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# A dynamic model of transmission and elimination of peste des petits ruminants in Ethiopia

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**Peste des petits ruminants (PPR), a devastating viral disease of sheep and goats, has been targeted by the global community for eradication within the next 15 years. Although an efficacious attenuated live vaccine is available, the lack of knowledge about the transmission potential of PPR virus (PPRV) may compromise eradication efforts. By fitting a metapopulation model simulating PPRV spread to the results of a nationwide serological survey in Ethiopia, we estimated the level of viral transmission in an endemic setting and the vaccination coverage required for elimination. Results suggest that the pastoral production system as a whole acts as a viral reservoir, from which PPRV spills over into the sedentary production system, where viral persistence is uncertain. Estimated levels of PPRV transmission indicate that viral spread could be prevented if the proportion of immune small ruminants is kept permanently above 37% in at least 71% of pastoral village populations. However, due to the high turnover of these populations, maintaining the fraction of immune animals above this threshold would require high vaccine coverage within villages, and vaccination campaigns to be conducted annually. Adapting vaccination strategies to the specific characteristics of the local epidemiological context and small ruminant population dynamics would result in optimized allocation of limited resources and increase the likelihood of PPR eradication.**

eradication | elimination | mathematical model | vaccination | control

**P**este des petits ruminants (PPR) is a disease of sheep and goats caused by a morbillivirus closely related to rinderpest virus. Highly transmissible, the disease has a devastating impact on small ruminants, as morbidity and mortality rates can reach near 100% in naive populations (1, 2). PPR virus (PPRV) is now endemic in most of Africa and throughout Asia, where it is one of the main constraints to small ruminant production and welfare, and therefore a threat to food security and livelihoods of the poorest communities, for which sheep and goats are often an important asset. Moreover, PPRV spillover from domestic to wild populations resulted in serious concerns for the conservation of some critically endangered species (3–6).

In the aftermath of the eradication of rinderpest, the World Organization for Animal Health and the Food and Agriculture Organization of the United Nations launched an initiative to eradicate PPR by 2030. The global strategy (7) heavily relies on the immunization of small ruminant populations through the organization of mass vaccination campaigns, due to the availability of an efficacious attenuated live vaccine producing lifelong immunity against all PPRV serotypes after a single administration (8). Such campaigns are, however, costly and difficult to implement in the field due to the vaccine's thermolability (8), the accessibility and mobility of some small ruminant populations, and the lack of precise census data and national animal identification systems. To reduce the costs of eradication efforts, it is essential to assess the PPRV transmission potential, so small ruminant populations acting as a viral reservoir

can be targeted, and within them, the minimal fraction of animals that needs to be immunized to prevent viral transmission can be estimated (7). Such information is, however, missing.

Among PPRV-endemic countries, Ethiopia has the seventh largest small ruminant population (FAOSTAT; [www.fao.org/faostat/en/#home](http://www.fao.org/faostat/en/#home)), which accounts for a substantial fraction of national demand for meat consumption and export earnings (9–11). PPR was first clinically suspected in the country in 1977, before serological and virological evidence of its presence were documented in 1984 and 1991 (12, 13). Before the first mass vaccination campaign, a nationwide serological survey was initiated in 1999 (13). By fitting a metapopulation model of PPRV transmission to these survey results, this study aims to estimate the level of PPRV transmission within and between Ethiopian small ruminant village populations, and the optimal vaccination coverage required for disease elimination.

## Results

**Estimation of Transmission Parameters.** The model simulated the spread of PPRV between small ruminants—sheep and goats—within and between Ethiopian villages. The relatively small number of small ruminants in an average village meant that PPRV did not persist at village level, but did at the metapopulation

## Significance

**Only two infectious diseases, smallpox in humans and rinderpest in cattle, have been eradicated so far. Peste des petits ruminants (PPR), a viral disease representing a major burden for sheep and goat farmers across Africa and Asia, is now targeted for eradication through mass vaccination campaigns. While an efficacious vaccine providing protective and lifelong immunity exists, the level of PPR virus transmission in animal populations is unknown. By combining the results from a nationwide serological survey with a dynamic model simulating viral spread, we estimated viral transmission potential in Ethiopia, where PPR virus is endemic, and vaccination coverage required for disease elimination. This approach is relevant to identify populations at high risk of viral persistence and to inform vaccination strategies.**

Author contributions: G.F., A.W.-S., D.U.P., and F.R. designed research; G.F. performed research; A.W.-S., A.C., and L.M.Y. contributed new reagents/analytic tools; G.F. and A.C. analyzed data; and G.F., A.W.-S., A.C., L.M.Y., D.U.P., and F.R. wrote the paper.

