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Analysis of population immunity to poliovirus following cessation of trivalent oral polio vaccine ⋆

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Abstract

Background: The global withdrawal of trivalent oral poliovirus vaccine (OPV) (tOPV, containing Sabin poliovirus strains serotypes 1, 2 and 3) from routine immunization, and the introduction of bivalent OPV (bOPV, containing Sabin poliovirus strains serotypes 1 and 3) and trivalent inactivated poliovirus vaccine (IPV) into routine immunization was expected to improve population serologic and mucosal immunity to types 1 and 3 poliovirus, while population mucosal immunity to type 2 poliovirus would decline. However, over the period since tOPV withdrawal, the implementation of preventive bOPV supplementary immunization activities (SIAs) has decreased, while outbreaks of type 2 circulating vaccine derived poliovirus (cVDPV2) have required targeted use of monovalent type 2 OPV (mOPV2).

Methods: We develop a dynamic model of OPV-induced immunity to estimate serotype-specific, district-level immunity for countries in priority regions and characterize changes in immunity since 2016. We account for the changes in routine immunization schedules and varying

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CRediT authorship contribution statement

Arend Voorman: Conceptualization, Methodology, Software. **Hil Lyons:** Conceptualization, Methodology, Software. **Caroline Bennette:** Methodology. **Stephanie Kovacs:** Methodology. **Jeevan K. Makam:** Supervision. **John Vertefeuille:** Supervision. **Graham Tallis:** Conceptualization, Supervision.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary material

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implementation of preventive and outbreak response SIAs, assuming homogenous coverages of 50% and 80% for SIAs.

Results: In areas with strong routine immunization, the switch from tOPV to bOPV has likely resulted in gains in population immunity to types 1 and 3 poliovirus. However, we estimate that improved immunogenicity of new schedules has not compensated for declines in preventive SIAs in areas with weak routine immunization. For type 2 poliovirus, without tOPV in routine immunization or SIAs, mucosal immunity has declined nearly everywhere, while use of mOPV2 has created highly heterogeneous population immunity for which it is important to take into account when responding to cVDPV2 outbreaks.

Conclusions: The withdrawal of tOPV and declining allocations of resources for preventive bOPV SIAs have resulted in reduced immunity in vulnerable areas to types 1 and 3 poliovirus and generally reduced immunity to type 2 poliovirus in the regions studied, assuming homogeneous coverages of 50% and 80% for SIAs. The very low mucosal immunity to type 2 poliovirus generates substantially greater risk for further spread of cVDPV2 outbreaks. Emerging gaps in immunity to all serotypes will require judicious targeting of limited resources to the most vulnerable populations by the Global Polio Eradication Initiative (GPEI).

Keywords

Polio; Population immunity; Disease Modeling

1. Introduction

Managing population immunity to the three poliovirus serotypes is a key programmatic function of the Global Polio Eradication Initiative (GPEI). Routine immunization (RI) delivery through primary health care systems according to age-based schedules is the first line of defense in protection from poliovirus. The vaccination activities supported by the GPEI are mass vaccination campaigns with oral poliovirus vaccines (OPV, containing Sabin poliovirus strains), called supplementary immunization activities (SIAs), typically directed toward under-immunized groups of children under the age of 5 years [1] for prevention, or interruption of active poliovirus circulation.

There are multiple formulations of OPV developed to address the different epidemiologic situations, including monovalent vaccines (mOPV) for each serotype, bivalent vaccine (bOPV) containing serotypes 1 and 3, and trivalent OPV (tOPV) containing serotypes 1, 2, and 3. Trivalent inactivated polio vaccine (IPV) is also used worldwide to provide serologic protection from paralysis from any of the three serotypes.

In addition to their use directed toward interruption of transmission of endemic type 1 wild poliovirus (WPV), SIAs supplement routine immunization with two other goals: outbreak response and outbreak prevention. Outbreak response SIAs address the acute need to control active poliovirus circulation, mitigating outbreak spread and protecting from further paralysis. Preventive SIAs address immunity gaps in uninfected communities to prevent outbreaks and mitigate potential outbreak size, either from WPV or vaccine-derived poliovirus (VDPV). Prevention and termination of poliovirus outbreaks are important

activities as progress is made toward global WPV eradication, as delays to eradication become costly and RI alone is insufficiently implemented in many areas to maintain high immunity.

