 | circulating vaccine- derived only |
| [Template:Flag](/wiki/Template:Flag) | 0 | 2 | circulating vaccine- derived only |
| [Template:Flag](/wiki/Template:Flag) | 0 | 2 | circulating vaccine- derived only |
| [Template:Flag](/wiki/Template:Flag) | 0 | 1 | circulating vaccine- derived only |
| **Total** | 71 | 27 |  |

[Template:See also](/wiki/Template:See_also)

Following the widespread use of poliovirus vaccine in the mid-1950s, the incidence of poliomyelitis declined dramatically in many industrialized countries. A global effort to [eradicate](/wiki/Eradication_of_infectious_disease) polio began in 1988, led by the [World Health Organization](/wiki/World_Health_Organization), [UNICEF](/wiki/UNICEF), and [The Rotary Foundation](/wiki/The_Rotary_Foundation).[[55]](#cite_note-55) These efforts have reduced the number of annual diagnosed cases by 99.9%; from an estimated 350,000 cases in 1988 to a low of 483 cases in 2001, after which it remained at a level of about 1,000 - 2000 cases per year for a number of years.<ref name=eradication>[Template:Cite journal](/wiki/Template:Cite_journal)</ref><ref name=morbidity>[Template:Cite journal](/wiki/Template:Cite_journal)</ref> In 2015, cases decreased to 98.[[3]](#cite_note-3)[[56]](#cite_note-56)Polio is one of only two diseases currently the subject of a global [eradication program](/wiki/Eradication_of_infectious_diseases), the other being [Guinea worm disease](/wiki/Guinea_worm_disease).[[57]](#cite_note-57) So far, the only diseases completely eradicated by humankind are [smallpox](/wiki/Smallpox), declared so, in 1980,<ref name=WHO\_smallpox>[Template:Cite web](/wiki/Template:Cite_web)</ref>[[58]](#cite_note-58) and [rinderpest](/wiki/Rinderpest), likewise, in 2011.[[59]](#cite_note-59)A number of eradication milestones have already been reached, and several regions of the world have been certified polio-free.

A concern is the presence of circulating vaccine-derived polioviruses (cVDPV). The OPV is not perfect: while the genetic characteristics are carefully balanced to maximize efficacy and minimize virulence, it is possible for the OPV to mutate. As a result, persons given the OPV can acquire acute or chronic infections; or can transmit (circulate) mutated OPV to other people. It is likely that cVDPV cases will exceed wild cases in the near future, making it desirable to discontinue use of the OPV as soon as safely possible.[[60]](#cite_note-60)

### Emergency declaration[[edit](/index.php?title=(none)&action=edit&section=19)]

In April 2012, the World Health Assembly declared the completion of polio eradication a programmatic emergency for global public health.[[61]](#cite_note-61)

### Americas[[edit](/index.php?title=(none)&action=edit&section=20)]

The [Americas](/wiki/Americas) were declared polio-free in 1994.<ref name=MMWR\_1994>[Template:Cite journal](/wiki/Template:Cite_journal)</ref>

### Western Pacific[[edit](/index.php?title=(none)&action=edit&section=21)]

In 2000 polio was declared to have been officially eliminated in 37 Western Pacific countries, including China and Australia.<ref name= Pacific>[Template:Cite journal](/wiki/Template:Cite_journal)</ref>[[62]](#cite_note-62) Despite eradication ten years prior, an outbreak was confirmed in China in September 2011 involving a strain prevalent in neighboring Pakistan.[[63]](#cite_note-63)

### Europe[[edit](/index.php?title=(none)&action=edit&section=22)]

[Europe](/wiki/Europe) was declared polio-free in 2002.<ref name=WHO\_Europe\_2002>[Template:Cite press release](/wiki/Template:Cite_press_release)</ref> On 1 September, 2015 WHO confirmed 2 cases of circulating vaccine-derived poliovirus type 1 in Ukraine.<ref name=WHO\_Europe\_2015>[Template:Cite web](/wiki/Template:Cite_web)</ref>