The authors declare no conflict of interest.

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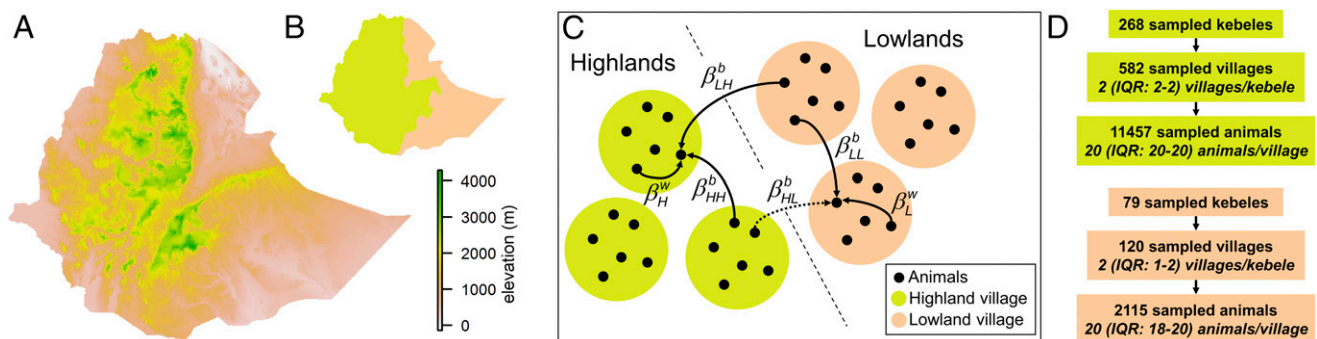
level, through a “rescue effect” (14). Variation in the Ethiopian landscape defines different agroecological zones associated with distinct livestock husbandry systems. While sedentary mixed livestock–crop farms prevail in the high central plateau, regions of low elevation are home to pastoralists, who heavily rely on livestock production for their livelihood (Fig. 1A and B) (15). Small ruminant flocks are notably larger and more mobile—in search for grazing and watering points—in the lowland pastoral than in the highland sedentary systems (10, 16–18). Modeled village populations were thus classified as sedentary or pastoral, and associated with different transmission potential. As illustrated in Fig. 1C, the within-village transmission parameters  $\beta_H^w$  and  $\beta_L^w$  referred to the number of effective contacts per unit of time (i.e., contacts that would result in infection if involving a susceptible and an infected small ruminant) made by a small ruminant with other small ruminants in the same village in highlands and lowlands, respectively. PPRV also spread between villages through population mixing at watering points or pasture, and through live-animal trade.  $\beta_{kr}^b$  was the number of effective contacts per unit of time that a small ruminant in a village in region  $r$  made with small ruminants from other villages of region  $k$ . Therefore,  $\beta_{LL}^b$ ,  $\beta_{HH}^b$ , and  $\beta_{LH}^b$  referred to PPRV transmission between lowland villages, between highland villages, and from lowlands to highlands, respectively (Fig. 1C).  $\beta_{HL}^b$  accounted for transmission from highlands to lowlands and was expressed as  $\beta_{HL}^b = r_{HL} \beta_{LH}^b (P_H/P_L)$ , with  $r_{HL}$ , the relative strength of mixing (if contacts were reciprocal,  $r_{HL} = 1$ ), and  $P_H/P_L$ , the ratio between highland and lowland population sizes. While intervillage contacts resulting from mixing at watering points and pastures were reciprocal, this was not the case with live-animal trading. It was strongly directed from lowlands, where prices and the humans-to-small ruminants ratio are low, into highlands, where prices and the humans-to-small ruminants ratio are high (10, 19, 20), suggesting that  $r_{HL} \ll 1$ . When estimated along other transmission parameters,  $r_{HL}$  was poorly determined, as its marginal posterior distribution remained similar to its prior. We fixed  $r_{HL} = 0$ , assuming that PPRV transmission from highlands to lowlands was epidemiologically negligible. This scenario maximized intervillage transmission in lowlands as  $r_{HL}$  and  $\beta_{LL}^b$  were negatively correlated. An alternative scenario, with  $r_{HL} = 0.5$ , is presented in *SI Appendix*. We used an approximate Bayesian computation method based on a sequential Monte-Carlo algorithm (ABC-SMC) to sample from the joint posterior distribution of the transmission parameters  $\{\beta_L^w, \beta_H^w, \beta_{LL}^b, \beta_{LH}^b, \beta_{HH}^b\}$  (21–24). This likelihood-free approach relies on matching a set of summary statistics (*Methods*) obtained from model simulations to the results of the serological survey (13). As such, the output of our ABC-SMC inference is actually an approximation of the posterior distribution, but for convenience it will be referred to as the posterior distribution throughout the text. The survey covered 7 of the 11 regions (first administrative

