HHS Public Access

Author manuscript

J Infect Dis. Author manuscript; available in PMC 2019 June 03.

Published in final edited form as:

J Infect Dis. 2014 November 01; 210(Suppl 1): S136–S142. doi:10.1093/infdis/jiu086.

Poliovirus Immunity Among Pregnant Females Aged 15–44 Years, Namibia, 2010

Cristina V. Cardemil[1], **Anna Jonas**[2], **Sue Gerber**[3], **William C. Weldon III**[4], **M. Steven Oberste**[4], **Anita Beukes**[3], **Souleymane Sawadogo**[3], **Sadhna V. Patel**[3], **Sikota Zeko**[2], **Clementine Muroua**[2], **Esegiel Gaeb**[5], **Kathleen Wannemuehler**[1], and **James L. Goodson**[1]

[1]Centers for Disease Control and Prevention, Global Immunization Division, United States

[2]Ministry of Health and Social Services, Namibia,

[3]Centers for Disease Control and Prevention, Division of Global HIV/AIDS, Namibia

[4]Centers for Disease Control and Prevention, Division of Viral Diseases, United States

[5]Namibia Institute of Pathology

Abstract

Background.—Poliovirus (PV) antibody seroprevalence studies assess population immunity, verify an immunization program's performance and vaccine efficacy, and guide polio eradication strategy. Namibia experienced a polio outbreak among adults in 2006, yet population seroimmunity was unknown.

Methods.—We tested 2061 specimens from Namibian pregnant females aged 15–44 years for neutralizing antibody to PV types 1–3 (PV1–3); all females were sampled during the 2010 National HIV Sentinel Survey. We determined the proportion of females seropositive for PV antibody by 5-year age strata, and analyzed factors associated with seropositivity, including age, gravidity, human immunodeficiency virus (HIV) infection status, residence, and antiretroviral treatment, by log-binomial regression.

Results.—The seroprevalence was 94.6% for PV1, 97.0% for PV2, and 85.1% for PV3. HIV-positive females had significantly lower seroprevalence than HIV-negative females for PV1 (91.8% vs 95.3%; $P < .01$) and PV3 (80.0% vs 86.1%; $P < .01$) but not for PV2 (96.4% vs 97.1%; $P = .3$). The prevalence ratio of seropositivity for HIV-positive females versus HIV-negative females was 0.95 (95% confidence interval [CI], .92–.98) for PV1, 0.99 (95% CI, .97–1.01) for PV2, and 0.92 (95% CI, .87–.96) for PV3.

Correspondence: Cristina V. Cardemil, MD, MPH, Centers for Disease Control and Prevention, 1600 Clifton Rd NE, MS A-19, Atlanta, GA 30333 USA (iyk8@cdc.gov).

Disclaimer. The findings and conclusions in this report are those of the authors and do not necessarily represent the official position of the Centers for Disease Control and Prevention.

Supplement sponsorship. This article is part of a supplement entitled "The Final Phase of Polio Eradication and Endgame Strategies for the Post-Eradication Era," which was sponsored by the Centers for Disease Control and Prevention.

Potential conflicts of interest. All authors: No reported conflicts.
All authors have submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Conflicts that the editors consider relevant to the content of the manuscript have been disclosed.

Conclusions.—Despite relatively high PV seroprevalence, Namibia might remain at risk for a PV outbreak, particularly in lower-seroprevalence populations, such as HIV-positive females. Namibia should continue to maintain high routine polio vaccination coverage.

Keywords

Poliovirus; polio; seroprevalence; OPV; Namibia; adults; pregnant women; population immunity; HIV; neutralizing antibody

Through efforts of the Global Polio Eradication Initiative, indigenous transmission of wild poliovirus (WPV) has been interrupted in all countries of the world except Nigeria, Afghanistan, and Pakistan. Nonetheless, polio outbreaks following WPV importations have been reported in 2013 from a number of countries where polio is not endemic, including Cameroon, Ethiopia, Kenya, Somalia, and Syria [1]. Although polio is traditionally a disease of childhood, several outbreaks in recent years have affected a larger than expected proportion of adults, likely because of immunity gaps, and threaten to spread to other areas with an accumulation of polio-susceptible persons [2–4].

