Effectiveness of Immunization against Paralytic Poliomyelitis in Nigeria

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ABSTRACT

BACKGROUND
The number of cases of paralytic poliomyelitis has declined in Nigeria since the introduction of newly licensed monovalent oral poliovirus vaccines and new techniques of vaccine delivery. Understanding the relative contribution of these vaccines and the improved coverage to the decline in incident cases is essential for future planning.

METHODS
We estimated the field efficacies of monovalent type 1 oral poliovirus vaccine and trivalent oral poliovirus vaccine, using the reported number of doses received by people with poliomyelitis and by matched controls as identified in Nigeria’s national surveillance database, in which 27,379 cases of acute flaccid paralysis were recorded between 2001 and 2007. Our estimates of vaccine coverage and vaccine-induced immunity were based on the number of doses received by children listed in the database who had paralysis that was not caused by poliovirus.

RESULTS
The estimated efficacies per dose of monovalent type 1 oral poliovirus vaccine and trivalent oral poliovirus vaccine against type 1 paralytic poliomyelitis were 67% (95% confidence interval [CI], 39 to 82) and 16% (95% CI, 10 to 21), respectively, and the estimated efficacy per dose of trivalent oral poliovirus vaccine against type 3 paralytic poliomyelitis was 18% (95% CI, 9 to 26). In the northwestern region of Nigeria, which reported the majority of cases during the study period, coverage with at least one dose of vaccine increased from 59 to 78%. Between 2005 and 2007, vaccine-induced immunity levels among children under the age of 5 years more than doubled, to 56%.

CONCLUSIONS
The higher efficacy of monovalent type 1 oral poliovirus vaccine (four times as effective as trivalent oral poliovirus vaccine) and the moderate gains in coverage dramatically increased vaccine-induced immunity against serotype 1 in northern Nigeria. Further increases in coverage in Nigerian states with infected populations are required to achieve the levels of vaccine-induced immunity associated with the sustained elimination achieved in other parts of the country.
By 2003, the Global Polio Eradication Initiative had interrupted the transmission of indigenous wild polioviruses in all but six countries worldwide, including Nigeria.\(^1\) In that year, however, the temporary suspension of all poliovirus immunization in one Nigerian state contributed to a national epidemic of poliomyelitis and the reinfection of at least 20 previously wild-type poliovirus–free countries.\(^2\) Although poliovirus immunization was re instituted within 12 months, vaccine coverage remained low across northern Nigeria, and case numbers continued to increase.

In early 2006, Nigerian health authorities adopted new tools and tactics to accelerate poliovirus eradication. To respond to lingering community concerns, Nigeria’s national immunization days have been replaced by “immunization-plus days,” during which a range of childhood vaccinations and other health interventions are offered along with the oral poliovirus vaccine. To enhance the effectiveness of immunization contacts against types 1 and 3 paralytic poliomyelitis, trivalent oral poliovirus vaccine was replaced with monovalent oral poliovirus vaccine on a number of immunization-plus days from February 2006 to the present (type 1) and from July 2007 to the present (type 3).\(^3,5\) In 2007, type 1 poliomyelitis cases in Nigeria fell by 86% as compared with 2006, contributing to an overall decline in poliomyelitis cases of 75% (see Tables 1A and 1B in the Supplementary Appendix, available with the full text of this article at www.nejm.org). International exportations of poliovirus also plummeted, suggesting a vast improvement in the prospects for global eradication.

Although both the use of monovalent type 1 oral poliovirus vaccine and the implementation of immunization-plus days are generally credited with the progress in 2007, a critical evaluation of the interventions used is essential in assessing the feasibility of completely eliminating wild-type poliovirus in Nigeria and, ultimately, the world. An estimate of the efficacy of the poliovirus vaccines is especially important in the context of the recent resurgence of cases of type 1 poliomyelitis in 2008 in northern Nigeria. We investigated the protective efficacies of monovalent type 1 oral poliovirus vaccine and trivalent oral poliovirus vaccine in Nigeria, the changes in vaccine coverage after the introduction of immunization-plus days, the resulting vaccine-induced population immunity over time, and the levels of immunity associated with local interruption of wild poliovirus in this setting. We discuss the implications of these results for the elimination of wild-type poliovirus from Nigeria.

