Interruption of Poliovirus Transmission in Ghana: Molecular Epidemiology of Wild-Type 1 Poliovirus Isolated from 1995 to 2008

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Described in detail is the molecular epidemiology of wild-type 1 poliovirus circulation in Ghana between 1995–2008, following the implementation of a surveillance system for cases of acute flaccid paralysis and poliovirus infection. Molecular phylogenetic analysis combined with a detailed evaluation of epidemiological indicators revealed that the geographical and temporal circulation of wild-type poliovirus in Ghana was determined by the quality of the implementation of global eradication strategies. The transmission of “indigenous” wild-type 1 poliovirus was eliminated in 1999. However, a drastic reduction in national immunization campaigns resulted in the importation in 2003 and 2008 of wild-type 1 poliovirus from neighboring countries. Both outbreaks were promptly interrupted following resumption of immunization activities. The results detailed here provide scientific evidence that supports the feasibility of polio eradication in Central West Africa, one of the remaining endemic areas for the disease, provided that comprehensive immunization campaigns and sensitive surveillance systems are in place.

The Global Polio Eradication Initiative (GPEI) followed a World Health Assembly resolution in 1988 with the aim of eradicating paralytic poliomyelitis by the year 2000 [1–3]. The main strategies for the eradication program are the use of extensive immunization campaigns with live-attenuated oral polio vaccine (OPV) and the surveillance of acute flaccid paralysis (AFP), the main clinical sign for the disease. Although the year 2000 target was not met, considerable progress has been made towards interrupting the transmission of wild-type poliovirus globally, resulting in a reduction of more than 99% in the incidence of poliomyelitis worldwide and a decrease in the number of polio-endemic countries from 125 in 1988 to 4 in 2010 [4]. Poliovirus, the causative agent of paralytic poliomyelitis, is a human enterovirus (HEV) of species C belonging to the family Picornaviridae, a group of nonenveloped, positive-strand, RNA viruses, and exists as 3 different serotypes – 1, 2 and 3. Wild-type 2 has been eliminated globally and was last seen in India in 1999. Three of the 6 WHO regions have been certified free of indigenous wild-type poliovirus: the American Region in 1991, the Western Pacific Region in 1997, and the European Region in 2002 [3, 5, 6]. The remaining 3 endemic regions – Eastern Mediterranean, South East Asia and Africa – are also progressing towards polio eradication.

The Expanded Programme on Immunization (EPI) was introduced in Ghana in 1978, and has been operational in all regions of the country since 1985 [7]. The GPEI program was formally introduced in 1996 and intensified routine polio immunization, implemented supplementary immunization activities (SIAs), and introduced active AFP surveillance for poliovirus with full laboratory support [8, 9]. A total of 37 wild-type 1 polioviruses were isolated in Ghana from AFP cases between 1995 and the end...
of 1999, at which time it was considered that circulation of wild-type poliovirus had been interrupted. However, after almost 3-1/2 years without a polio case, 8 wild-type 1 poliovirus isolates were identified in 2003 from AFP samples. In 2003, 15 isolates were also obtained from a survey that involved 100 children in an area known to have poor sanitation and a low vaccination coverage. In addition, following another long period of >5 years without polio cases, 9 more wild-type 1 poliovirus isolates were identified in 2008 from AFP cases. No wild-type poliovirus isolates have been found in Ghana since November 2008.

This study describes the molecular analysis of wild-type 1 poliovirus strains isolated in Ghana between 1995 and 2008. VP1 nucleotide sequences were used to explore the phylogenetic relationship between the Ghana strains and wild-type 1 polioviruses isolated in other parts of the world. The occurrence of variations in key epidemiological and surveillance quality indicators during this period was also analyzed. The data were used to identify possible geographical and temporal transmission pathways, to detect importation events, and to assess the impact of vaccination and AFP surveillance on poliovirus circulation during the period of investigation. In addition, partial nucleotide sequences were determined in the 5’ and 3’ regions of the genome to ascertain whether genomic recombination between poliovirus and other species of C HEVs was a common event during poliovirus transmission and evolution in Ghana.

The results shown here represent the first comprehensive study that covers the period between the establishment of AFP surveillance and supplementary immunization activities and the complete interruption of wild-type poliovirus circulation in a country endemic for polio, studying both the molecular properties of poliovirus isolates and the quality of the implementation of polio eradication strategies.