Namibia, a country in southern Africa that gained independence from South Africa in 1990, had an estimated population of 2.1 million in 2009 and a population density of 2.1 persons/km^2, the second lowest population density in the world [5]. The capital city is Windhoek, and the country is administratively organized into 34 health districts in 14 regions, including those with the highest populations, which are located in the northern part of the country, along the border with Angola, and those in the central and the southern parts of the country [6]. More than 67% of Namibian residents are rural dwellers [7]. In 2012, Namibia had one of the highest human immunodeficiency virus (HIV) prevalence rates for adults aged 15–49 years in the world, at 13.3% [8], and the overall prevalence was high, compared with that in other countries in sub-Saharan Africa [9]. The Expanded Programme on Immunization began in 1990 after independence. The current childhood immunization schedule includes oral polio vaccine (OPV), given at birth and at 6, 10, and 14 weeks of age, with additional OPV doses at 15 months, 5 years, and 10 years of age [10]. Since 2000, Namibia implemented ≥2 annual mass OPV campaigns.

In 2006, after 10 years of no reported polio cases, Namibia experienced an outbreak of WPV type 1 (WPV1) infection following a WPV1 importation from Angola, with 277 acute flaccid paralysis cases reported. Of the reported acute flaccid paralysis cases, 19 were laboratory-confirmed polio cases, and 26 were classified as polio-compatible cases [11]. Of the 45 reported polio cases, all were among persons ≥14 years of age, and the case-fatality ratio was 24%. Reported confirmed polio cases were distributed in the north, along the Angolan border, as well as in and around Windhoek [11]. In response to this outbreak, the Namibia Ministry of Health and Social Services implemented 3 nationwide supplementary immunization activities (SIAs) in 2006, using both house-to-house and fixed-site vaccine delivery strategies [11, 12]. The first 2 SIAs targeted adults and children of all ages with monovalent OPV type 1, and the third SIA provided trivalent OPV to children <5 years of age [12]. Administrative data indicated >95% vaccination coverage in >80% of the 34 districts [11]. Since the immunization activities associated with the 2006 outbreak response,

Namibia has conducted 2 polio national immunization days per year [13]. In 2006, estimated routine coverage with 3 doses of OPV among 1-year-old individuals was 74%, increasing to 84% in 2012 [10].

The 2006 outbreak of WPV1 infection in Namibia exemplified how suboptimal immunization coverage can lead to a lapse in population immunity and to a large number of accumulated susceptible persons, particularly among older age groups. Although this outbreak was followed by multiple OPV rounds in all age groups, likely diminishing the risk for another polio outbreak, this outbreak and other recent large-scale outbreaks of WPV infection provide evidence that adults may be contributing to sustained poliovirus (PV) transmission, highlighting the importance of maintaining strong surveillance and ensuring high population immunity across all age groups [2–4,14].

In 2009, the independent Advisory Committee on Poliomyelitis Eradication, which provided technical advice to the World Health Organization, recommended conducting PV seroprevalence surveys to assess population immunity, verify immunization program performance and vaccine efficacy, and guide eradication strategies [15]. Additionally, WPV-endemic countries and countries at risk of WPV importation were encouraged to develop plans for strengthening routine immunization services. Following these recommendations, to determine population immunity to PV types 1, 2, and 3 (PV1–3) in Namibia and examine factors associated with polio seropositivity, stored serum samples from the 2010 national HIV survey were tested. PV antibody seroprevalence estimates provide a tool to (1) assess polio immunity in adults following the 2006 outbreak, (2) evaluate response immunization activities, and (3) guide the national immunization program's risk assessment to decrease the risk of future polio outbreaks following PV importations.

METHODS

In 2010, the Namibia Ministry of Health and Social Services conducted a nationwide sentinel survey to estimate HIV prevalence among pregnant females aged 15–49 years. The survey design accorded with the World Health Organization's standardized methods for HIV prevalence surveys, using convenient consecutive sampling of females attending antenatal clinics (ANCs) selected on the basis of geographic representation from all regions and health districts, urban and rural clinics, areas with different population densities and sizes, and females with different socioeconomic status [16, 17]. All pregnant females 15–49 years of age were included in the survey if they attended an ANC for the first time during their current pregnancy, were not referred from another health facility, and agreed to collection of a blood sample for routine syphilis screening.

The 2010 survey enrolled 7983 pregnant females from all 34 districts, which included 35 main hospitals and 93 satellite health centers and clinics; 7888 of enrollees (98.8%) had specimens collected during 22 March-6 September 2010 [17]. Un-linked, de-identified specimens were tested for HIV antibodies, de-identified data from all data fields (ie, unique identification, district abbreviation and site number, facility type, date of ANC visit, age, gravidity, place of residence, antiretroviral therapy participation, and counseling for

prevention of maternal-to-child transmission) were retained electronically, and specimens were stored at 4–8°C at the Namibia Institute of Pathology in Windhoek.

