

UPDATE ON POLIO ERADICATION IN AFRICA

Nicksy Gumede-Moeletsi, Melinda Suchard, Barry Schoub

Centre for Vaccines and Immunology, NICD
Division of Virology and Communicable Diseases Surveillance, School of Pathology, University of Witwatersrand, Johannesburg

Background

The Global Poliomyelitis Eradication Initiative (GPEI) was initiated in 1988, after the declaration of the eradication of smallpox in 1980 by the World Health Assembly, which now aims to eradicate poliomyelitis globally by 2018.¹ The GPEI has been successful in reducing poliomyelitis cases: from 350,000 recorded in 1988 to fewer than 1000 cases recorded by the end of 2011. Pakistan, Afghanistan and Nigeria are the only remaining endemic countries.²

Monitoring for the presence of polio is based on acute flaccid paralysis (AFP) surveillance. In order to ensure that a polio case is not missed, AFP surveillance targets a symptom as opposed to a specific disease.³ The clinical case definition of AFP is an acute onset of flaccid paralysis or paresis in any child under 15 years of age. Differential diagnoses for AFP include Guillain-Barre syndrome, transverse myelitis, enterovirus infections and traumatic neuritis. In general, AFP surveillance is a GPEI strategy designed to detect poliovirus circulation, re-importation of wild poliovirus into polio-free areas or regions and emerging vaccine derived polio viruses (VDPVs). As polio eradication is approaching, it is crucial to maintain high quality AFP surveillance

www.hpsc.ie/hpsc/A-Z/VaccinePreventable/Polio/.../File,2461,en.pdf.

Functions of the NICD national and regional reference laboratory

Since 1995, the molecular polio unit of the National Institute for Communicable Diseases (NICD) of South Africa has hosted a WHO-supported AFP surveillance

network at both national and regional levels. At national level, the NICD serves seven countries including South Africa, Angola, Botswana, Lesotho, Mozambique, Namibia and Swaziland. The molecular polio unit also serves as a reference laboratory for many of the countries outside of the southern African region which fall under the WHO African Regional Office. Sequence analyses conducted at the molecular polio unit have been used to answer several epidemiological questions regarding the likely location of endemic poliovirus reservoirs and patterns of virus transmission. These analyses have also been used to determine if an unknown viral isolate is similar to endemic strains or has been introduced (imported).

AFP Surveillance in South Africa

The last wild polio virus in South Africa was detected in 1989. The criterion for adequate surveillance of AFP is 2 cases per 100 000 population of children less than 15 years of age coupled with an isolation rate of non-polio enteroviruses from AFP cases of greater than 10%. A total of 866 specimens from South African AFP cases was received in 2012. From this sample, adequate surveillance with an overall Non-Polio AFP rate of 2.0 per 100 000 children and a non polio enterovirus isolation rate of 12.5% was demonstrated (figure 1).

Wild-type polio viruses in Africa

During 2012, 352 poliovirus isolates were characterized as vaccine or wild-type. These isolates were sent to the NICD from National and Regional laboratories throughout Africa namely: Central African Republic, Ethiopia, Ghana, Kenya, Madagascar, Democratic Republic of Congo, Senegal, South Africa, Uganda and Zambia (figure 2).