METHODS

Virus Isolation and Typing
Fecal specimens from suspected AFP cases were processed at the National Polio Laboratory in Ghana according to standard protocols for virus isolation and characterization [8].

Reverse Transcription, PCR and Nucleotide Sequencing of Poliovirus Genomes
Poliovirus RNA was purified from cell culture supernatants and used for reverse transcription and PCR reactions using standard procedures. The nucleotide sequencing analysis covered the 5’NCR (nt 68–530), the entire capsid protein VP1 coding region (nt 2479–3479), and a fragment of the non-structural coding region (nt 5844–6473), which included part of the 3C and 3D genes. Viral cDNA sequences were determined using a Big-Dye Terminator Cycle Sequencing Ready Reaction Kit with an ABI Prism DNA 377 Sequencer as specified by the manufacturer. The nucleotide sequence data reported in this article are available from the EMBL Data Library under accession numbers JN393014 to JN393205.

Sequence Analysis
Phylogenetic relationships between strains were established by comparing the sequences determined and aligning them using the alignment program CLUSTAL X [10]. The degree of nucleotide sequence identity and of protein similarity between strains was determined using the default scoring matrices. Phylogenetic relationships between sequences were inferred by the maximum likelihood method with DNADIST/NEIGHBOR of PHYLIP (Phylogeny Inference Package) version 3.6 software [11]. The robustness of phylogenies was estimated by bootstrap analyses with 1000 pseudoreplicate data sets generated with the SEQBOOT program of PHYLIP. Phylogenetic trees were constructed using the neighbor-joining feature of PHYLIP and drawn using TREEVIEW software [12]. The MEGA 4.0 software package was used to perform most of these analyses [13].

Epidemiological Analysis
Data from official national records, laboratory books, and computer databases were used for these analyses.

RESULTS

Epidemiology of Poliomyelitis in Ghana
Ghana uses OPV supplied by UNICEF and WHO for all polio immunizations. Routine OPV3 (4 doses) coverage was initiated at approximately 50% in 1996 and rose to 80% in 2000, 85% in 2005 and 92% in 2008, and has since remained stable at that rate [14]. SIAs were instituted in Ghana in 1996 and included national and sub-national immunization days (NIDs and sub-NIDs) during which large numbers of children less than 5 years of age were vaccinated with OPV on the same day (Figure 1). Two relatively long periods during which no NIDs or incomplete NIDs were performed occurred between November 2001 and October 2003 and between November 2006 and February 2009, respectively (Figure 1).

Table 1 describes the evolution of the most representative AFP performance indicators in Ghana during the period of study. As shown, the levels of performance were low during the initial stages of the program, particularly in regard to the adequacy of sample collection from AFP cases. By the year 2000, adequate levels had been reached for most AFP performance indicators, which have been maintained to ensure an efficient surveillance for poliovirus. AFP surveillance was first established in the more developed southern regions where a good healthcare system existed, and was later introduced in the poorer northern areas of the country. The 4 most
Figure 1. Timeline of wild-type 1 poliovirus isolations in Ghana between 1995–2008. The number of polio cases (left axis scale) are indicated by light grey (genotype I), dark grey (genotype II), and white (genotype III) columns. Black columns indicate unclassified isolates. Triangles indicate the number of children immunized in NIDs or sub-NIDs (right axis scale). Arrows indicate gaps in immunization. Routine OPV coverage for 2002, 2003, 2007, and 2008 was 99%, 80%, 94% and 92%, respectively [14].

Table 1. AFP Surveillance Quality Indicators in Ghana between 1995 and 2008 (%)