To estimate PV antibody seroprevalence within each 5-year age group, it was determined that 428 specimens per age group were necessary, assuming a seroprevalence of 50%, a desired precision of ±5%, a probability of achieving the desired precision of 0.95, and a 10% loss due to specimens that were not found or inadequate. We excluded women 45–49 years because the number of specimens available in this group was too few to result in meaningful estimates. We included all specimens for women 40–44 years because the number available was less than the target number. To control the distribution of HIV-infected females within each age group, we determined the target sample sizes of the HIV-positive and HIV-negative groups by using the observed distribution in the ANC sentinel survey [17].

Samples were tested in triplicate by using a standard microneutralization assay for antibodies to PV1–3 according to established protocols at the Global Polio Specialized Laboratory, CDC [18,19]. Briefly, 80–100 50% cell culture infectious doses of each PV serotype and 2-fold serial dilutions of serum were combined and preincubated at 37°C for 3 hours before addition of HEp-2(C) cells. After incubation for 5 days at 37°C and 5% CO_2, plates were stained with crystal violet, and cell viability measured by optical density in a spectrophotometer. Each specimen was run in triplicate, with parallel specimens from one study subject tested in the same assay run. Neutralization titers were estimated by the Spearman-Kärber method [20] and reported as the reciprocal of the calculated 50% end point. Each run contained multiple replicates of a reference antiserum pool, starting at a 1:32 dilution, to monitor performance variation. A serum sample was considered positive if antibodies were present at a dilution of ≥1:8. The samples with unobserved titers in starting and final dilutions were assigned values of <8 and ≥1448, respectively.

Seroprevalence estimates and 95% confidence intervals (determined by the Wilson-score method) were calculated for each age group, as well as within the following subpopulations: urban-rural residence, HIV status, antiretroviral therapy status, gravidity, facility type (hospital, health center, or clinic), and health district. Health districts with a high HIV prevalence were defined as districts with 2010 prevalence of >22% according to sentinel survey data. The Cochran-Mantel-Haenszel general association test was used to compare seroprevalence among HIV-positive females with that among HIV-negative females, after adjustment for age. The ratio of the seroprevalence (prevalence ratio) among HIV-positive females versus HIV-negative females was calculated by log-binomial regression for each serotype separately, and each model was examined after controlling for age, urban/rural residence, facility type, gravidity, and antiretroviral therapy status, including 2-way interaction terms for the aforementioned variables, until the best fitting and most parsimonious model for each serotype was determined. Titers were compared using a stratified Wilcoxon nonparametric test, with adjustment for age. All analyses included sampling weights, which were calculated on the basis of the probability of selection within each of the 12 age-HIV infection strata and adjusted for nonresponse (ie, defined as specimens that were not available or inadequate for testing). Data were analyzed using SAS, version 9.3 (SAS Institute, Cary, NC). This study received ethical approval from the CDC and the Namibia Ministry of Health and Social Services.

RESULTS

On the basis of the sample size calculation, 2692 specimens were selected for inclusion in the study; of these, 388 (14%) were not available, 213 (8%) had insufficient volume, and 29 (1%) were hemolyzed and therefore could not be used for laboratory testing. The remaining 2062 specimens were shipped to the CDC Global Specialized Poliovirus Laboratory for evaluation and testing. Of the 2062 shipped specimens, 2061 were identified by the laboratory as suitable for testing for PV1, PV2, and PV3. Of these 2061 specimens, 1 had volume sufficient for testing of PV1 and PV3 but not PV2 and was excluded from testing for PV2; the remaining 2060 samples had sufficient volume for testing for all 3 PV types. Supplementary Table 1 shows the target sample size, the observed sample size, and percentage of samples not tested, by age group and HIV status. The analysis comparing demographic characteristics of females for whom specimens were not available, insufficient, or hemolyzed to the demographic characteristics of females for whom specimens were tested and included in analysis found no substantial differences for age group, HIV status, urban/rural residence or gravidity (data not shown).