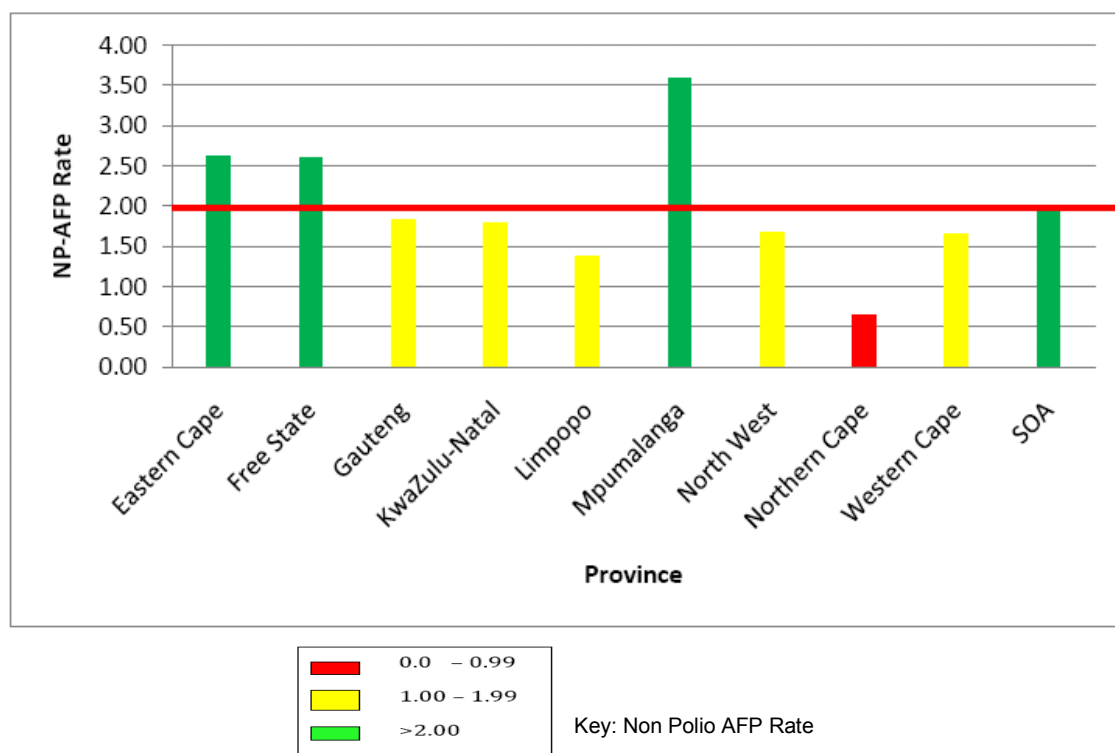


Figure 1: Annualised Non-Polio AFP rate per 100 000 children < 15 yrs of age by province, South Africa. Source: Directorate: Child and Youth Health; Sub-Directorate: Expanded Programme on Immunization (EPI; National Department of Health, South Africa). SOA = South Africa.