### South-East Asia[[edit](/index.php?title=(none)&action=edit&section=23)]

The last case of polio in the region was in India in January 2011.[[64]](#cite_note-64) Since January 2011, there have been no reported cases of the wild polio infections in India, and in February 2012 the country was taken off the WHO list of polio endemic countries. It was reported that if there are no cases of wild polio in the country for two more years, it would be declared as a polio-free country.[[65]](#cite_note-65)[[66]](#cite_note-66) On March 27, 2014 the WHO announced the eradication of poliomyelitis in the South-East Asia Region, which includes eleven countries: [Bangladesh](/wiki/Bangladesh), [Bhutan](/wiki/Bhutan), [North Korea](/wiki/North_Korea), [India](/wiki/India), [Indonesia](/wiki/Indonesia), [Maldives](/wiki/Maldives), [Myanmar](/wiki/Myanmar), [Nepal](/wiki/Nepal), [Sri Lanka](/wiki/Sri_Lanka), [Thailand](/wiki/Thailand) and [Timor-Leste](/wiki/Timor-Leste).[[67]](#cite_note-67) With the addition of this region, 80 per cent of the world population lives in polio-free regions.[[67]](#cite_note-67)

### Syria[[edit](/index.php?title=(none)&action=edit&section=24)]

In 2015, polio was believed to remain [naturally spreading](/wiki/Endemic_(epidemiology)) in only two countries: [Pakistan](/wiki/Pakistan), and [Afghanistan](/wiki/Afghanistan),<ref name=eradication1/><ref name=GlobalPolio2015>[Template:Cite web](/wiki/Template:Cite_web)</ref>[[68]](#cite_note-68)[[69]](#cite_note-69) although it continued to cause epidemics in other nearby countries due to hidden or reestablished transmission.[[70]](#cite_note-70) In May 2014, the World Health Organization declared polio's renewed spread a [world health emergency](/wiki/PHEIC).[[74]](#cite_note-74)[[75]](#cite_note-75) A vaccination campaign in Syria operated literally under fire and lead to the deaths of several vaccinators,[[76]](#cite_note-76) but returned vaccination coverage to pre-war levels.[[77]](#cite_note-77) No new cases have been reported since January 2014; vaccination continues, and Iraq is also being closely monitored.[[78]](#cite_note-78)[[79]](#cite_note-79)

### Africa[[edit](/index.php?title=(none)&action=edit&section=25)]

In 2003 in [northern Nigeria](/wiki/Northern_Region,_Nigeria)—a country which at that time was considered provisionally polio free—a [fatwa](/wiki/Fatwa) was issued declaring that the polio vaccine was designed to render children sterile.[[80]](#cite_note-80) Subsequently, polio reappeared in Nigeria and spread from there to several other countries. In 2013, nine health workers administering polio vaccine were targeted and killed by gunmen on motorcycles in [Kano](/wiki/Kano), but this was the first[[81]](#cite_note-81) and last attack.[[82]](#cite_note-82) Local traditional and religious leaders and polio survivors worked to revive the campaign,[[83]](#cite_note-83) and Nigeria has not had a polio case since July 24, 2014. In September 2015, Nigeria is removed from polio-endemic list. If no new cases appear, it will be declared polio-free in 2017.[[84]](#cite_note-84) In 2013 the Center for Disease Control received reports of 183 cases of polio in [Somalia](/wiki/Somalia), 14 in [Kenya](/wiki/Kenya) and 8 cases in the [Somali Region](/wiki/Somali_Region) of Ethiopia,<ref name=cdcp>[Template:Cite web](/wiki/Template:Cite_web)</ref> but Africa had no confirmed cases of wild poliovirus (WPV) in 2015.<ref name = GlobalPolio2015/> A case of circulating vaccine-derived poliovirus (cVDPV) type 2 was detected in [Siguiri](/wiki/Siguiri) in [Guinea's](/wiki/Guinea) [Kankan](/wiki/Kankan) region, in August 2014.[[85]](#cite_note-85) In September 2015, a case of cVDPV in Mali was confirmed by the WHO.[[85]](#cite_note-85)