Table 1 shows the overall PV antibody seropositivity for all 3 serotypes. The seroprevalence was 94.6% (95% CI, 93.6%– 95.5%) for PV1, 97.0% (95% CI, 96.1%–97.6%) for PV2, and 85.1% (95% CI, 83.5%–86.6%) for PV3. Seropositivity for any of the 3 serotypes was 99.1% (95% CI, 98.6%–99.4%). Seropositivity for all 3 serotypes was 80.6% (95% CI, 78.8%–82.2%). Table 2 shows the PV antibody seroprevalence by age group, urban/rural status, gravidity, and facility type; no substantial differences were seen in seroprevalence after stratification by these factors. Supplementary Table 2 shows the results for PV antibody seroprevalence by health district; seroprevalence varied by health district and ranged from 77.6% (95% CI, 62.3%– 88.0%) to 100.0% (95% CI, 84.5%–100.0%) for PV1, from 77.0% (95% CI, 61.3%–87.6%) to 100.0% (95% CI, 84.5–100.0) for PV2, and from 74.8% (95% CI, 56.0%–87.4%) to 94.8% (95% CI, 84.8%–98.4%) for PV3.

Table 3 shows the PV antibody seroprevalence, by HIV status. After controlling for age, HIV-positive females had lower PV1 and PV3 seroprevalence than HIV-negative females (PV1, 91.8% vs 95.3%; PV3, 80.0% vs 86.1%), and these differences were statistically significant (PV1, P =.0015; PV3, P = .0027). HIV-positive females had slightly lower PV2 seroprevalence than HIV-negative females (PV2, 96.4% vs 97.1%), but this difference was not statistically significant (P = .2588).

After controlling for age, the ratio of the prevalence of seropositivity among HIV-positive females to that among HIV-negative females was 0.95 (95% CI, .92–.98; P = .0024) for PV1 and 0.99 (95% CI, .97–1.01, P = .3724) for PV2. After controlling for age, urban/rural residence, and gravidity status, the ratio of the prevalence of PV3 seropositivity among HIV-positive females to that among HIV-negative females was 0.92 (95% CI, .87–.96; P = .0009).

Among females with positive PV antibody titers, the median titer in the HIV-positive group was lower than that in the HIV-negative group for PV1 and PV2 but not for PV3 (PV1, 7.2 vs 8.5 [P<.0001]; PV2, 6.8 vs 7.5 [P<.0001]; PV3, 5.5 vs 5.5 [P = .89]). There were no differences in PV antibody seroprevalence for HIV-positive females after stratification by

HIV treatment status. No association was found between PV antibody seroprevalence among health districts after stratification by high versus low HIV prevalence districts.

DISCUSSION

In 2010, PV antibody seroprevalence among adult pregnant females sampled during the ANC sentinel survey in Namibia was >90% for PV1 and PV2 and slightly lower for PV3. HIV status affected PV antibody seroprevalence: HIV-positive females had lower PV antibody seroprevalence than HIV-negative females for PV1 and PV3 but not for PV2. Variation in PV antibody seroprevalence for all 3 types across health districts was observed, with the lowest point estimate reaching 74.8% for PV3 in one health district.

This is the first study of adult PV antibody seroprevalence in Namibia. The levels of polio population immunity in Namibia could be due to past immunization and/or natural exposure to WPV, including during the 2006 outbreak of WPV1 infection. In the past decade, there have been a number of PV antibody seroprevalence studies conducted among adult populations in Europe that have yielded comparable estimates (seropositivity for PV1, 73.3%–99.3%; for PV2, 89.9%–99.1%: and for PV3, 70%–98.8%) [21–28]. One exception is a study in Uruguay, where seroprotection among adults 20–39 years of age against PV1–3 ranged from 20% to 60% [29]. Although the herd immunity threshold above which one can guarantee the prevention of an outbreak is unknown for African settings such as Namibia, it is believed that polio outbreaks in industrialized countries can be prevented with population immunity levels of 66%–80% [30]. In developing countries with suboptimal sanitation and hygiene leading to the potential for increased PV transmission and greater force of infection, WPV outbreaks could theoretically occur with population immunity levels as high as 94%–97% [30]. Moreover, PV outbreaks including sustained PV transmission among fully immunized children have been documented in developing countries [31]. Therefore, until polio is eradicated globally, Namibia remains at risk for PV importation, and polio could spread among vulnerable populations. To avoid outbreaks following WPV importation and to mitigate the spread of a potential outbreak, Namibia should continue to maintain high routine polio vaccination coverage and strong acute flaccid paralysis surveillance for rapid case detection and response.