**Methods**

**Data Collection**

We identified cases of poliomyelitis in the national database maintained by the government of Nigeria, which since December 1996 has recorded all cases of acute flaccid paralysis detected and reported among children under the age of 15 years.\(^6\) Acute flaccid paralysis is characterized by the rapid onset of weakness and progression to maximum severity within several days to weeks, with the absence of spasticity or other signs of disordered motor tracts in the central nervous system, such as hyperreflexia, clonus, or extensor plantar responses.\(^7\) Since 2001, the quality of national surveillance for cases of acute flaccid paralysis not caused by poliovirus has exceeded the international performance standard of at least 1 case reported annually per 100,000 children.\(^8\) Consequently, the cases of acute flaccid paralysis examined had reported dates of onset of paralysis between January 1, 2001, and December 31, 2007. All such cases are subject to standard clinical, epidemiologic, and laboratory investigations, including the collection of two stool samples, at least 24 hours apart, to test for wild and vaccine poliovirus. The number of doses of oral poliovirus vaccine received by each child with acute flaccid paralysis (as reported by a parent or guardian) was recorded during interviews before laboratory testing of the stool samples to ensure blinding of both parents (or other caregivers) and investigators.

Other data collected in the initial interviews included date of birth, date of onset of paralysis, and location of residence (administratively, Nigeria is divided into 6 zones, 37 states, and 774 local government areas). A total of 27,379 cases of acute flaccid paralysis were in the data set. The following cases were excluded from the study: those in which there was residual paralysis compatible with poliomyelitis and for which two adequate stool specimens were not available (3.0%), those in which vaccine virus was isolated from stool (5.4%), and those in which the number of vaccine doses received or the age at the onset of paralysis were not reported (9.3% and 3.9%, respectively). Consequently, a total of 21,815 cases of acute flaccid...
paralysis (79.7%) were included in the study (see Fig. 1 in the Supplementary Appendix).

A case of type 1 or type 3 poliomyelitis was defined as any case of acute flaccid paralysis with virologic confirmation of type 1 or type 3 wild poliovirus, respectively, from at least one stool sample. The sensitivity of testing for poliovirus in two stool samples was estimated with the use of published methods (see the Methods section in the Supplementary Appendix). In this study, cases of acute flaccid paralysis not caused by poliovirus were defined as those in which neither wild nor vaccine-related poliovirus was isolated. Acute flaccid paralysis not caused by poliovirus may result from a range of other conditions, including the Guillain–Barré syndrome and infection with other enteroviruses. Routine immunization coverage in Nigeria is low; nationwide in 2006, just 37% of children had received three doses of trivalent oral poliovirus vaccine by 1 year of age. Routine coverage is extremely heterogeneous nationwide and particularly low in northern states. Most poliovirus vaccine doses are received through supplementary immunization activities, which were introduced in 1996. We therefore assumed that children received all doses of oral poliovirus vaccine through supplementary immunization activities rather than through routine immunization.

The number of these activities during which a child with acute flaccid paralysis was exposed to each vaccine type was calculated on the basis of the child’s date of birth, age at onset of paralysis, and state of residence (see Table 2 in the Supplementary Appendix). The fraction of the rounds in which each vaccine type was used was multiplied by the number of reported doses received to yield an expected number of doses of each type received. The sensitivity of the results to the assumption that all doses were received by means of supplementary immunization activities was assessed by assuming that the first three doses received by randomly chosen children in the analysis were trivalent oral poliovirus vaccine provided during routine services and that the remaining doses were provided during supplementary immunization activities and consisted of monovalent type 1 oral poliovirus vaccine, trivalent oral poliovirus vaccine, and monovalent type 3, as described above. The fraction of children assumed to have received routine trivalent oral poliovirus vaccine doses in this sensitivity analysis was based on the reported routine immunization coverage in each zone; the random analysis was repeated 100 times (see the Methods section in the Supplementary Appendix). The potency of each monovalent vaccine is equivalent to the individual components of the trivalent vaccine. Thus, a dose of monovalent type 1 oral poliovirus vaccine contains $10^6$ median cell-culture infectious doses (CCID$_{50}$), and a dose of monovalent type 3 oral poliovirus vaccine contains $10^5$–8 CCID$_{50}$, as found in the trivalent formulation.