division) and 84 of the 546 weredas (third administrative division) into which Ethiopia was divided. Out of 11,457 and 2115 samples collected in highlands and lowlands (Fig. 1D), 4.6% and 16.6% were positive. As the village of origin was not specified for most samples (*SI Appendix*), the proportion of positive animals within a kebele (subdistrict, fourth administrative division) was reported (Fig. 2F).

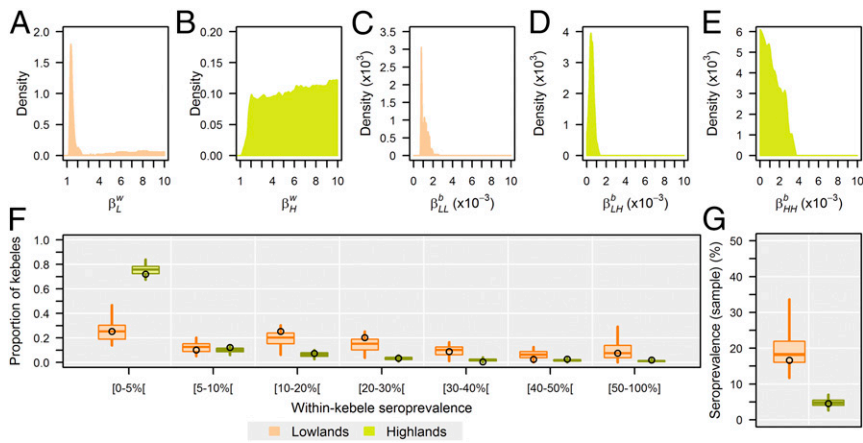
Model simulations adequately reproduced the serological survey results in both areas (Fig. 2F and G). Nevertheless, the proportion of surveyed kebeles with low seroprevalence (<5%) in highlands and with a seroprevalence ranging between 11 and 30% in lowlands were respectively overestimated and underestimated. By the time the serological survey was implemented, 20–25 y following the first PPRV incursion, the simulated animal-level seroprevalence already fluctuated around its long-term average (*SI Appendix*, Fig. S1). The animal-level prevalence of infection was on average five times higher in lowlands than in highlands. Likewise, epidemics were more frequent in lowland than in highland villages (Fig. 3).

Marginal posterior distributions of transmission parameters are presented in Fig. 2A–E, and summarized in Table 1. *SI Appendix*, Fig. S6 shows the posterior predictive distributions of village-level reproduction numbers. The highest posterior density of the level of within-village transmission in lowland was concentrated at low  $\beta_L^w$  values (range, 1.2–2.4) with the maximum a posteriori equal to 1.37 (Fig. 2A). However, a second, low-probability, and almost-uniform mode was located at high  $\beta_L^w$  values. Indeed, for  $\beta_L^w > 5$ , the model simulations were insensitive to further increases in  $\beta_L^w$  (*SI Appendix*, Fig. S2), until it reaches the upper bound of the prior distribution. The identifiability of highland transmission parameters was limited, with a trade-off between intervillage transmission routes from other highland or lowland villages,  $\beta_{LH}^b$  and  $\beta_{HH}^b$  being negatively correlated (*SI Appendix*, Fig. S5). While this lack of identifiability prevented us from precisely inferring actual highland parameter values, the joint posterior distribution was restricted to a region of the parameter space corresponding to the highland village-level reproduction number  $r_{HH}^b < 1$ , suggesting that PPRV could not be maintained within highlands, but only within lowlands (i.e.,  $r_{LL}^b > 1$ ) (Table 1). PPRV incursions into highlands would ultimately fade out unless the virus was reintroduced.