Following the certification of the global eradication of WPV type 2 in 2015[2], the GPEI in 2016 coordinated a major change in vaccination schedules with the global withdrawal of tOPV from routine immunization, and substitution with multiple doses of bOPV and a single dose of IPV into RI schedules. bOPV and IPV were expected to improve population serologic and mucosal immunity to types 1 and 3 poliovirus, with the recognition that population mucosal (transmission blocking) immunity to type 2 poliovirus would decline. The addition of one dose of IPV was aimed to provide some serologic protection from paralysis following infection by type 2 poliovirus. However, over the period since tOPV withdrawal, the implementation of preventive bOPV SIAs has decreased, while the introduction of IPV was delayed in some countries and has not achieved high coverage in many areas [3]. Outbreaks of cVDPV2 have required targeted use of monovalent type 2 OPV (mOPV2) from the global stockpile in outbreak response SIAs.

Understanding immunity at subnational spatial scales is important for polio program planning, particularly in light of changes to the polio vaccination schedule. Immunity or vaccination coverage estimates that identify susceptible populations regularly serve as inputs into risk modeling and SIA planning within GPEI [4-9].

Several methods have been used to estimate population immunity for risk modeling [6,10,11]. These methods often use reported dose histories collected during investigations of acute flaccid paralysis (AFP) case surveillance data with doses attributed to different vaccine formulations that were employed in SIAs in that region and time. Estimated per-dose effectiveness by vaccine serotype is applied to this attribution, optionally with spatiotemporal smoothing methods to address the sparsity of AFP cases. These methods often aggregate data within time intervals, and thus becomes less temporally responsive to individual SIAs.

Alternative methods have sought to estimate the implied coverage of SIAs by the combination of dose history reporting and SIA exposures, which may then be applied to understand the impact of SIAs on population immunity in a way that is sensitive to the timing of these SIAs [7,12,13]. An analysis has shown that dose histories from AFP surveillance data are poorly related to SIA exposures for many countries, implying that the dose histories are not a universally reliable gauge of population immunity even apart from their low sampling rate [5]. In addition, in many countries of interest, the histories of doses received through RI and SIAs are not reported separately, complicating both attribution to vaccine serotype and SIA coverage estimates. There is also the potential of recall and/or field investigator biases of the SIA doses obtained for the child as SIA doses are not documented.

We describe a novel synthesis of data sources to create an OPV-induced immunity construct for all three serotypes that is temporally responsive to SIAs, explicit with respect to vaccine formulations used in SIAs and RI, of sub-national resolution, and regional in scope. It is

suitable for identifying emerging susceptibility gaps at subnational scales, estimation of SIA requirements, and as temporally relevant inputs into spatiotemporal risk prediction. We apply this construct to examine changes in immunity by serotype due to the tOPV to bOPV vaccine switch and include the subsequent extensive responses to cVDPV2 outbreaks with mOPV2 SIAs.

2. Methods

Population mucosal immunity is estimated using a simplified variant of a model developed for Nigeria, and subsequently used in recent immunity estimates for SIA planning [12]. We ignore any impact of IPV on population mucosal immunity, assuming that it provides little to no mucosal immunity alone and subsequently negligible transmission blocking. The below lists details of the assumptions and calculations.

2.1. Age groups

We estimate immunity among 6–36-month-olds, among which most (77%) of polio cases occur, assuming a uniform-distributed age distribution. Other age groups may be of interest, and as a sensitivity analysis, we also considered immunity among children aged 6–59 month, and immunity weighted by the age distribution of polio cases (mean age of 21 months, data in the 2000–2019 World Health Organization AFP dataset, analysis available on request).

2.2. SIA coverage

The method developed for Nigeria used age-specific SIA coverage rates estimated from dose histories collected during investigations of AFP cases which are classified as non-polio (NP-AFP) based on laboratory testing and other criteria. However, these are not valid or comparable in many countries, and so we assume coverage scenarios, applying within each scenario a common coverage across all countries [5]. We estimate immunity in a well-served population with 80% SIA coverage, and an under-served population with 50% SIA coverage, and where possible, express a range showing immunity under these alternate coverage scenarios. Averaging these estimates can be seen as a method to account for nonindependence of SIA coverage.

2.3. Vaccine effectiveness

Numerous studies have estimated serotype-specific per-dose effectiveness against disease of OPV formulations used in endemic/outbreak settings from a case-control approach using surveillance data, and these estimates can be lower than serotype-specific per-dose seroconversion data from clinical-trials [11,13-16]. We assume that serotype-specific per-dose effectiveness is the same in SIAs and RI. We have taken a logic-driven consensus of these various estimates in the Table 1 below, with an ordering of immunogenicity, high to low, of type 2, type 1, and type 3.