Surveillance includes investigation of all potential cases of vaccine-associated paralytic poliomyelitis. Among children who have received the first dose of vaccine, the incidence of vaccine-associated paralytic poliomyelitis is 1 case per 1 million children, and the incidence is much lower after subsequent doses. The Expert Review Committee in Nigeria examines all potential cases of vaccine-associated paralytic poliomyelitis and has reported no increase in the incidence since the introduction of monovalent type 1 oral poliovirus vaccine. Analysis of the National Surveillance database, which tracks the use of standard vaccines licensed by the National Regulatory Authority of the Government of Nigeria for use in Nigeria, without potential for individual case identification, does not require institutional ethics approval. All authors vouch for the completeness and accuracy of the data reported.

**Statistical Analysis**

Two matched case–control studies (one for type 1 poliovirus, and one for type 3) were used to estimate the efficacies of the vaccines against paralytic poliomyelitis (type 2 wild poliovirus was eradicated globally in 1999). Children with poliomyelitis were matched in a 1:1 ratio with controls randomly selected from the database of children with acute flaccid paralysis not caused by poliovirus; matching was based on the age at onset of paralysis (within 6 months), the date of onset of paralysis (within 6 months), and region (same local government area). These matching criteria were chosen to maximize the number of case–control pairs while minimizing the biases that might be introduced by a failure to control for differences in exposure between cases and controls. Children with acute flaccid paralysis that was not caused by poliovirus were considered appropriate controls because all cases of acute flaccid paralysis were investigated before confirmation.
of poliovirus status, ensuring the blinding of investigators and parents (or guardian) during dose-reporting interviews. Conditional logistic-regression analysis of the 1:1 matched data was used to relate the log-odds of disease to the number of doses of vaccine (see the Methods section in the Supplementary Appendix). To assess the validity of reporting a constant efficacy per dose of vaccine, the number of doses was entered as a continuous variable and as a categorical variable in separate models, and these models were compared by means of a likelihood-ratio test. Variations in vaccine efficacy by zone and potential interactions between the vaccines were investigated. The sensitivity of the efficacy estimates to the matching criteria was also examined.

Vaccine coverage was estimated on the basis of the reported number of doses in cases of acute flaccid paralysis not caused by poliovirus, with cases weighted to match the underlying distribution of the population by state and single years of age. Differences in vaccine coverage between 2005 and 2007 were tested with the use of a multinomial chi-square test (see the Methods section in the Supplementary Appendix). The fraction of children younger than 5 years of age who were protected by direct vaccination against type 1 paralytic poliomyelitis (vaccine-induced population immunity) was calculated from the estimated coverage with monovalent type 1 oral poliovirus vaccine and trivalent oral poliovirus vaccine and their estimated efficacies (see the Methods section in the Supplementary Appendix). The contribution of changes in coverage only to changes in population immunity was estimated by recalculating these levels with the assumption that trivalent oral poliovirus vaccine was used in all rounds. The correlation between the proportion of children younger than 5 years of age who were protected by vaccination in each state and the reported numbers of cases of type 1 poliomyelitis was estimated with the use of Spearman's rank-correlation coefficient.

**RESULTS**

**VACCINE EFFICACY**

A total of 2532 children were reported to have type 1 poliomyelitis and 1092 were reported to have type 3 poliomyelitis during the study period. Of these children, 1174 with type 1 poliomyelitis (46%) and 502 with type 3 poliomyelitis (46%) were matched with a suitable control (Table 1). The estimated efficacy of trivalent oral poliovirus vaccine against paralysis from type 1 poliomyelitis across Nigeria was 16% (95% confidence interval [CI], 10 to 21) per dose, and the efficacy of monovalent type 1 oral poliovirus vaccine was 67% (95% CI, 39 to 82) per dose. The estimated efficacy of trivalent oral poliovirus vaccine against paralysis from type 3 poliomyelitis was 18% (95% CI, 9 to 26) per dose. There was insufficient use of monovalent type 3 oral poliovirus vaccine to allow the efficacy of this vaccine to be estimated reliably.