These findings should be considered in light of limitations. First, there was no immunization history available for participants, so it is unclear whether polio seroimmunity is due to past OPV receipt and/or natural immunity, and we could not examine dose-response effects. Second, only pregnant females 15–44 years old were examined in this study, and the ANC survey was not a random cross-section of the population of pregnant females. Therefore, the results may not be generalizable to all pregnant women in Namibia nor other age groups or populations. Third, we did not have information on $CD4^+$ T-cell count in the HIV-positive females, so we could not examine associations between the level of immunosuppression and polio seroprevalence. Fourth, sample sizes were small at the health district level, limiting conclusions regarding geographic differences.

The effect of HIV status on seroprevalence for PV1 and PV3 in our study was modest but indicated a higher susceptibility to PV infection among HIV-positive females. Past studies

that assessed the effect of HIV status on polio immunity are few in number but are in line with our results. A study in Italy of HIV-positive and HIV-negative adult drug users found lower seropositivity for all 3 PV types in the HIV-positive group [32]. Also, Zimbabwean HIV-infected children born to HIV-infected mothers showed significantly lower rates of seroconversion to all 3 PV serotypes, compared with HIV-uninfected children, and HIV-infected children who seroconverted following polio vaccination had lower geometric mean titers than HIV-uninfected children [33, 34]. The authors of these studies concluded that the association between pediatric HIV infection and poor immunologic response to OPV could potentially pose an obstacle to global polio eradication. In studies of HIV infection and viral shedding, HIV positivity has not been associated with prolonged viral shedding that could potentially lead to the emergence of an immunodeficient vaccine-derived poliovirus [34, 35]. Our finding that HIV-positive pregnant females had lower PV1 and PV3 seroprevalences than HIV-negative females and that PV seropositive HIV-positive females had lower absolute PV1 and PV2 antibody titers than PV seropositive HIV-negative females not only could represent higher susceptibility to polio for those females, but also might potentially lead to lower polio immunity in their offspring, particularly in HIV-exposed infants who become HIV positive.

While WPV2 was eradicated in 1999, the continued use of trivalent OPV, which is known to be less immunogenic for PV1 and PV3 than bivalent OPV, in settings with suboptimal PV population immunity carries the risk of the emergence of circulating vaccine-derived poliovirus type 2 (cVDPV2) and subsequent outbreaks [36]. In light of this, the Strategic Advisory Group of Experts Working Group recommended that countries switch from trivalent OPV to bivalent OPV in the infant routine immunization schedule [36]. However, with this switch, a small risk of the emergence of cVDPV2 exists. Therefore, the Global Polio Eradication Initiative Strategic Plan 2013–2018 includes recommendations for introducing inactivated polio vaccine in routine immunization services and concurrent use of inactivated polio vaccine and bivalent OPV during routine immunization until the time of global certification of polio eradication [37]. This strategy will ensure high population immunity for all 3 PV types, in Namibia and globally, and will reduce the risk of emergence and sustained transmission of vaccine-derived polioviruses.

Conducting periodic seroprevalence surveys in areas at high risk for polio, including polio-endemic and polio-outbreak countries, and countries considered at risk for PV importation is valuable for identifying geographic areas and subpopulations with low polio immunity. Results from these surveys can provide some evidence for determining target age groups for SIAs, outbreak response, and prioritizing the new recommendations for introduction of inactivated polio vaccine and concurrent use of bivalent OPV. Given the high HIV prevalence in the African region, further studies in HIV-positive persons and the effect of HIV infection on polio seroconversion and seroimmunity might further inform global polio eradication strategies.

Acknowledgments.

We thank all staff of the Expanded Programme on Immunization, surveillance officers and laboratory staff in Namibia, and the polio laboratory and data management staff at the Centers for Disease Control and Prevention.

Financial support. This work was supported by the Centers for Disease Control and Prevention.