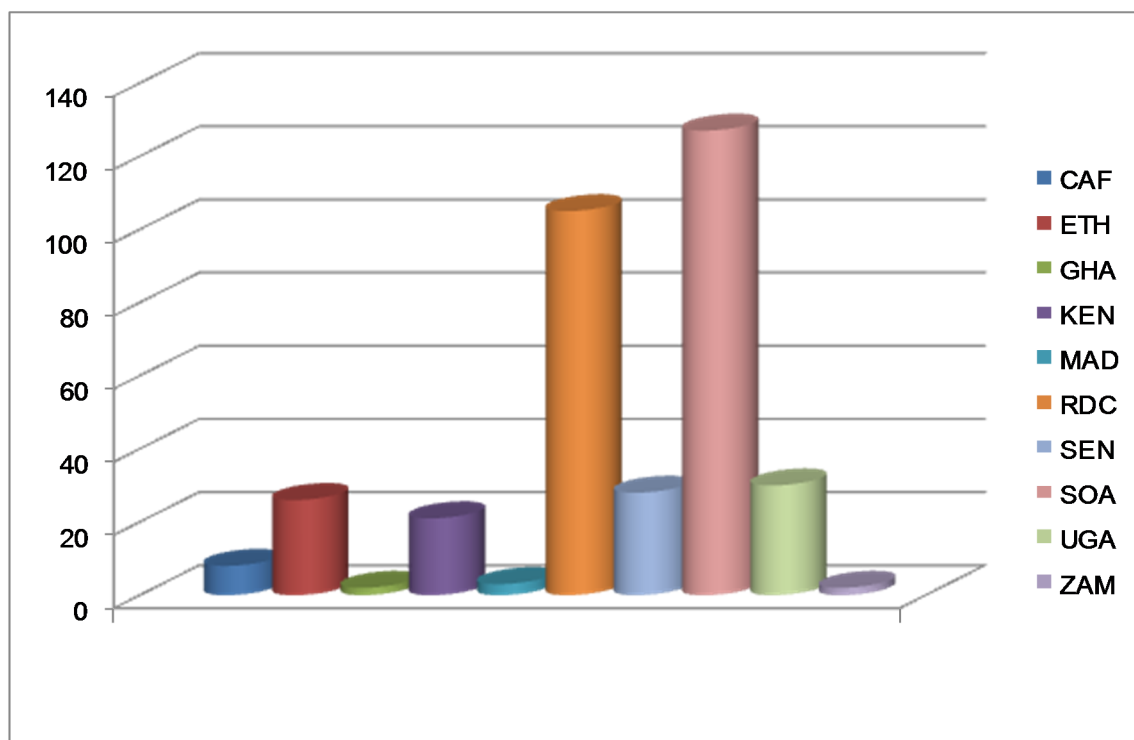


Figure 2: Number of poliovirus isolates from countries served by the AFP surveillance network of South Africa in 2012. Central African Republic (CAF), Ethiopia (ETH), Ghana (GHA), Kenya (KEN), Madagascar (MAD), Democratic Republic of Congo (DRC), Senegal (SEN), South Africa (SOA), Uganda (UGA) and Zambia (ZAM).

For wild-type poliovirus 3 (PV3), only Nigeria reported the WEAf-B wild poliovirus type 3 in 2012, with a total of 19 cases compared to 13 cases in 2011. The genetic clusters circulating the most were F4 and F6 affecting seven Nigerian states namely: Bauchi, Borno, Jigawa,

Kaduna, Kano, Taraba and Yobe. The F4 cluster was dominant in Borno state (data not shown) while F6 affected the rest of the states (figure 4). Serotype 3 polioviruses from environmental samples were also reported.