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<tr>
<td>Number of AFP cases reported</td>
<td>...</td>
<td>...</td>
<td>23</td>
<td>77</td>
<td>170</td>
<td>78</td>
<td>316</td>
<td>278</td>
<td>136</td>
<td>199</td>
<td>163</td>
<td>173</td>
<td>174</td>
<td>165</td>
<td>260</td>
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<tr>
<td>Annualized non-polio AFP rate /100 000 &lt;15 y population</td>
<td>1</td>
<td>...</td>
<td>0.20</td>
<td>0.49</td>
<td>0.50</td>
<td>1.40</td>
<td>1.90</td>
<td>2.80</td>
<td>2.70</td>
<td>2.00</td>
<td>1.7</td>
<td>1.76</td>
<td>1.65</td>
<td>1.6</td>
<td>1.92</td>
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<td>Proportion of AFP cases with 2 stool specimens collected within 14 d of onset of paralysis (%)</td>
<td>80</td>
<td>...</td>
<td>43.5</td>
<td>22.1</td>
<td>30</td>
<td>50</td>
<td>47</td>
<td>64.6</td>
<td>86.3</td>
<td>77.1</td>
<td>83.3</td>
<td>76.7</td>
<td>78.6</td>
<td>78.6</td>
<td>77.1</td>
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<td>Proportion of stool specimens arriving at the national level within 3 d of being sent (%)</td>
<td>80</td>
<td>...</td>
<td>...</td>
<td>92</td>
<td>90.7</td>
<td>95.7</td>
<td>92.9</td>
<td>97</td>
<td>87</td>
<td>81.2</td>
<td>96.4</td>
<td>95.7</td>
<td>91.0</td>
<td>87.9</td>
<td></td>
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<tr>
<td>Proportion of stool specimens arriving at the lab in good condition (%)</td>
<td>90</td>
<td>80</td>
<td>87</td>
<td>71.4</td>
<td>97.1</td>
<td>97.4</td>
<td>92.4</td>
<td>97.5</td>
<td>92.6</td>
<td>96.4</td>
<td>72.4</td>
<td>85.9</td>
<td>90.7</td>
<td>86.0</td>
<td>89.0</td>
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<td>Proportion of stool specimens for which lab results were sent within 28 d of receipt at the lab (%)</td>
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<td>100</td>
<td>...</td>
<td>...</td>
<td>76.5</td>
<td>85.1</td>
<td>95.6</td>
<td>98.9</td>
<td>100</td>
<td>78.4</td>
<td>97.8</td>
<td>99.4</td>
<td>99.4</td>
<td>84.0</td>
<td>98.3</td>
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<tr>
<td>Proportion of stool specimens from which non-polio enterovirus was isolated (%)</td>
<td>10</td>
<td>0</td>
<td>4.3</td>
<td>5.2</td>
<td>4.5</td>
<td>4.2</td>
<td>13.3</td>
<td>14</td>
<td>16.9</td>
<td>12.6</td>
<td>11.9</td>
<td>14.5</td>
<td>9.8</td>
<td>8.0</td>
<td>7.9</td>
</tr>
<tr>
<td>Number of wild poliovirus</td>
<td>...</td>
<td>2</td>
<td>9</td>
<td>2</td>
<td>21</td>
<td>3</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>8</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>8</td>
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<tr>
<td>Number of Sabin virus</td>
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<td>0</td>
<td>0</td>
<td>4</td>
<td>4</td>
<td>18</td>
<td>13</td>
<td>7</td>
<td>20</td>
<td>9</td>
<td>10</td>
<td>1</td>
<td>5</td>
<td>14</td>
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northerly regions, Brong Ahafo, Northern, Upper West, and Upper East, account for 28% of the total population in Ghana but only produced 13% and 5.2% of the total AFP cases in 1996 and 1997, respectively. From 1998 onward, however, the situation improved and the numbers of AFP cases were more evenly distributed geographically. Consequently, most of the wild-type polioviruses isolated in Ghana during 1995–1997 (92.3%) came from southern districts (Figure 2).

Isolation of Poliovirus
The National Polio Laboratory processed approximately 4000 stool specimens between 1996 and 2008 that were collected from AFP cases through the active surveillance system in Ghana.

Testing revealed 56 wild-type 1 and 99 Sabin-like polioviruses, together with 234 non-polio enteroviruses. Furthermore, from 1997 to 2003, approximately 2500 samples were collected from healthy children, and 15 wild-type 1 and 65 Sabin-like polioviruses, as well as 314 non-polio enteroviruses, were found among these samples.

Details of all wild-type poliovirus isolations from 1995 to 2008 in Ghana, including their geographical distribution, are given in Tables 2 and 3 and Figures 1 and 2. The genotype was determined by analysis of the nucleotide sequence in the VP1 coding region (see the Results

As shown in Figure 1, the polio cases appeared to follow a seasonal distribution with a peak between October and December, just after the rainy season, apart from the 2003 cases that occurred before the expected seasonal peak and did not follow the normal pattern.
A total of 63 wild-type 1 polioviruses were characterized. They represent 91.3% (63 of 69) of the total number of wild-type 1 viruses isolated in Ghana during the period of study. The 6 remaining isolates were either no longer present in the laboratory when this study was initiated or failed to grow in tissue culture. However, VP1 sequence data for 4 of these isolates were available and kindly provided by the CDC (Table 2).