References

1. Global Polio Eradication Initiative. Data and monitoring: wild poliovirus cases. http://www.polioeradication.org/Dataandmonitoring/Poliothisweek.aspx. Accessed 20 December 2013.
2. Centers for Disease Control and Prevention. Outbreaks following wild poliovirus importations-Europe, Africa, and Asia, January 2009– September 2010. Morb Mortal Wkly Rep 2010; 59:1393–9.
3. Centers for Disease Control and Prevention. Notes from the field: poliomyelitis outbreak-republic of the Congo, September 2010–February 2011. Morb Mortal Wkly Rep 2011; 60:312–3.
4. Patel MK, Konde MK, Didi-Ngossaki BH, et al. An outbreakof wild poliovirus in the Republic of Congo, 2010–2011. Clin Infect Dis 2012; 55:1291–8. [PubMed: 22911642]
5. 2001 Namibia Housing and Population Census. http://www.paris21.org/sites/default/files/825.pdf. Accessed 20 December 2013.
6. World Health Organization—Namibia. 2011 annual report. Geneva: WHO, 2012.
7. Namibia Demographic and Health Survey 2006–07. http://www.measuredhs.com/countries/country_main.cfm?ctry_id=28&c=Namibia. Accessed 20 December 2013.
8. UNAIDS Country Reports HIV and AIDS estimates 2012: Namibia. http://www.unaids.org/en/Regionscountries/Countries/Namibia/. Accessed 6 December 2013.
9. AIDSinfo [database online]. http://www.aidsinfoonline.org/devinfo/libraries/aspx/Home.aspx. Accessed 20 December 2013.
10. WHO Vaccine-Preventable Diseases Monitoring System. 2013 global summary, Namibia. http://apps.who.int/immunization_monitoring/globalsummary/countries?countrycriteria%5Bcountry%5D%5B%5D=NAM&commit=OK. Accessed 20 December 2013.
11. Yusuf N, de Wee R, Forster N, et al. Outbreak of type 1 wild poliovirus in adults, Namibia, 2006. J Infect Dis 2014; 210(suppl 1):S353–60. [PubMed: 25316855]
12. Centers for Disease Control and Prevention. Outbreak of polio in adults - Namibia, 2006. MMWR Morb Mortal Wkly Rep 2006; 55:1198–201. [PubMed: 17093385]
13. Kärber G, Beitrag zur kollektiven. Behandlung pharmakologischer Reihenversuche. Naunyn-Schmiedebergs Archiv für experimentelle Pathologie und Pharmakologie 162:480–483.
14. Kidd S, Goodson JL, Aramburu J, et al. Poliomyelitis outbreaks in Angola genetically linked to India: Risk factors and implications for prevention of outbreaks due to wild poliovirus importations. Vaccine 2011; 29:3760–6. [PubMed: 21440639]
15. World Health Organization. Conclusions and recommendations of the Advisory Committe on Poliomyelitis Eradication. Wkly Epidemiol Rec 2010; 85:1–11.
16. UNAIDS/WHO Working Group on Global HIV/AIDS and STI Surveillance. Guidelines for Conducting HIV Sentinel Serosurveys among Pregnant Women and Other Groups. http://data.unaids.org/Publications/IRC-pub06/jc954-anc-serosurveys_guidelines_en.pdf. Accessed 20 December 2013.
17. Report on the 2010 National HIV Sentinel Survey: HIV prevalence rate in pregnant woman, biannual survey 1992–2010, Namibia. Windhoek: Ministry of Health and Social Services, Republic of Namibia, 2010 http://www.mhss.gov.na/documents/hiv_aids/National%20HIV%20Sentinel%20Survey%202010.pdf. Accessed 20 December 2013.
18. World Health Organization. Manual for the virological investigation of polio EPI/GEN/97.01.EPV1. Geneva: World Health Organization, 1997.
19. Report of a WHO informal consultation on polio neutralization antibody assays, Nashville, 5–6 December 1991 WHO/EPI/RD/91.3 Rev 1. Geneva: World Health Organization 1991.
20. Kärber G Archiv fuer experimentelle pathologie und pharmakologie 1931; 162:480–3.
21. Affanni P, Veronesi L, Rizziero S, et al. Status of immunity against poliomyelitis: a study among European and extra-European young immi-grants living in Parma. Acta Biomed 2005; 76:157–63. [PubMed: 16676565]

22. Calderon-Margalit R, Sofer D, Gefen D, et al. Immune status to poliovirus among immigrant workers in Israel. Prev Med 2005; 40: 685–9. [PubMed: 15850865]

23. Baldo V, Baldovin T, Cocchio S, et al. Seroepidemiology of polioviruses among university students in northern Italy. Clin Vaccine Immunol 2012; 19:1292–5. [PubMed: 22739695]

24. de Melker HE, van den Hof S, Berbers GA, et al. Evaluation of the national immunisation programme in the Netherlands: immunity to diphtheria, tetanus, poliomyelitis, measles, mumps, rubella and Haemophilus influenzae type b. Vaccine 2003; 21:716–20. [PubMed: 12531347]

25. Frantzidou F, Diza E, Halkia D, Antoniadis A. A seroprevalence study of poliovirus antibody in the population of northern Greece. Clin Microbiol Infect 2005; 11:68–71. [PubMed: 15649308]

26. Pires de Miranda M, Carmo Gomes M, Rebelo de Andrade H. Seropre-valence of antibodies to poliovirus in individuals living in Portugal, 2002. Euro Surveill 2007; 12:E7–8.