2.4. Routine immunization

RI coverage was obtained from the Institute for Health Metrics and Evaluation’s (IMHE) local burden of disease program, which provides estimates of reported DTP1 and DTP3

coverage (diphtheriatetanus-pertussis vaccine dose series number) in 5x5 km cells, based on geo-located survey data [17]. These are summarized at the district level by taking a population-weighted average using WorldPop estimates of population of the under-5 population [18]. DTP2 is estimated as the average of DTP1 and DTP3. Unpublished updates were provided by the authors for all countries in the World Health Organization (WHO) African Region (AFR) and Eastern Mediterranean Region (EMR), covering RI data from 2000 through 2018. This allows harmonization of old survey information with new geographical boundaries, so that immunity of a population living in each area can be tracked over time. We use DTP coverage estimates, since DTP is given at the same time in the routine childhood immunization schedule as OPV, which avoids the confusion of OPV doses administered by RI and SIAs among surveys that rely on parental recall. Coverage estimates for subsequent years use the last observation carried forwards.

We account for dropout by adding the effectiveness achieved by those who received exactly 1, 2, or 3 doses of OPV in routine, assuming independent probability of mucosal immunity for each dose:

$$r_{tj} = dpt3_{tj} * \left(1 - (1-\phi)^3\right) + (dpt2_{tj} - dpt3_{tj}) * \left(1 - (1-\phi)^2\right) + (dpt1_{tj} - dpt2_{tj}) * \phi$$

where r_{tj} is the RI-induced immunity for a cohort born in year t in the j^{th} district, ϕ is the per-dose effectiveness of the vaccine used in RI at the time of the child's birth (tOPV prior to April 2016, and bOPV after), and DTP1, DTP2, and DTP3_j are the estimates of the proportion of children receiving 1, 2, or 3 doses of DTP vaccine in the j^{th} district.

2.5. SIA calendar

The SIA calendar is obtained from GPEI's Polio Information System (POLIS). SIAs were harmonized across years so that plausible SIA histories could be obtained for current administrative divisions vs. previous. Where geography of an SIA activity was ambiguous, fractional SIAs were counted according to the percentage of the population targeted. SIAs that occurred after March 15, 2020 or were otherwise explicitly labeled as cancelled or delayed are removed.

2.6. Geography of interest

We aim to provide immunity estimates for countries in priority WHO regions (AFR and EMR) at the district level, for 2010 through 2020. When required for ease of visualization, we display immunity at the province level by taking a population-weighted average of district estimates. Note that since DTP is not available reliably through IHME for Saudi Arabia and Qatar, they are removed from estimates. In accompanying files, we include Type 1 immunity estimates for Pakistan and Afghanistan but note that there are special considerations there (accessibility in Afghanistan, larger underserved subpopulations in Pakistan) which may not be well-represented by the assumptions of this model. Geographical boundary data for the analysis was provided by the WHO, maintained for GPEI programmatic purposes.

2.7. Estimating immunity

For population j, let $t_1, \cdots, t_N$ be the dates of SIAs, and let ϕ_{jt_k} be the effectiveness – the probability of seroconversion/induction of mucosal immunity – of the vaccine used in the campaign at time t_k and p_{jt_k} be the associated campaign coverage. Assuming that age-eligible children are independently covered by a campaign at time t_k and seroconversion given a dose occurs independently, we estimate immunity for a child of age a as

$$I(t, a, j) = 1 - (1 - r_{t-aj}) \prod_{k: t-a < t_k < t} (1 - p_{jt_k} \phi_{jt_k}).$$

Immunity in the jth district at a point in time can then be calculated by integrating over an age distribution A for the population of interest $I(t, j) = \int I(t, a, j) dA(a)$. Generally, population mucosal immunity for an aggregate area is a population-weighted average of the immunity in each sub-area or sub-population, for example aggregation to a province level from districts. For a single coverage scenario, we assume that campaign coverage is constant over time (t_k) and by geography (j).

3. Results

Fig. 1 illustrates the results of the immunity estimation method applied to a single district in Banadir, Somalia, and the consequences of SIAs with varying OPV formulations and estimated SIA coverage. Prior to 2013, most SIAs used tOPV and immunity of the three serotypes thus immunity of the three serotypes exhibited identical patterns of spiking after SIAs followed by gradual reductions as infants with no SIA exposure replace older children in the age group of study. Following a WPV1 outbreak in 2013 nearly exclusive use of bOPV resulted in decline of immunity to type 2 and increases in immunity to types 1 and 3. A series of tOPV campaigns in 2015–2016 prior to its withdrawal increased type 2 immunity, after which it fell precipitously until an outbreak of cVDPV2 in 2017 (in continuous, previously undetected circulation after 2013) required mOPV2 use. Thereafter type 2 immunity fell rapidly. In November 2019, cVDPV2 was detected once again requiring additional outbreak response with mOPV2. During this post-switch period, less frequent implementation of preventive bOPV SIAs have resulted in steady declines in types 1 and 3 immunity, despite the higher immunogenicity of bOPV for these serotypes relative to tOPV. When using different coverage assumptions, the estimates follow the same trends and are similar when there are frequent SIAs or few SIAs.