### Afghanistan and Pakistan[[edit](/index.php?title=(none)&action=edit&section=26)]

This is the last remaining region with wild polio cases. Both major sides of the Afghan civil war support polio vaccination[[86]](#cite_note-86) and polio rates are declining rapidly in Afghanistan, with only 19 cases in 2015.[[83]](#cite_note-83) In [Pakistan](/wiki/Pakistan) there were 53 cases in 2015, the highest number for any country.[[83]](#cite_note-83) Vaccination in Pakistan is hindered by conflict and organizational problems. The militant [Pakistani Taliban](/wiki/Tehrik-i-Taliban_Pakistan) claims vaccination is a Western plot to [sterilise](/wiki/Sterilization_(medicine)) local children.<ref name=BBC2016/> 66 vaccinators were killed in 2013 and 2014.[[87]](#cite_note-87)[[88]](#cite_note-88) Cases have dropped by 70% in 2015; reasons include Dh440 million support from the [United Arab Emirates](/wiki/United_Arab_Emirates) to vaccinate more than ten million children,[[88]](#cite_note-88)[[89]](#cite_note-89) changes in the military situation, and arrests of some of those who attacked polio workers.<ref name = BBC2016>[Template:Cite web](/wiki/Template:Cite_web)</ref>[[90]](#cite_note-90)

## History[[edit](/index.php?title=(none)&action=edit&section=27)]

[Template:Main](/wiki/Template:Main) [thumb|left|An](/wiki/File:Polio_Egyptian_Stele.jpg) [Egyptian](/wiki/Egypt) [stele](/wiki/Stele) thought to represent a polio victim, [18th Dynasty](/wiki/18th_Dynasty) (1403–1365 BC)

The effects of polio have been known since [prehistory](/wiki/Prehistory); [Egyptian](/wiki/Ancient_Egypt) paintings and carvings depict otherwise healthy people with withered limbs, and children walking with canes at a young age.<ref name=Paul\_1971>[Template:Cite book](/wiki/Template:Cite_book)</ref> The first clinical description was provided by the English physician [Michael Underwood](/wiki/Michael_Underwood_(physician)) in 1789, where he refers to polio as "a debility of the lower extremities".<ref name=Underwood\_1789>[Template:Cite book](/wiki/Template:Cite_book)</ref> The work of physicians [Jakob Heine](/wiki/Jakob_Heine) in 1840 and [Karl Oskar Medin](/wiki/Karl_Oskar_Medin) in 1890 led to it being known as *Heine–Medin disease*.<ref name=Pearce\_2005>[Template:Cite journal](/wiki/Template:Cite_journal)</ref> The disease was later called *infantile paralysis*, based on its propensity to affect children.