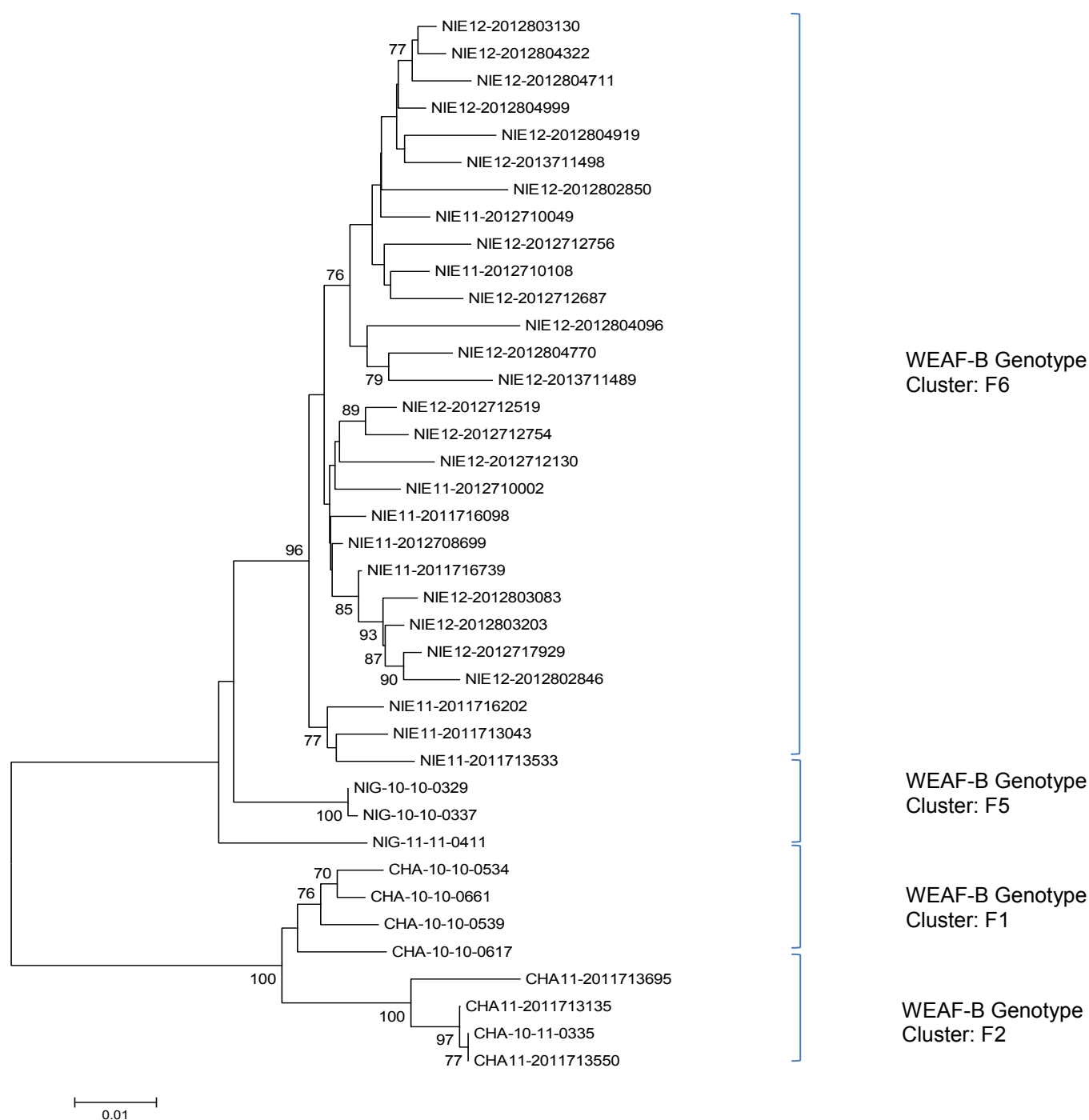


Figure 4: Neighbour-joining tree of a VP1 gene of WEAf-B wild PV3 representatives of isolates from Africa. Bootstrap values of greater than 70% are shown at the branch nodes. NIE = Nigeria, NIG = Niger, CHA = Chad.

Identification of wild poliovirus type 1 in Somalia in 2013

Only three countries reported wild polioviruses (WPV) in 2012 (Nigeria, Niger and Chad) compared to 13 countries in 2011. On 18th April 2013, a 32-month-old girl from Banadir region in Somalia presented with onset of paralysis. This case represents the first wild polio virus in Somalia since March 2007 and the first outbreak outside of an endemic country in 2013. In large areas of south-central Somalia, immunization campaigns have not been implemented since 2009 due to inaccessibility, affecting more than 500,000 children aged <5 years. Populations in this area are at high risk of polio as a consequence of this outbreak. This area is also affected by an ongoing circulating Vaccine Derived Poliovirus type 2 (cVDPV 2) outbreak, which has resulted in 18 cases in the country since 2009 (the most recent cVDPV2 case had onset of paralysis on 9th January 2013).

Characterisation of circulating vaccine-derived polioviruses in Africa

Live, attenuated oral poliovirus vaccine (OPV) is still the vaccine of choice for developing countries. However, reversion to virulence may occur during OPV replication in humans, resulting in person-to-person transmission and circulation of vaccine-derived polioviruses (cVDPV) in areas with low rates of vaccine coverage.⁴ Vaccine-derived polioviruses show significant sequence drift (> 1% nucleotide difference in types 1 and 3, > 0.6% nucleotide difference in type 2), indicating prolonged replication of the vaccine strain in human populations and consequent changes in the phenotypic properties of neurovirulence and transmissibility.^{4,5} Poliomyelitis outbreaks associated with cVDPVs have been reported in several countries including Egypt (1982-1993), Haiti (2000-2001), Dominican Republic (2000-2001),

Philippines (2001), Madagascar (2002 and 2005), China (2004), Cambodia (2005-2006), Indonesia (2005) and Nigeria (2005-2010).⁵⁻¹² In 2012, five African countries reported cVDPV type 2 including Chad, Somalia, Nigeria, Kenya and the DRC. Seventeen cases were reported in the DRC in 2012 compared to eleven cases recorded in 2011. The DRC outbreak has been continuous since 2008. The cVDPVs detected in DRC are associated with the two most important biological properties of wild polioviruses namely (i) the capacity to cause paralytic disease in humans and (ii) the capacity for continuous person-to-person transmission.

As a result of accumulating evidence about the emergence and spread of cVDPV, there are plans for synchronized cessation of the use of OPV and the implementation of more widespread use of inactivated polio vaccine (IPV).¹³⁻¹⁵ A better understanding of VDPV persistence and circulation is important for decision making about when and how to stop immunization with OPV after the global eradication of wild polioviruses.¹⁶⁻¹⁸ South Africa is the first African country to introduce IPV into its routine immunization schedule.

Acknowledgements

We thank the NICD Polio working group (Raffaella Williams, Lerato Seakamela, Wayne Howard, Shelina Moonsamy, Portia Ngcobondwana, Heleen Du Plessis, Doris Lebambo, Elliot Motaung, Megan Vandecar, Cynthia Simelane, Abraham Sehata and Sibulelo Sidumo), the Polio working group at the Centers for Disease Control and Prevention in Atlanta, USA and the Global and African Polio network for technical support. This project was funded by the World Health Organization as well as the NICD/NHLS.