The reduction in preventive vaccine usage noted above is a general trend among SIAs in AFR and EMR, as indicated in Fig. 2. With the vaccine switch in 2016, use of tOPV ceased until recent use in Pakistan and Afghanistan to mitigate transmission of both endemic WPV1 and cVDPV2. All the tOPV SIAs in 2016 occurred in the first four months of the year, before the vaccine switch; this, as well as several SIAs in 2015, reflects a planned surge in tOPV campaigns in order to increase type 2 immunity in vulnerable countries in advance of withdrawal of type 2 containing vaccine. With implementation of bOPV and tOPV SIAs in 2016, over 1 billion doses of type 1-containing vaccine were distributed, with similar levels distributed during 2010 to 2015.

However, starting in 2016 we can see a marked decline in type 1-containing vaccine use in AFR (tOPV or bOPV), and rising use of mOPV2. This was driven by multiple emergences of cVDPV2 in the two regions, as well as subsequent expansion of those outbreaks. Type 1-containing vaccine use in EMR has not increased despite the presence of the two remaining endemic countries and risks of WPV1 importation. In addition, bOPV use in Pakistan in 2019 slowed due to resistance to SIAs in key geographies. Overall, due to these and other factors, use of type 1 containing vaccine in SIAs has dropped to less than 500 million doses in 2019 and 2020, at less than a third of the annual vaccine usage in the pre-switch period.

Fig. 3 and Fig. 4 illustrate the effect of these transitions on global and regional immunity to type 1 and type 2 poliovirus, as well as demonstrate the spatial resolution of the immunity estimates. Areas with low levels of RI coverage and reduced access to SIAs with bOPV (or tOPV) show a decline in type 1 immunity from 2016 to 2020. Angola is one such example, where cVDPV2 outbreaks in 2019, required prioritizing mOPV2 SIA in 2019 and 2020. Illustrated in Fig. 4, countries with relatively high RI cover-age and limited SIAs (Rwanda, for example), are estimated to have improved type 1 immunity due to a slighter higher assumed immunogenicity for bOPV over tOPV.

Fig. 3 also illustrates the expected, marked decline in type 2 immunity since 2016, and then the increases in areas induced by mOPV2 responses to outbreaks. For the age group 6–36 months there is no OPV-derived immunity to type 2 for vast areas of the two regions three years after the switch, creating conditions of general susceptibility. In contrast, areas that have recently received mOPV2 (such as Angola, DR Congo, or Nigeria) become regions of relatively high immunity, immunity that will again decline over time after exposure to vaccine, as seen in Somalia in Fig. 1. For example, two short-succession mOPV2 campaigns assuming 80% coverage with per-dose effectiveness of 0.7 will induce immunity near 80%, three campaigns near 90% immunity, and both diminishing to 0 by definition after three years for the age group 6–36 months.

Countries are often heterogeneous at sub-national levels with different RI coverage and SIA exposures. In Fig. 4 this heterogeneity is illustrated for the three most populous countries in the WHO African Region, the Democratic Republic of the Congo (DR Congo), Ethiopia, and Nigeria, with type 1 immunity for each province plotted under different assumptions of SIA coverage. This figure identifies some of the challenges in the management of population immunity at a subnational level.

First, it is apparent that heterogeneities in type 1 immunity are present for all three countries, most notably for Nigeria. Baseline heterogeneities are exacerbated by different SIA coverages, as demonstrated by the difference between the 50% and 80% coverage scenarios. As illustrated by the number of provinces above, say, the 90% immunity threshold, many provinces are robust to these different coverage scenarios, implying that SIA frequency in both scenarios was sufficient to supplement immunity from RI, fewer SIAs being necessary in areas with higher RI coverage.

Second, the threshold drawn – for example 80% or 90% immunity – interacts with the interpretation of immunity deficits. Assuming 80% coverage, most provinces exceed 80%

immunity, but many provinces in both scenarios and most in the 50% coverage scenario do not exceed 90% immunity. This underscores how assumptions driving immunity estimates interact with the program requirements for attaining given immunity levels: SIAs may be planned to meet immunity targets under certain coverage assumptions, but these coverage assumptions may not be accurate, and the immunity targets specified may or may not ultimately be sufficient to stop transmission of poliovirus. This is further complicated by obtaining accurate coverage assessments. Though most coverage surveys show relatively high SIA vaccination coverage, this often conflicts with epidemiological data or reported dose histories [12,19].

Last, displacement of bOPV SIAs either due to the need to execute mOPV2 SIA in outbreak response, funding limitations, supply constraints, and COVID restrictions results in immunity deficits for areas with poor RI, while areas with strong RI have generally seen improvements in immunity since 2016 due to more immunogenic vaccine routine vaccination (Fig. 5) In particular, DR Congo and Ethiopia saw marked declines in type 1 immunity in 2020 compared to past years.