Vaccine efficacies did not differ significantly according to the zone of the country (Table 1), nor were there significant differences when efficacies were estimated separately according to year or age (see Tables 3A and 3B in the Supplementary Appendix). There was no evidence of an interaction between monovalent type 1 oral poliovirus vaccine and trivalent oral poliovirus vaccine in the type 1 model (P=0.82). Comparisons of the fit of the model with a constant efficacy per dose of vaccine with a model that allowed the protective efficacy to vary according to the dose number showed that a constant efficacy per dose was a reasonable assumption for both monovalent type 1 oral poliovirus vaccine and trivalent oral poliovirus vaccine in the type 1 model (P=0.93 and P=0.085, respectively, for monovalent and trivalent oral poliovirus vaccine against type 1 paralytic poliomyelitis; P=0.36 for trivalent oral poliovirus vaccine against type 3) (see Fig. 2 in the Supplementary Appendix). These findings are consistent with the all-or-nothing mode of action of these live-virus vaccines. Tighter matching of cases and controls according to age and date at the onset of paralysis did not substantially alter the estimates of vaccine efficacy (see Table 4 in the Supplementary Appendix). Sensitivity analyses showed that our results were robust with respect to various assumptions regarding the number of doses received through routine immunization (see Table 5 in the Supplementary Appendix).

The estimated sensitivity of laboratory testing for wild poliovirus was 99% and 98% for types 1 and 3, respectively. The prevalence of type 1 and type 3 poliovirus among cases of acute flaccid paralysis was estimated at 9.3% and 3.9%, respec-
tively. Therefore, the probability of misclassifying a case of acute flaccid paralysis not caused by poliovirus as a case of type 1 or type 3 poliomyelitis was 0.0011 and 0.0006, respectively.

**Immunization Coverage and Program Effectiveness**

Significant improvements in immunization coverage were registered between 2005 (before the administration of monovalent type 1 oral poliovirus vaccine and immunization-plus days) and 2007 (after the administration of monovalent type 1 oral poliovirus vaccine and immunization-plus days) in the northwestern and northeastern zones, which reported the majority of poliomyelitis cases ($P<0.001$) (Table 2). Coverage in the southeastern zone declined significantly between 2005 and 2007 ($P<0.001$) (Table 2). Coverage in the three remaining zones was high, and there were no significant changes between 2005 and 2007.

The combination of improved coverage and use of the more efficacious monovalent type 1 oral poliovirus vaccine has resulted in substantial increases in the proportion of children protected through direct immunization against type 1 paralytic poliomyelitis (Fig. 1 and Table 2). In the northwestern zone, 33% of the improvement in vaccine-induced immunity between 2005 and 2007 was due to improved coverage, whereas in the southern zones of the country, the improvement in vaccine-induced immunity was solely due to the introduction of the monovalent vaccine (see Tables 6 and 7 in the Supplementary Appendix).

The estimated fraction of children who were protected against type 1 paralytic poliomyelitis strongly correlates with the reported numbers of cases of type 1 poliomyelitis (Fig. 1). The correlation was significant in all years, rising significantly in more recent years ($P=0.02$) (see Table 8 in the Supplementary Appendix). Plotting the fraction of children younger than 5 years of age who were protected from type 1 paralytic poliomyelitis against the incidence of poliomyelitis for each year and state suggests that in some areas a level of 80% protection has been sufficient to achieve local elimination (Fig. 2).

**Table 1. Estimated Protective Efficacy of Oral Poliovirus Vaccines against Type 1 and Type 3 Paralytic Poliomyelitis in Nigeria According to Zone, 2001–2007.**

<table>
<thead>
<tr>
<th>Zone</th>
<th>Total No. of Cases</th>
<th>Case–Control Matches</th>
<th>Efficacy of Monovalent Vaccine</th>
<th>Efficacy of Trivalent Vaccine</th>
<th>Total No. of Cases</th>
<th>Case–Control Matches</th>
<th>Efficacy of Trivalent Vaccine</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>no. (%)</td>
<td>% (95% CI)</td>
<td>no. (%)</td>
<td>% (95% CI)</td>
<td>no. (%)</td>
<td>% (95% CI)</td>
<td>% (95% CI)</td>
</tr>
<tr>
<td>Northwest</td>
<td>1899</td>
<td>925 (49)</td>
<td>63 (27–82)</td>
<td>13 (6–19)</td>
<td>851</td>
<td>402 (47)</td>
<td>17 (7–26)</td>
</tr>
<tr>
<td>Northeast</td>
<td>437</td>
<td>158 (36)</td>
<td>58 (0–88)</td>
<td>23 (7–37)</td>
<td>173</td>
<td>67 (39)</td>
<td>18 (0–40)</td>
</tr>
<tr>
<td>North Central</td>
<td>154</td>
<td>68 (44)</td>
<td>100 (0–100)</td>
<td>23 (1–40)</td>
<td>52</td>
<td>16 (31)</td>
<td>24 (0–56)</td>
</tr>
<tr>
<td>South*</td>
<td>42</td>
<td>23 (55)</td>
<td>—†</td>
<td>54 (4–78)</td>
<td>16</td>
<td>8 (50)</td>
<td>12 (0–54)</td>
</tr>
<tr>
<td>All zones</td>
<td>2532</td>
<td>1174 (46)</td>
<td>67 (39–82)</td>
<td>16 (10–21)</td>
<td>1092</td>
<td>502 (46)</td>
<td>18 (9–26)</td>
</tr>
</tbody>
</table>