**Immunity Threshold and Vaccination Coverage.** This source–sink dynamics suggests that vaccinating lowland populations could eliminate PPRV in both regions. The lowest fraction of immune animals preventing PPRV spread for all values of the posterior distribution was reached if  $p_a = 37\%$  of small ruminants in  $p_v = 70.7\%$  of villages were protected against infection (Fig. 4A). When ignoring immunity resulting from past infection, keeping the immunity level above  $p_a$  within a village for a whole year would require the immunization of 61.7% of animals, adults and



**Fig. 1.** Model structure and serological survey coverage. (A) Elevation in Ethiopia. (B) Division into lowlands (Afar and Somali regions) (brown) and highlands (green). (C) Modeled animals are grouped into villages, which are differentiated as lowlands or highlands.  $\beta_r^w$  refers to PPRV transmission within a village in region  $r$ , and  $\beta_{kr}^b$  to intervillage transmission from region  $k$  to  $r$ . An arrow is dashed as  $\beta_{HL}^b$  was set to 0. (D) The number of sampled units.



**Fig. 2.** Marginal posterior distributions and comparison between simulated and observed survey results. (A–E) Marginal posterior distributions: the number of effective contacts/animal within (A) a lowland village and (B) a highland village; the number of effective contacts/animal (C) between lowland villages, (D) from the lowlands into the highlands, and (E) between highland villages. (F) Observed (dots) and posterior predictive (boxplots) distributions of surveyed kebeles according to their seroprevalence in the lowlands (brown) and highlands (green). (G) Observed (dots) and posterior predictive (boxplots) proportions of seropositive surveyed small ruminants; boxplots show the 5th, 25th, 50th, 75th, and 95th percentiles.

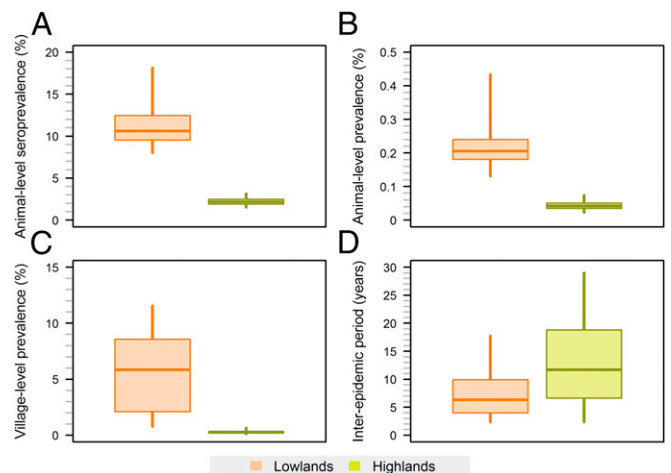
young, as 40% of the population was renewed every year under the baseline scenario (Fig. 4B). Over subsequent years, annual campaigns immunizing 61.7% of young animals born since the preceding campaign would prevent PPRV spread. Vaccination programs are recommended to be run over a 3- to 4-y period (7). Maintaining the immunity level above the elimination threshold for a period of 4 y would suppress PPRV circulation, and requires 16.6 million animals to be vaccinated in lowlands, assuming that the vaccine results in complete and lifelong immune protection in all vaccinated animals (Fig. 4C). Compared with a vaccination strategy targeting all lowland small ruminants during the first round and all young small ruminant during subsequent rounds, the number of required vaccine doses would be reduced by 56.4%. However, as turnover increased (Fig. 4D and E) and vaccine effectiveness decreased (SI Appendix, Table S5), the required vaccination coverage rose. Assuming that only 80% of vaccinated animals are effectively immunized meant a 25% increase in coverage. Variation in the number of villages, PPR case fatality rate, and demographic parameters did not have a major impact on infection parameter estimates and immunization thresholds required for PPRV elimination (SI Appendix).