Two poliovirus strains were isolated from AFP case No. 64. No wild-type 2 or 3 polioviruses were isolated in Ghana during 1995–2008.

As shown in Figure 3A, the Ghanaian wild-type 1 poliovirus isolates from 1995–2008 were grouped into 3 different genotypes, as judged by a comparison of VP1 nucleotide sequences. The groups were named I, II, and III for the purpose of this study. The 2 viruses from 1995 were relatively close to each other (2.59% sequence difference in VP1) but were distant from isolates of genotype I from subsequent years (between 8.58%–10.52% VP1 nucleotide differences). The other genotype I isolates grouped closely together (average VP1 sequence difference of 3.46%) (Figure 3A). The genotype I poliovirus isolates from 1996 showed an average VP1 sequence difference of 2.59%.
Figure 3. Neighbor-joining trees representing phylogenetic analyses of wild-type 1 poliovirus strains isolated in Ghana between 1995–2008. (A) Relationship between the 1995–2008 poliovirus isolates from Ghana. VP1 coding region (nucleotides 2480–3385). (B) Relationship between selected type 1 poliovirus isolates from Ghana (those with GHA in their name) and other type 1 poliovirus isolates from around the world. VP1 coding region (nucleotides 2480–3385). EMBL Data Library accession numbers for VP1 sequences are shown in the tree. The positions of isolates from West Africa genotypes A, B, and C (WEAF-A, WEAF-B, and WEAF-C, respectively) are indicated in the tree. (C) Relationship between the 1995–2008 poliovirus isolates from Ghana. 5′NCR (nucleotides 68–537). (D) Relationship between the 1995–2008 poliovirus isolates from Ghana. Partial 3CD coding region (nucleotides 5846–6478). Numbers at nodes indicate the percentage of 1000 bootstrap pseudoreplicates supporting the cluster. The sequences of PV1/Mahoney and PV2/Sabin 2 reference strains were introduced for correct rooting of the tree.
sequence difference between them of 3.19%, while the average
difference between genotype I isolates from 1998 was only
1.12%. Genotype II included viruses from 1998 and 1999
(Figure 3A), which were all very closely related and showed an
average 1.40% nucleotide difference between them in the VP1
sequence. All isolates from healthy and AFP cases from 2003
were phylogenetically close to each other (the average VP1 se-
quence difference between them was 1.32%) and belonged to a
third genotype, genotype III, with very different VP1 se-
quencies from viruses of genotypes I and II (17.92% and
20.44% VP1 nucleotide differences, respectively) (Figure 3A).
Isolates from 2008 also belonged to genotype III and had an
8.39% average VP1 sequence difference from 2003 isolates and a
0.85% VP1 sequence difference between themselves (Figure 3A).
Their VP1 nucleotide sequences were 18.15% and
20.01% different from viruses of genotypes I and II, respec-
tively. Two poliovirus strains with 6 nucleotide differences
between them in the VP1 coding region were isolated from
case No. 64 in 2008 (Table 3).

Representatives of the 3 genotypes identified above were se-
lected and their VP1 sequences compared to other wild-type 1
poliovirus VP1 sequences available in public databases. The
Ghanaian viruses from genotype I and II were classified
within the West Africa A (WEAF-A) and West Africa C
(WEAF-C) genotypes, respectively, which circulated in the
African region in the late 1990s and early 2000s (Figure 3B).
Viruses related to Ghanaian genotype III isolates seemed to
have been well established in Central and West Africa well
before the 2003 outbreak in Ghana, and the Ghanaian 2003
and 2008 strains were found to be closely related to several
African isolates from 1999 to 2009. They all belong to the
West Africa B (WEAF-B) genotype, which is still currently
dominant in West and Central Africa. The Ghanaian isolates
had no close genetic relationship with other wild-type 1
viruses identified in other parts of the world for which nucleo-
tide sequences are publicly available.