4. Discussion

These results show that use of types 1- and 3-containing OPV in SIAs has declined appreciably in the WHO African and Eastern Mediterranean regions, resulting in declines in type 1 and type 3 immunity in many areas. In many vulnerable areas with low RI coverage, reductions in SIAs have negated a potential positive impact of replacing tOPV with bOPV and IPV due to higher immunogenicity for types 1 and 3, instead substantively increasing vulnerability to cVDPV1/cVDPV3 outbreaks and susceptibility to WPV1 importation. Some of the decline in type 1 and type 3 immunity is attributable to changes in SIA plans because of cVDPV2 epidemiology, as indicated by the increasing mOPV2 requirements for outbreak response SIAs since 2016. In some countries, the need to vaccinate with mOPV2 displaced preventive SIAs with bOPV, resulting in type 1 immunity deficits. Further, the larger than expected number and size of cVDPV2 outbreaks required reassignment of finances intended for SIAs in the two regions to mOPV2 outbreak response campaigns. The modeled type 1 immunity deficits are heterogeneous between and within countries based on variable RI coverage and SIA exposure.

As a central function of GPEI is management of population immunity to reduce poliovirus transmission risk and secure the global absence of poliovirus transmission, it is important to be able to identify emerging or chronic susceptibility gaps induced by the confluence of contributing factors. Our framework synthesizes the drivers of OPV-derived population immunity -- RI, SIA exposures, and vaccine choices -- in a spatially and temporally explicit way that can provide GPEI with timely updates to monitor the risks. This method can be used in turn to suggest the number of SIAs required to reach a given threshold of immunity, and has been used to do so for recent SIA prioritization exercises in the GPEI.

The existence and incorporation of high-resolution, rasterized time series of RI estimates admits flexible summarization that can adapt to changes in subnational administrative boundaries, and is crucial for understanding baseline RI conditions [17].

The framework is enabled by well-maintained history of SIA timings, scopes, and vaccine choices. Further, due to its responsiveness to SIA exposures, prospective SIA calendars may also be evaluated and thus it is a suitable tool for programmatic planning and decision-making for SIAs. While not shown here, it is possible to leverage the framework to estimate the number of SIAs required at subnational scales to meet programmatic immunization objectives. Likewise, the framework allows ready assessment of how vaccine policies and choices have affected or may affect population immunity.

There are several limitations in our approach. While we estimate immunity under a wide range of SIA impact assumptions (coverage × effectiveness), in practice both coverage and effectiveness may vary among areas and over time. Further, these may covary, for example due to population structure and possibly compounded risk factors (RI access, chronically missed children, and enteropathy-affected immune responses). Data on population inaccessibility to vaccinators may be unavailable through the data source for SIAs, meaning that population immunity is overstated, just as coverage or SIA exposure is overstated - for example, Borno state in Nigeria [12,20]. Although we have not characterized it here, different coverages and uncertainty can be accommodated in the framework - for example, see the inclusion of SIA uncertainty in related past work [12]. However, there is a dearth of accurate, spatiotemporally resolved data on immunization history and serology to resolve variability due to these risk factors. Absent these data, there will always be some uncertainty as to the effectiveness of vaccination efforts and the potential for susceptible sub-populations, and so we have opted to include a range of estimates from different assumptions.

We have not considered single-dose IPV vaccination, which might increase the level of individual protection from paralysis but confers little protection from transmission compared to OPV-induced immunity [21]. Nor have we considered the complexities of waning mucosal immunity and susceptibility to re-infection, or immunity derived from infection with WPV or VDPV, instead focusing on seroconversion and field estimates of per-dose effectiveness attributable to OPVs.

In addition, as a model of how SIA exposures build on a base of immunity from RI, the method relies on accurate records of the geographic scope, timing, and vaccine formulations of SIAs. Inaccuracies in the historical record of SIAs could thus affect the results.