* Results for the South include all three southern zones (Southwest, South-South, and Southeast).
† During the study period, there were no reported cases of type 1 paralytic poliomyelitis in the South after the introduction of monovalent type 1 oral poliovirus vaccine.

**Discussion**

This study provides estimates of the efficacy of oral poliovirus vaccines in Nigeria and the effect of these vaccines on population immunity. Although trivalent oral poliovirus vaccine was successfully used to interrupt transmission of indigenous type 1 wild polioviruses across southern Nigeria, the recently licensed monovalent type 1 oral poliovirus vaccine, with an estimated efficacy per dose that is four times that of trivalent oral poliovirus vaccine, substantially improves the prospects for accelerating elimination in northern Nigeria. Because of improvements in immunization coverage and the introduction of monovalent type 1 oral poliovirus vaccine, vaccine-induced immunity in the northwestern and northeastern zones (which reported 96% of all type 1 cases in Nigeria in 2006) more than doubled between 2005 and 2007. However, further gains in immunization coverage are required to achieve elimination, since 21% of children in the northwestern zone (where the majority of cases have occurred)
effectiveness of immunization against poliomyelitis in Nigeria

Still have never received a single dose of vaccine, with a further 55% having received fewer than the recommended four doses.

The estimated efficacy of trivalent oral poliovirus vaccine in Nigeria is similar to its efficacy in India, with the exception of the northern Indian state of Uttar Pradesh, where the substantially lower efficacy of oral poliovirus vaccines appears to be the result of a high prevalence of diarrheal and other infections that interfere with seroconversion. The efficacy of trivalent oral poliovirus vaccine did not show significant variation across Nigeria, although case numbers in the South were limited (Table 1). The efficacy of monovalent type 1 oral poliovirus vaccine is higher in northern Nigeria than in Uttar Pradesh and is similar to rates of seroconversion after administration of monovalent type 1 oral poliovirus vaccine in other developing countries. This finding suggests that the persistence of poliovirus in northern Nigeria is not a result of environmental conditions that can compromise vaccine efficacy, as it is in northern India.

The precision of our efficacy estimates could be affected by the accuracy of the reporting of oral poliovirus vaccine doses and potential differences in exposure to poliovirus between cases and controls. However, the fact that the parent (or guardian) is unaware of the poliovirus status of a child when being interviewed minimizes the risk of systematic bias in dose reporting. In addition, since controls came from the same area as case subjects, the two groups are likely to have had similar exposure to virus; indeed, efficacy estimates were robust to closer matching of case subjects with controls. Nearly 50% of case subjects were matched to a control, and the resulting exclusion of some case subjects does not appear to have introduced any bias (see Tables 9A and 9B in the Supplementary Appendix). Perhaps more important, irrespective of the precision of the point estimate of vaccine efficacy, the calculated relative gain in efficacy for monovalent type 1 oral poliovirus vaccine as compared with trivalent oral poliovirus vaccine against type 1 paralytic poliomyelitis was robust with respect to these potential biases. Vaccine quality is unlikely to have been an issue, since temperature monitors on vaccine vials have been used nationwide since 2000 to prevent the administration of vaccine with low potency.