Recombination among Wild-Type 1 Polioviruses from Ghana
Four major genetic groups were found when the genetic rela-
tionships among isolates in the 5’NCR were analyzed
(Figure 3C). Two isolates from 1995 segregated as an indepen-
dent cluster from their genotype I counterparts and showed
large genetic distances with respect to the rest of the viruses
from genotype I and strains from genotypes II and III.

As shown in Figure 3D, the phylogenetic tree constructed
with 3CD sequences had a rather different topology. Five
major genetic clusters can be clearly seen, with viruses from
genotype I splitting into 2 separate clusters consisting of 1 that
included most isolates from 1996 and 1997 and the other
comprising all isolates from 1998. Viruses from genotype III
also split into 2 separate clusters, with 1 including most iso-
lates from 2003 and the other consisting of the majority of
virus from 2008. All strains from genotype II segregated as a
separate group. Nine isolates, 5 from genotype I and 4 from
genotype III (3 from 2003 and 1 from 2008), showed as single
separate branches that could not be assigned to any of the 5
genetic clusters. There was low genetic similarity between clus-
ters and viruses in single branches.

Amino Acid Substitutions among Ghanaian Poliovirus Isolates
The Ghanaian wild-type 1 isolates shared 65.6% of VP1 nucleo-
tides and had 258 (85.4%) common amino acids. The se-
quence similarity in the VP1 coding region was significantly
higher among isolates within a genotype. Viruses from geno-
types I, II and III showed 80.5%, 92.9% and 82.3% VP1 nucleo-
tide identity and 93.4%, 97.0% and 93.7% amino acid
identity, respectively. Relevant mutations were identified at or
close to outer surface amino acids that form the neutralizing
antigenic sites [15]. VP1-96, VP1-252 and VP1-257 in site 1,
and VP1-215, 221 and 223 in antigenic site 2. The combina-
tion of mutations at these positions in the Ghanaian isolates
was different for each genotype, with minor differences ap-
pearing between the 2 strains from 1995 and the rest of the
WEAF-A isolates and between the 2003 and the 2008 WEAF-
B strains. Remarkably, among these amino acids, only residue
VP1-252 was different between the 3 serotypes, a Thr in geno-
type WEAF-A viruses, a Lys in genotype WEAF-B strains, and
an Ile in WEAF-C isolates.

DISCUSSION
The interruption of the circulation of wild-type 1 and 3 polio-
viruses in the last few endemic areas in Central Western Africa,
Northern India, Afghanistan, and Pakistan is proving to be
difficult, as there have been approximately 1500 polio cases
since 2005 [4]. An important number of polio cases are due to importations from endemic areas into countries
that had been polio-free [16].

In this report, we assessed the effectiveness of GPEI strate-
gies used during the process of interruption of wild-type po-
livirus transmission in Ghana, a country located in Central
Western Africa. The molecular properties of 63 wild-type 1
polioviruses isolated in Ghana during the period 1995–2008
were analyzed in the context of key epidemiological factors
such as the coverage and chronology of immunization cam-
paigns, the evolution of the quality indicators for AFP surveil-
lance, and the adequacy of field and laboratory investigation
of AFP cases.

Nucleotide sequence comparisons in the capsid VP1 coding
region identified 3 different genotypes among the Ghanaian
isolates. The viruses were related to African genotypes WEAF-
A, -B and -C, which are the 3 genotypes that have circulated
in Central and West Africa since the early 1990s [17, 18]. Phy-
logenetic analyses of virus sequences that incorporated
epidemiological data indicated that wild-type 1 polioviruses of genotype WEAF-A were circulating widely in Ghana when AFP surveillance started in 1995–1996. They were first detected in southern areas during 1995–1997 and were not found in northern districts until 1998. However, it is likely that genotype WEAF-A polioviruses were also present in northern areas before 1998 but not detected due to poor surveillance. A rapid improvement in AFP surveillance and effective immunization campaigns helped to quickly eliminate genotype WEAF-A from the southern provinces, and viruses of this genotype were not detected here from May 1997 onwards. Similarly, viruses from genotype WEAF-C detected in Ghana from July 1998 had a much lesser impact in the southern provinces. There is no evidence of its presence in Ghana prior to July 1998. The short-term distribution of viruses from this genotype, together with the fact that all strains showed a very close genetic relationship in all of the 3 genetic regions that were analyzed, would suggest that the isolates represent a single infection event, possibly as a result of importation from a neighboring country.