References

1. Centers for Disease Control and Prevention. "Update on vaccine-derived polioviruses-worldwide. January 2008-June2009." MMWR Morb Mortal Wkly Rep. 2009a; 58(36):1002-6
2. Centers for Disease Control and Prevention. "Update on vaccine-derived polioviruses". MMWR Morb Mortal Wkly Rep. 2006c; 55(40):1093-7
3. Centers for Disease Control and Prevention." Progress towards poliomyelitis eradication-African Region, 1999-March2000". JAMA. 2000a; 284(14):1781-2.
4. Kew OM, Sutter RW, de Gourville EM, Dowdle WR, Pallansch MA. Vaccine-derived polioviruses and the end-game strategy for global polio eradication. Annu Rev Microbiol. 2005;59:587-635.
5. http://www.polioeradication.org/Portals/0/Document/Resources/GPLN_publications/GPLN_Meeting_recommendations_2010.pdf
6. Liang X, Zhang Y, Xu W, Wen N, Zuo S, Lee LA, et al. An outbreak of poliomyelitis caused by type 1 vaccine-derived poliovirus in China. The Journal of Infectious Diseases. 2006 Sep 1;194(5):545-51.
7. Tong YB, Zhang DY, Zou J, Zhang L, Yu H, Liu M. An epidemiological study on vaccine derived polio virus circle in Zhenfeng County of Guizhou Province. Zhonghua yu fang yi xue za zhi. Chinese Journal of Preventive Medicine. 2005 Sep;39(5):321-3.
8. Centers for Disease Control and Prevention. Global update on vaccine-derived polioviruses, January 2006-August 2007. Weekly Epidemiological Record 2007 Sep 28;82(39):337-43.
9. Estivariz CF, Watkins MA, Handoko D, Rusipah R, Deshpande J, Rana BJ, et al. A large vaccine-derived poliovirus outbreak on Madura Island--Indonesia, 2005. The Journal of Infectious Diseases. 2008 Feb 1;197(3):347-54.
10. Jenkins HE, Aylward RB, Gasasira A, Donnelly CA, Mwanza M, Corander J, et al. Implications of a circulating vaccine-derived poliovirus in Nigeria. The New England Journal of Medicine. Jun 24;362(25):2360-9.
11. Wassilak S, Pate MA, Wannemuehler K, Jenks J, Burns C, Chenoweth P, et al. Outbreak of type 2 vaccine-derived poliovirus in Nigeria: emergence and widespread circulation in an underimmunized population. The Journal of Infectious Diseases. Apr 1;203(7):898-909.
12. Kew O, Morris-Glasgow V, Landaverde M, Burns C, Shaw J, Garib Z, et al. Outbreak of poliomyelitis in Hispaniola associated with circulating type 1 vaccine-derived poliovirus. Science (New York, NY). 2002 Apr 12;296(5566):356-9.
13. Rousset D R-AM, Razafindratsimandresy R, Randriamanalina B, Guillot S, Balanant J, et al. Recombinant vaccine-derived polioviruses in Madagascar. Emerging Infectious Diseases. 2003;9:885-7.
14. Yang CF, Naguib T, Yang SJ, Nasr E, Jorba J, Ahmed N, et al. Circulation of endemic type 2 vaccine-derived poliovirus in Egypt from 1983 to 1993. Journal of Virology. 2003 Aug;77(15):8366-77.
15. Jarzabek Z. [End phase challenges of poliomyelitis eradication programme realization]. Przegląd Epidemiologiczny. 2005;59(1):59-68.
16. "Endgame" issues for the global polio eradication initiative. Clin Infect Dis. 2002 Jan 1;34(1):72-7. Epub 2001 Nov 19.
17. Wood DJ, Sutter RW, Dowdle WR. Stopping poliovirus vaccination after eradication: issues and challenges. Bull World Health Organ. 2000;78(3):347-57.
18. Fine PE, Carneiro IA. Transmissibility and persistence of oral polio vaccine viruses: implications for the global poliomyelitis eradication initiative. Am J Epidemiol. 1999 Nov 15;150(10):1001-21.