Nonetheless, we quantify the likely impacts of changes in population immunity following cessation of tOPV, which has been a steep decline in OPV-derived immunity to type 2, and a general decrease in immunity to types 1 and 3. While some decline in immunity to types 1 and 3 may be acceptable, owing to lower WPV1 risk due to its elimination from Africa, maintenance of immunity to reduce the risk of outbreaks following WPV1 importation and of emergence of cVDPV1 and 3 will need to continue. In the context of increasingly limited resources, prioritization and subnational geographic targeting can allow the GPEI to focus immunization activities where they are most needed, and where our methodology can be used. Likewise, we illustrate the widespread, low immunity to type 2 poliovirus (zero three years after the vaccine switch absent mOPV2 usage for the 6–36 age group) and the fleeting increase resulting from outbreak response to cVDPV2, leaving areas vulnerable

to re-emergence and reimportation of cVDPV2s shortly after a response, and adjoining areas not covered in outbreak response vulnerable to spread and emergence of cVDPV2. As cVDPV2 outbreaks continue to spread, careful monitoring of immunity will be critical to assess risk in both newly affected geographies and areas with previous responses. A separate article in this issue illustrates the utility of the immunity estimates for this purpose [22]. Lastly, as areas with current cVDPV2 outbreaks often suffer from gaps in immunity to types 1 and 3 polioviruses, managing risks using different vaccines will require careful consideration of tradeoffs between immunization with bOPV, mOPV2, and potentially tOPV and a novel, genetically stabilized OPV2 (nOPV2).

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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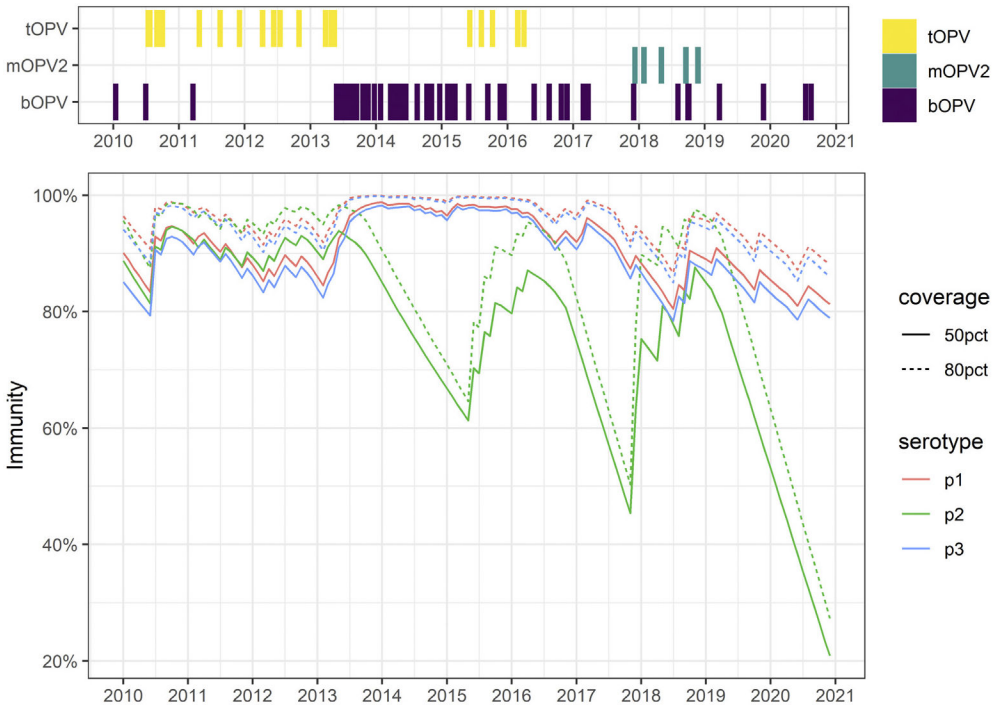

Fig. 1.

Immunity and SIA schedule in Banadir, Somalia. The bars in the top panel indicate dates of SIAs including Banadir. The plot at the bottom indicates the resulting immunity estimates to each serotype, under 50% and 80% coverage assumptions.

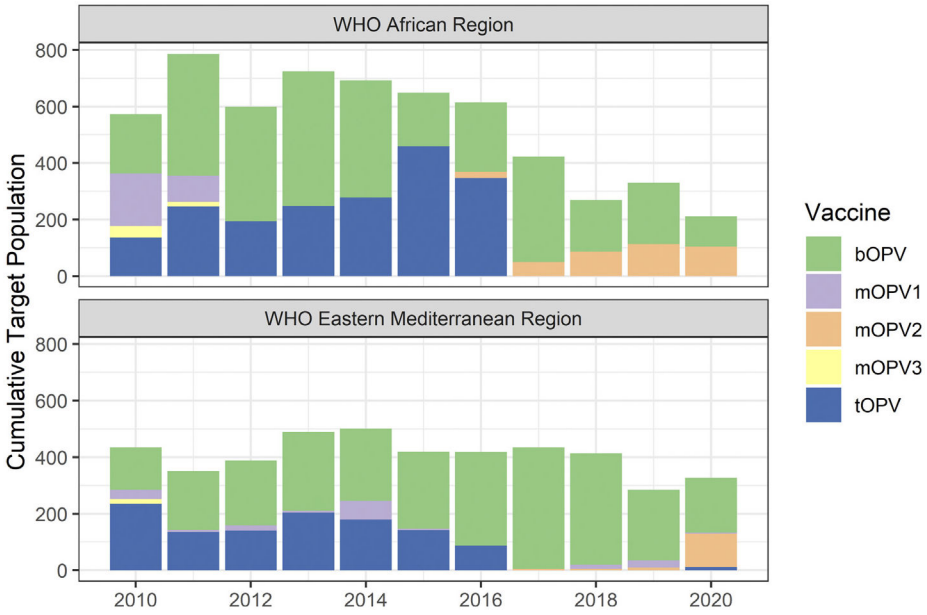

Fig. 2.