Our estimates of population immunity do not

<table>
<thead>
<tr>
<th>Year</th>
<th>Northwest</th>
<th>Northeast</th>
<th>North Central</th>
<th>Southwest</th>
<th>Southeast</th>
<th>South-South</th>
<th>Whole Country</th>
</tr>
</thead>
<tbody>
<tr>
<td>2005</td>
<td>909</td>
<td>358</td>
<td>529</td>
<td>480</td>
<td>219</td>
<td>229</td>
<td>2724</td>
</tr>
<tr>
<td>No. of children</td>
<td>2007</td>
<td>740</td>
<td>318</td>
<td>522</td>
<td>450</td>
<td>273</td>
<td>424</td>
</tr>
<tr>
<td>No. of reported doses</td>
<td></td>
<td>0</td>
<td>21</td>
<td>9</td>
<td>3</td>
<td>1</td>
<td>7</td>
</tr>
<tr>
<td>1–3</td>
<td>55</td>
<td>59</td>
<td>38</td>
<td>27</td>
<td>46</td>
<td>31</td>
<td>43</td>
</tr>
<tr>
<td>&gt;3</td>
<td>24</td>
<td>32</td>
<td>59</td>
<td>72</td>
<td>46</td>
<td>66</td>
<td>48</td>
</tr>
<tr>
<td>Children protected (95% CI)</td>
<td></td>
<td>22 (15–28)</td>
<td>31 (28–35)</td>
<td>49 (46–52)</td>
<td>56 (51–62)</td>
<td>50 (45–54)</td>
<td>53 (49–57)</td>
</tr>
<tr>
<td>2007</td>
<td>740</td>
<td>318</td>
<td>522</td>
<td>450</td>
<td>273</td>
<td>424</td>
<td>2727</td>
</tr>
<tr>
<td>No. of reported doses</td>
<td></td>
<td>0</td>
<td>21</td>
<td>9</td>
<td>3</td>
<td>1</td>
<td>7</td>
</tr>
<tr>
<td>1–3</td>
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<td>59</td>
<td>38</td>
<td>27</td>
<td>46</td>
<td>31</td>
<td>43</td>
</tr>
<tr>
<td>&gt;3</td>
<td>24</td>
<td>32</td>
<td>59</td>
<td>72</td>
<td>46</td>
<td>66</td>
<td>48</td>
</tr>
<tr>
<td>Children protected (95% CI)</td>
<td></td>
<td>56 (41–71)</td>
<td>61 (48–73)</td>
<td>65 (53–77)</td>
<td>83 (72–94)</td>
<td>71 (57–84)</td>
<td>81 (72–91)</td>
</tr>
</tbody>
</table>

Distribution of the number of doses among case subjects differed significantly between 2005 and 2007 in the northwestern, northeastern, and southeastern zones and across the whole country (P<0.001 for all four comparisons between the 2 years).
account for secondary transmission of vaccine virus, maternal antibodies in the first few months of life, or natural immunity from exposure to circulating wild poliovirus. However, they do allow a direct comparison of vaccine-induced immunity between states that have had supplementary immunization activities with different types of vaccine and variable frequency and coverage, thereby providing a good relative indication of program effectiveness. The improvements observed in vac-
cine coverage indicate that increases in immunity levels in northwestern Nigeria are not purely a result of the higher efficacy of monovalent type 1 oral poliovirus vaccine. Many factors probably contributed, including the introduction of immunization-plus days and other such efforts by field staff and local and national leaders to address the concerns of community leaders and parents that had contributed to the suspension of the use of oral poliovirus vaccine in 2003.

The encouraging findings of this study must be interpreted with caution in assessing the overall outlook for the elimination of poliovirus in Nigeria. With substantial population movements and an annual birth rate of approximately 40 births per 1000 population, the growth of the susceptible population is considerable. Although a level of direct immunity from vaccination of approximately 30% can reduce the transmission of type 1 poliovirus in Nigeria (Fig. 2), a level of at least 80% is required for sustained local elimination of transmission. As of 2007, some degree of control appears to have been achieved in many northern states, but considerable further improvements are still required to reach a level at which poliovirus can be eradicated (Fig. 1). The consequences of such ongoing immunity gaps were evident in the recent emergence of a circulating vaccine-derived poliovirus in the North and a resurgence of type 1 cases in early 2008 that have spread to the South. Nonetheless, giving each child in the northwestern zone just one additional dose of monovalent type 1 oral poliovirus vaccine could increase direct population immunity to more than 80% in much of this area — a substantial increase from the 56% immunity recorded in 2007.

India and Nigeria reported 88% of poliomyelitis cases worldwide in 2007 and are central to global eradication. In contrast with India, in northern Nigeria vaccine coverage remains a significant barrier to completing the elimination of poliovirus. The relatively high efficacy of monovalent type 1 oral poliovirus vaccine in Nigeria offers the potential for rapid eradication of type 1 poliovirus, provided that vaccination coverage continues to improve in the northern part of the country.

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