Both AFP surveillance and immunization rates reached high standards in the entire country soon after the 1998 epidemic, and only 4 viruses were detected in Ghana in 1999. However, wild-type 1 polioviruses were again found in Ghana in 2 separate outbreaks in 2003 and 2008 after long periods without polio cases. All 2003 and 2008 viruses belonged to a third genotype, WEAF-B, and were closely related to contemporary viruses found in Central and West Africa (Figure 3B). The Ghanaian isolates from 2003 and 2008 showed a very close genetic relationship within the same year, as determined by the length of horizontal branches joining any 2 isolates in the VP1 phylogenetic tree, which suggested that there were no significant gaps in surveillance. However, possible gaps in surveillance were identified between 1995 and 1997. The 2 only isolates from 1997 were very distant from each other despite being collected on the same day and the same region. Virus GHA-ASH-AME-97-001 formed a separate branch within viruses of genotype WEAF-A, distant from 1996 and 1998 viruses. The situation improved after 1997, and 1998 viruses from both circulating genotypes WEAF-A and WEAF-C showed close genetic distances within each group despite known deficiencies in the timely collection of some AFP samples.

It is likely that a decrease in population immunity against poliovirus due to reduced immunization campaigns played a significant role in the generation of both the 2003 and 2008 outbreaks. Due to global programatic priorities and a vaccine shortage, only 1 sub-NID was carried out in Ghana in October 2002 and another in March 2003 when approximately 2 and 0.8 million children, representing 44% and 17% of the target children, respectively, were immunized. Similarly, only a single NID was conducted in Ghana in 2006 (November) that covered only 85% of the target group, while no NID was carried out in 2007. The most likely origin of both the 2003 and 2008 outbreaks in Ghana was Nigeria, where significant deficiencies in immunization and surveillance activities had been ongoing since 2002 and were responsible for large outbreaks that occurred between 2003 and 2008 [19–21]. Because of the ongoing deficiencies in routine vaccination services in many countries of the region, wild-type 1 poliovirus spread into previously polio-free countries, followed by intercontinental spread to the Middle East and Asia [22].

Sequencing analyses in the 5′ and 3′ regions of the genome (Figure 3C and 3D) indicated that frequent recombination events had likely occurred during the natural history of the Ghanaian polioviruses, which confirmed previous observations in other settings [23–25]. New non-structural genomic sequences were incorporated into genotype WEAF-A viruses at some time between August 1995 and September 1996 and at some time between February 1997 and June 1998, giving rise to 2 major genetic clusters among genotype WEAF-A isolates. Similarly, sometime between September 2003 and September 2008, novel non-structural sequences were acquired by genotype WEAF-B viruses. As a consequence of these recombination events, comparison of nucleotide sequences in different regions of the genome can provide different levels of resolution to help establish links between poliovirus isolates in different situations.

The results from Ghana clearly showed that eradication of poliovirus is achievable if there is an efficient AFP surveillance system in place, as well as the implementation of high-quality, rapid, and efficient SIA immunization campaigns. Our results illustrate the impact of AFP surveillance during the early years of polio eradication efforts in Ghana, the importance of national immunization days, and mop-up campaigns to eliminate wild-type poliovirus circulation and the risks of importation of wild-type poliovirus from other countries in the absence of high-population immunity. Overall, the elimination of poliovirus circulation in Ghana can be considered a success, even in a context where neighboring countries have maintained or re-established wild-type poliovirus endemicity, and would suggest that changing from a polio eradication effort to a control strategy [26] is not optimal, particularly in areas where maintaining high levels of routine immunization remains a challenge and GPEI strategies appear to be the best means to fight and eliminate this deadly disease. The use of improved GPEI strategies such as the new monovalent OPV and an advanced poliovirus laboratory isolation algorithm has recently contributed to a significant reduction in polio cases in Nigeria and India, with only 21 and 42 confirmed polio cases in 2010, respectively [4]. There were 62 polio cases that were reported in Nigeria in 2011 [27], and remarkably, as of March 2012, no polio cases have been reported in India for more than 1 year [28, 29]. This means that the country is no longer considered endemic, and only 3 countries in the world
remain in which indigenous wild poliovirus transmission has never been interrupted.

Notes

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Potential conflicts of interest. All authors: No reported conflicts.

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