Before the 20th century, polio infections were rarely seen in infants before six months of age, most cases occurring in children six months to four years of age. Poorer [sanitation](/wiki/Sanitation) of the time resulted in a constant exposure to the virus, which enhanced a natural [immunity](/wiki/Immunity_(medical)) within the population. In developed countries during the late 19th and early 20th centuries, improvements were made in community sanitation, including better [sewage](/wiki/Sewage) disposal and clean water supplies. These changes drastically increased the proportion of children and adults at risk of paralytic polio infection, by reducing childhood exposure and immunity to the disease.[[91]](#cite_note-91) Small localized paralytic polio [epidemics](/wiki/Epidemic) began to appear in Europe and the United States around 1900.<ref name = Trevelyan>[Template:Cite journal](/wiki/Template:Cite_journal)</ref> Outbreaks reached [pandemic](/wiki/Pandemic) proportions in Europe, North America, Australia, and New Zealand during the first half of the 20th century. By 1950 the peak age incidence of paralytic poliomyelitis in the United States had shifted from infants to children aged five to nine years, when the risk of paralysis is greater; about one-third of the cases were reported in persons over 15 years of age.<ref name=Melnick\_1990>[Template:Cite book](/wiki/Template:Cite_book)</ref> Accordingly, the rate of paralysis and death due to polio infection also increased during this time.<ref name = Trevelyan/> In the United States, the 1952 polio epidemic became the worst outbreak in the nation's history. Of nearly 58,000 cases reported that year 3,145 died and 21,269 were left with mild to disabling paralysis.[[92]](#cite_note-92) [Intensive care medicine](/wiki/Intensive_care_medicine) has its origin in the fight against polio.[[93]](#cite_note-93) Most hospitals in the 1950s had limited access to [iron lungs](/wiki/Negative_pressure_ventilator) for patients unable to breathe without mechanical assistance. Respiratory centers designed to assist the most severe polio patients, first established in 1952 at the Blegdam Hospital of [Copenhagen](/wiki/Copenhagen) by [Danish](/wiki/Denmark) [anesthesiologist](/wiki/Anesthesia) [Bjørn Ibsen](/wiki/Bjørn_Aage_Ibsen), were the harbingers of subsequent [intensive care units](/wiki/Intensive_care_unit) (ICU). (A year later, Ibsen would establish the world's first dedicated ICU.)[[94]](#cite_note-94) The polio epidemics not only altered the lives of those who survived them, but also brought profound cultural changes, spurring [grassroots](/wiki/Grassroots) fund-raising campaigns that would revolutionize medical [philanthropy](/wiki/Philanthropy), and giving rise to the modern field of [rehabilitation therapy](/wiki/Physical_therapy). As one of the largest disabled groups in the world, polio survivors also helped to advance the modern [disability rights movement](/wiki/Disability_rights_movement) through campaigns for the social and civil rights of the [disabled](/wiki/Disabled). The World Health Organization estimates that there are 10 to 20 million polio survivors worldwide.<ref name= NewsDesk>[Template:Cite web](/wiki/Template:Cite_web)</ref> In 1977 there were 254,000 persons living in the United States who had been paralyzed by polio.[[95]](#cite_note-95) According to doctors and local polio support groups, some 40,000 polio survivors with varying degrees of paralysis live in Germany, 30,000 in Japan, 24,000 in France, 16,000 in Australia, 12,000 in Canada and 12,000 in the United Kingdom.<ref name= NewsDesk/> Many [notable individuals have survived polio](/wiki/List_of_polio_survivors) and often credit the prolonged immobility and residual paralysis associated with polio as a driving force in their lives and careers.[[96]](#cite_note-96) The disease was very well publicized during the polio epidemics of the 1950s, with extensive media coverage of any scientific advancements that might lead to a cure. Thus, the scientists working on polio became some of the most famous of the century. Fifteen scientists and two laymen who made important contributions to the knowledge and treatment of poliomyelitis are honored by the [Polio Hall of Fame](/wiki/Polio_Hall_of_Fame), which was dedicated in 1957 at the [Roosevelt Warm Springs Institute for Rehabilitation](/wiki/Roosevelt_Warm_Springs_Institute_for_Rehabilitation) in [Warm Springs, Georgia](/wiki/Warm_Springs,_Georgia), US. In 2008 four organizations (Rotary International, the World Health Organization, the U.S. Centers for Disease Control and UNICEF) were added to the Hall of Fame.[[97]](#cite_note-97)[[98]](#cite_note-98) World Polio Day (24 October) was established by [Rotary International](/wiki/Rotary_International) to commemorate the birth of [Jonas Salk](/wiki/Jonas_Salk), who led the first team to develop a vaccine against poliomyelitis. Use of this inactivated poliovirus vaccine and subsequent widespread use of the oral poliovirus vaccine developed by [Albert Sabin](/wiki/Albert_Sabin) led to establishment of the Global Polio Eradication Initiative (GPEI) in 1988. Since then, GPEI has reduced polio worldwide by 99%.[[99]](#cite_note-99)

### Etymology[[edit](/index.php?title=(none)&action=edit&section=28)]

The term derives from the [Ancient Greek](/wiki/Ancient_Greek) [Template:Transl](/wiki/Template:Transl) ([Template:Lang](/wiki/Template:Lang)), meaning "grey", [Template:Transl](/wiki/Template:Transl) ([Template:Lang](/wiki/Template:Lang) “marrow”), referring to the grey matter of the [spinal cord](/wiki/Spinal_cord), and the suffix [*-itis*](/wiki/Wikt:-itis), which denotes [inflammation](/wiki/Inflammation).,<ref name=Chamberlin\_2005>[Template:Cite book](/wiki/Template:Cite_book)</ref> i.e., inflammation of the spinal cord’s grey matter, although a severe infection can extend into the brainstem and even higher structures, resulting in [polio*encephal*itis](/wiki/Polioencephalitis), producing a [lack of ability to breathe](/wiki/Apnea) that requires mechanical assistance such as an [iron lung](/wiki/Negative_pressure_ventilator).

## Society and culture[[edit](/index.php?title=(none)&action=edit&section=29)]

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## References[[edit](/index.php?title=(none)&action=edit&section=30)]

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## Further reading[[edit](/index.php?title=(none)&action=edit&section=31)]

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