Cumulative population targeted with OPV SIAs in GPEI-funded countries, by year, in the WHO African Region and Eastern Mediterranean Region.

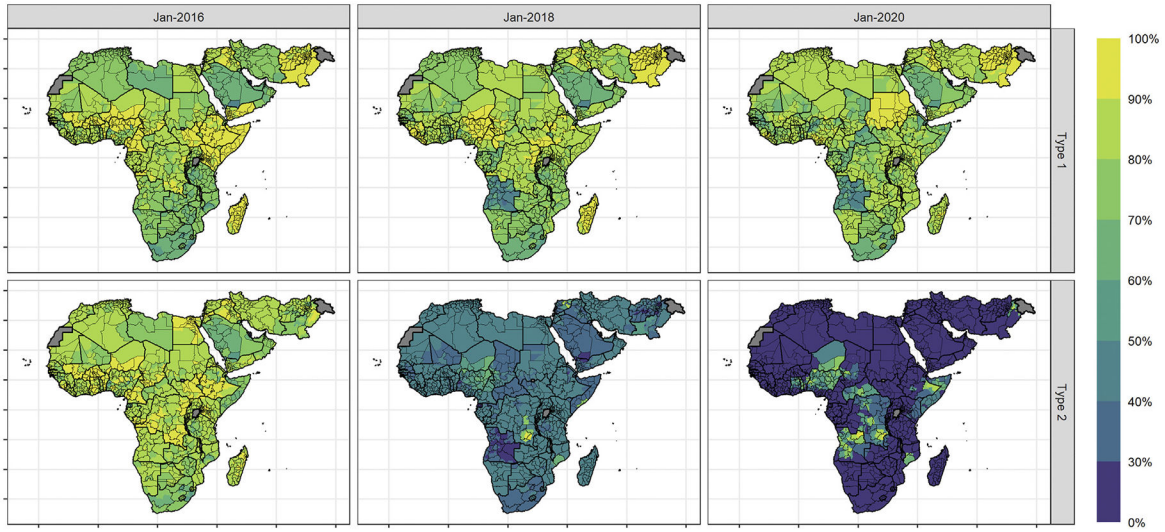

Fig. 3.

Estimated Immunity to types 1 and 2 poliovirus, January 2016, 2018 and 2020, WHO African Region and Eastern Mediterranean Region. Colors indicate district-level (2nd administrative unit) immunity estimates under the 50% coverage assumption[1]. [1]The publication of this map does not imply the expression of any opinion whatsoever on the part of WHO concerning the legal status of any territory, city, or area or of its authorities, or concerning the delimitation of its frontiers or boundaries. WHO does not endorse or approve the sub-national boundaries in this map.