27. Signorini L, Barbi M, Matteelli A, et al. Prevalence of anti-poliovirus type 1, 2 and 3 antibodies in unvaccinated Italian travelers. J Travel Med 2004; 11:34–6. [PubMed: 14769285]

28. Wicker S, Rabenau HF, Gottschalk R, et al. Seroprevalence of vaccine preventable and blood transmissible viral infections (measles, mumps, rubella, polio, HBV, HCV and HIV) in medical students. Med Microbiol Immunol 2007; 196:145–50. [PubMed: 17273881]

29. Pfrez MC, Olivera I, Diabarboure H, et al. Seroprevalence of anti-polio antibodies in a population 7 months to 39 years of age in Uruguay: Implications for future polio vaccination strategies. Vaccine 2009; 27:2689–94. [PubMed: 19428880]

30. Sutter RW, Kew O, Cochi SL. Poliovirus vaccine—live In: Plotkin SA, Orenstein WA, eds. Vaccines. 4th ed, ch 26. Philadelphia: W. B. Saunders, 2004.

31. Sutter RW, Patriarca PA, Cochi SL, et al. Outbreakof paralytic poliomyelitis in Oman: evidence for widespread transmission among fully vaccinated children. Lancet 1991;338:715–20. [PubMed: 1679866]

32. Pregliasco F, Minolfi V, Boschin A, et al. A seroepidemiologic survey of immunity against poliomyelitis in a group of HIV positive and HIV negative drug addicts. Eur J Epidemiol 1995; 11:693–5. [PubMed: 8861854]

33. Gnanashanmugam D, Troy SB, Musingwini G, et al. Immunologic response to oral poliovaccine in human immunodeficiencyvirus-infected and uninfected Zimbabwean children. Pediatr Infect Dis J 2012; 31:176–80. [PubMed: 22146742]

34. Troy SB, Musingwini G, Halpern MS, et al. Vaccine poliovirus shedding and immune response to oral polio vaccine in HIV-infected and -uninfected Zimbabwean infants. J Infect Dis 2013; 208:672–8. [PubMed: 23661792]

35. Khetsuriani N, Helfand R, Pallansch M, et al. Limited duration of vaccine poliovirus and other enterovirus excretion among human immunodeficiency virus infected children in Kenya. BMC Infect Dis 2009 http://www.biomedcentral.com/content/p.df/1471-2334-9-136.pdf.

36. Meeting of the Strategic Advisory Group of Experts on Immunization, April 2012—conclusions and recommendations. Wkly Epidemiol Rec 2012; 87:201–16. http://wwwwhoint/wer. [PubMed: 24340402]

37. Global Polio Eradication Initiative. Polio Eradication and Endgame Strategic Plan: 2013–2018, 2013 http://wwwpolioeradicationorg/Portals/0/Document/Resources/StrategyWork/EndgameStratPlan_20130409_ENGpdf. Accessed 6 December 2013.

Table 1.

Overall and Type-Specific Poliovirus Seroprevalence Among Pregnant Women Aged 15–44 Years, Namibia, 2010

Poliovirus Type	Females, No. (%)	95% CI, %
Positive for type 1 (n = 2061)	1954(94.6)	93.6–95.5
Positive for type 2 (n = 2060)	2000(97.0)	96.1–97.6
Positive for type 3 (n = 2061)	1762(85.1)	83.5–86.6
Positive for any type	2042(99.1)	98.6–99.4
Positive for any type	1671 (80.6)	78.8–82.2
Negative for all types	18 (0.9)	.6–1.4

Abbreviation: CI, confidence interval.

Table 2.