**Discussion**

Model outputs suggest that PPRV transmission was sustained in Ethiopia’s lowland pastoral region through viral transmission between small ruminant village populations. Lowlands thus acted as a reservoir of infection from which PPRV spilled over into the highland sedentary region where its maintenance was unlikely. The trade of sheep and goats from Ethiopian lowlands into neighboring countries and Gulf states (17, 19, 25) makes PPRV elimination from Ethiopia’s lowlands not only a national, but also a regional and even global priority. Based on our estimation, ensuring that at least 37% animals were immune in at least 70.7% of pastoral villages would prevent PPRV spread. However, due to the high population turnover and not all vaccinated animals developing a protective immunity, vaccination coverage would need to be substantially higher (26). Potential causes for inadequate immunity may include individual variations in immune response, improper vaccine administration, and the use of ineffective vaccine batches. Currently available vaccines are thermolabile (8), requiring the maintenance of the cold chain until their administration. This is a major challenge as most PPR-endemic countries have poor infrastructure and periods of hot climatic conditions. The recent development of a thermostable presentation would facilitate vaccine delivery (27). The model assumed random selection of vaccinated villages and animals. If selection was purposive, for example, based on accessibility, higher coverage would be needed to prevent persistence of the infection in unvaccinated population clusters. The number of animals to be vaccinated each year seems achievable: apart from the first vaccination round, it was lower than the coverage achieved during each annual mass vaccination

campaign conducted between 2005 and 2011 in the whole country (11). Although an economic analysis would be required to assess the most cost-effective strategy balancing the overall number of vaccine doses used and the number of vaccinated villages, vaccination efforts would be lower than suggested by the global control and eradication strategy (7), which recommended the vaccination of almost all small ruminants above 3 mo of age. The authors of the strategy, however, recommended adapting this generic strategy to local conditions, and emphasized that targeting at-risk populations, especially pastoral flocks, and estimating context-specific elimination thresholds would reduce eradication costs.

The model suggested frequent PPRV incursions into highlands from lowlands. In the search for grazing and watering points, pastoral flocks may move toward highlands, where they then mix with sedentary flocks. Moreover, goats and sheep traded from lowlands into highlands are moved through several markets, over long distances (10). Such marketing systems are likely to promote viral amplification, as observed with other species (28). While most animals traded from lowlands would end up in abattoirs, they could infect highland animals brought to markets. Unsold highland animals returning to their village of origin could then spread the infection. If these interfaces between pastoral and sedentary populations were characterized spatiotemporally, they could be targeted by vaccination to reduce viral spillover. The level of



**Fig. 3.** Posterior predictive distribution of (A) animal-level seroprevalence, (B) animal-level viral prevalence, (C) village-level viral prevalence, and (D) interepidemic periods. Young and adults were considered. A village was classified as infected if the prevalence  $\geq 0.5\%$ . Interepidemic period: the length of time between two successive epidemic peaks in a given village. The 5th, 25th, 50th, 75th and 95th percentiles are shown.



or higher levels of susceptibility in sheep than goats are also documented (34, 36). It is therefore important to quantify potential variation in infectiousness, as it would affect optimal vaccination coverage. Although other domestic (1, 2, 35) and wild (5, 40) animal species are susceptible to PPRV, current knowledge suggests that control of the infection in small ruminants would prevent disease outbreaks in other species (6, 41, 42), as observed with rinderpest following its control in cattle.

Another limitation of the model was the lack of reliable small ruminant population data, especially for pastoral flocks, and the lack of specific data about spatiotemporal variation in population sizes and demographic profiles, farming and trading practices. Although pastoralists prevail in Afar and Somali regions, and sedentary flocks in the other parts of the country, production systems are more diversified and their distribution more heterogeneous than assumed in the model (10, 17). Lowland pastoral populations outside Afar and Somali were only subject to limited sampling in the 1999 survey, but they should be included in vaccination programs targeting pastoral flocks. As live-animal trade networks are consistently highly heterogeneous in multiple settings (43, 44), this is also likely to be the case for Ethiopian small ruminants (45). Identifying and targeting the most at-risk populations at a higher spatial resolution would allow further reducing the required vaccination coverage (46), but this needs detailed data on demographic processes, including their spatiotemporal variation (47). Moreover, the way in which village populations are repopulated in the aftermath of an outbreak is not documented, but it is of importance to understand the speed at which susceptible populations are replenished as this may promote PPRV endemicity. Seasonality in infection patterns and population dynamics were not explored. However, seasonal variation in environmental conditions affects the availability of grazing, and consequently demographic processes (e.g., variations in birth rates during the year in some husbandry systems), animal movements, mixing patterns within and between husbandry systems (17, 18), and therefore PPRV transmission. Likewise, trade patterns are likely to vary according to seasonal religious or other festivals, as observed for other livestock species and countries (48, 49). Accounting for these seasonal patterns would allow the identification of the most suitable time periods for vaccination.