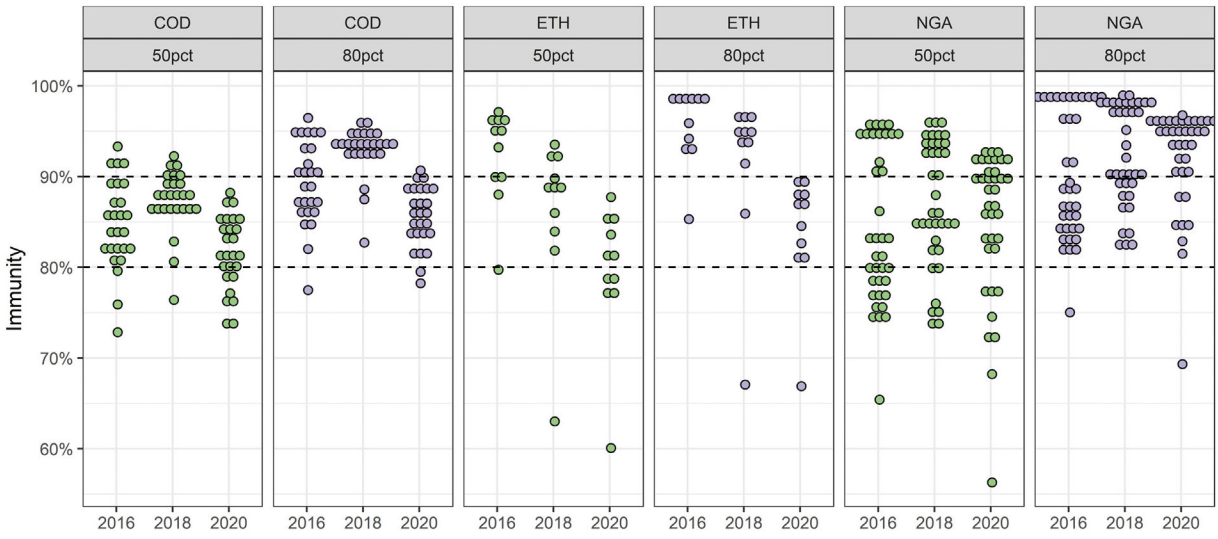

Fig. 4.

Province-level (first administrative unit) type 1 poliovirus immunity estimates in January 2016, 2018, and 2020, for the Democratic Republic of the Congo (COD), Ethiopia (ETH) and Nigeria (NGA), using either the 50% or 80% coverage assumptions. Example immunity targets (80% and 90%) shown in dotted lines.

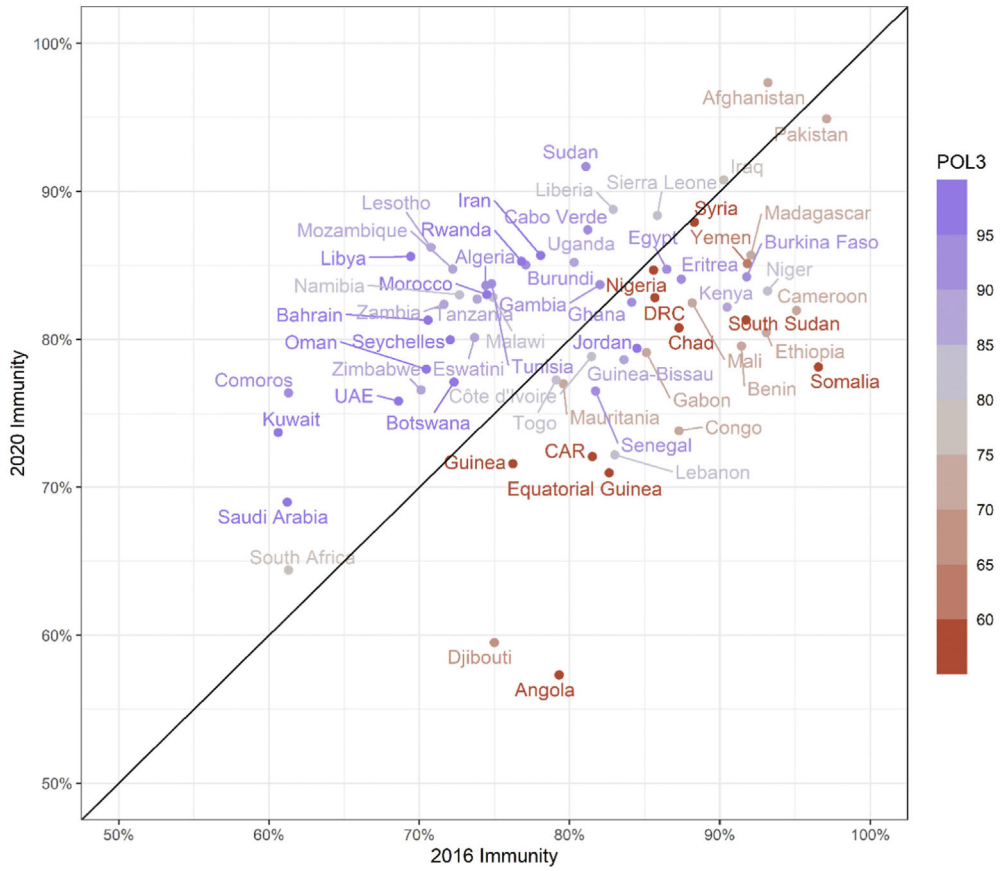

Fig. 5.

National-level type 1 poliovirus immunity estimates for countries in the WHO African and Eastern Mediterranean Regions, using the 50% coverage assumption. Colors indicate the WHO-UNICEF best estimates of national immunization coverage (WUENIC)[23] for the 3rd dose of oral poliovirus vaccine (pol3) in 2016.

Table 1

Vaccine effectiveness assumptions used for estimating polio mucosal immunity.

Vaccine	Type 1	Type 2	Type 3
tOPV	0.40	0.40	0.37
bOPV	0.50	0	0.46
mOPV1	0.65	0	0
mOPV2	0	0.7	0
mOPV3	0	0	0.6
IPV	0	0	0