Seroprevalence of Poliovirus Types 1–3 (PV1–3) Among Pregnant Females Aged 15–44 Years, by Age Group, Residence, Gravida, and Facility Type, Namibia, 2010

	PV1 (n = 2061)		PV2 (n = 2060)		PV3 (n = 2061)	
Variable	**Females, No. (%)**	**95% CI, %**	**Females, No. (%)**	**95% CI, %**	**Females, No. (%)**	**95% CI, %**
Age, y						
15–19	346 (94.6)	91.8–96.5	352 (96.5)	94.7–97.7	310 (84.7)	80.7–88.1
20–24	343 (93.0)	90.6–94.7	356 (96.5)	94.6–98.4	308 (83.5)	80.3–86.2
25–29	351 (96.2)	94.0–97.5	354 (97.0)	95.6–98.9	317 (86.8)	83.5–89.6
30–34	352 (95.4)	92.6–97.2	361 (97.8)	95.8–99.1	316 (85.6)	81.5–88.9
35–39	352 (94.4)	90.3–96.8	365 (97.9)	94.8–99.1	313 (84.0)	78.3–88.4
40–44	210 (95.8)	88.7–98.5	212 (96.8)	90.1–99.0	198 (90.4)	81.6–95.2
Residence						
Urban	864 (94.7)	93.1–95.9	884 (96.7)	95.3–97.7	771 (84.1)	81.6–86.3
Rural	1090 (94.6)	93.1–95.8	1116 (97.2)	96.1–98.0	991 (86.0)	83.9–88.0
Gravidity						
1	531 (93.2)	91.0–94.8	542 (95.7)	93.9–97.0	464 (81.2)	78.0–83.9
2	381 (96.0)	93.8–97.3	392 (98.9)	97.5–99.5	343 (86.1)	82.3–88.9
3	342 (96.1)	93.6–97.6	344 (96.1)	93.7–97.7	304 (87.2)	83.4–90.2
≥4	700 (94.3)	91.9–96.0	722 (97.4)	95.6–98.5	651 (87.9)	84.8–90.5
Facility type						
Hospital	237 (95.9)	92.6–97.7	242 (97.0)	94.0–98.5	210 (85.2)	80.2–89.0
Health center	282 (95.3)	92.3–97.2	288 (97.6)	95.1–98.8	258 (86.9)	82.6–90.3
Clinic	1435 (94.3)	93.0–95.3	1470 (96.8)	95.8–97.6	1294 (84.8)	82.9–86.5

Abbreviation: CI, confidence interval.

Table 3.

Seroprevalence of Poliovirus Types 1–3 (PV1–3) Among Pregnant Women Aged 15–44 Years, by Human Immunodeficiency Virus (HIV) Infection Status and Age Group, Namibia, 2010

	HIV Positive (n = 435)		HIV Negative (n = 1627)		
Age in y, by PV Type	**Females, No. (%)**	**95% CI, %**	**Females, No. (%)**	**95% CI, %**	***P* Value**
PV1					
Overall	398 (91.8)	88.7–94.2	1556 (95.3)	94.2–96.2	.0015
15–19	21 (84.0)	64.4–93.8	325 (95.3)	92.5–97.1	
20–24	44 (93.6)	85.7–97.3	299 (92.9)	90.4–94.8	
25–29	76 (92.7)	86.2–96.2	275 (97.2)	95.0–98.4	
30–34	100 (90.9)	83.7–95.1	252 (97.3)	94.3–98.7	
35–39	110 (94.0)	84.9–97.8	242 (94.5)	89.4–97.2	
40–44	47 (87.0)	65.7–95.9	163 (98.8)	91.5–99.8	
PV2					
Overall	421 (96.4)	94.0–97.8	1579 (97.1)	96.2–97.8	.2588
15–19	22 (88.0)	69.1–96.0	330 (97.1)	94.7–98.4	
20–24	45 (95.7)	88.6–98.5	311 (96.6)	94.7–97.8	
25–29	79 (96.3)	91.0–98.6	275 (97.2)	95.0–98.4	
30–34	107 (97.1)	91.9–99.1	254 (98.1)	95.4–99.2	
35–39	115 (98.3)	91.0–99.7	250 (97.7)	93.6–99.2	
40–44	53 (98.2)	80.3–99.9	159 (96.4)	87.8–99.0	
PV3					
Overall	353 (80.0)	60.0–91.4	1409 (86.1)	84.4–87.7	.0027
15–19	20 (80.0)	64.3–95.7	290 (85.0)	80.9–88.5	
20–24	38 (80.9)	70.6–88.1	270 (83.9)	80.5–86.7	
25–29	65 (79.3)	70.8–85.8	252 (89.1)	85.5–91.8	
30–34	90 (81.8)	73.1–88.2	226 (87.3)	82.4–90.9	
35–39	94 (80.3)	68.5–88.5	219 (85.6)	78.8–90.4	
40–44	46 (85.2)	63.5–95.0	152 (92.1)	82.1–96.8	

Abbreviation CI, confidence interval.