In conclusion, identifying and targeting high-risk populations through vaccination campaigns informed by the estimation of context-specific PPRV transmission levels would not only reduce the cost of PPR eradication, but by setting more achievable vaccination coverage also increase the likelihood of success. Further information would be needed on spatiotemporal variation in PPRV distribution and small ruminant population dynamics to more precisely identify high-risk populations, to refine optimal vaccination coverage and to identify the most suitable time periods during which to vaccinate.

## Methods

**Small Ruminant Population Data.** As an estimated 80–90% of pastoralists were grouped in the two eastern regions of Afar and Somali (50, 51), sheep and goats in Afar and Somali regions are here referred to as the pastoralist, lowland, small ruminant population, and sheep and goats in the rest of the country as the sedentary, highland, small ruminant population (Fig. 1 A and B). Partitioning sedentary and pastoral systems according to an elevation threshold of 1,000 m (10) did not affect seroprevalence patterns (SI Appendix, Table S1). In the serological survey to which the model was fitted, the sampling of lowland populations in the south and west of the country was very limited. Serological results for regions other than Afar and Somali thus reflected infection patterns in their highland areas. To our knowledge, a census of Ethiopian villages was not available. We estimated plausible values based on the literature and the human population census. In the main text, we considered 10,000 lowland and 100,000 highland villages. Alternative scenarios are detailed in SI Appendix. The number of small ruminants in highlands and lowlands was estimated at 27.2 and 17.4 million, respectively (52, 53) (SI Appendix).

**Model. PPRV transmission within a village.** While goats are sometimes reported to be more susceptible to PPRV than sheep (1), the results of the serological survey

did not suggest any difference in the seroprevalence between both species (13). We did not, therefore, differentiate sheep and goats, using small ruminant as the unit of the model. All villages were similar according to their region of origin. Highland and lowland villages only differed in their population size and PPRV transmission potential. As production was extensive and animals from multiple flocks could mix within a village, homogeneous mixing was assumed within villages. PPRV dynamics within a village was first explored using a stochastic model (SI Appendix). For the investigated range of small ruminant population sizes, a PPRV incursion caused an epidemic followed by extinction, that is, it could not become endemic. To reduce computing time, within-village PPRV transmission was modeled as a deterministic process. Viral fade-out was simulated by setting the number of infected animals to 0 when the epidemic curve reached its trough following the epidemic peak. The number of infectious animals then remaining in the village depended on the population size and the level of PPRV transmission, but it was always lower than 3.5, in agreement with the high risk of fade-out observed in the stochastic simulations.

Small ruminants were divided into two age categories, young (<1 y old) and adults (>1 y old), which differed in their non-PPR-related mortality rates,  $\gamma_{a=1}$  and  $\gamma_{a=2}$ . New sheep and goats entered into the village  $i$ , through births, which occurred all year long, as breeding was generally uncontrolled (10, 54). Small ruminants could pass through three successive and mutually exclusive health states: susceptible, infected, and recovered. Susceptible animals became infected following an effective contact with an infected small ruminant. Infected small ruminants could either survive and acquire lifelong immunity to PPR (1), or die due to the disease. We did not discriminate the infection state into latency (i.e., infected but not infectious) and infectiousness as the model was run in discrete time, with the duration of a time step being equal to the length of the infection period  $\tau$  (i.e., assuming a fixed infection period for all animals). The number of susceptible ( $S_{r,i,a,t}$ ), infected ( $I_{r,i,a,t}$ ), and recovered ( $R_{r,i,a,t}$ ) small ruminants of age  $a$  in a village  $i$  in a region  $r$  (highlands or lowlands) at time  $t$  were expressed by the difference equations below. The rates of demographic processes being much lower than the transmission rate, they were approximated as follows:

$$\begin{aligned} S_{r,i,a=1,t} &= b_{r,i,t} + (1-\delta)(1-\gamma_{a=1})(1-\lambda_{r,i,t})S_{r,i,a=1,t-\tau} \\ I_{r,i,a=1,t} &= (1-\delta)(1-\gamma_{a=1})\lambda_{r,i,t}I_{r,i,a=1,t-\tau} \\ R_{r,i,a=1,t} &= (1-\delta)(1-\gamma_{a=1})R_{r,i,a=1,t-\tau} + (1-\rho)(1-\delta)(1-\gamma_{a=1})I_{r,i,a=1,t-\tau} \\ S_{r,i,a=2,t} &= (1-\gamma_{a=2})(1-\lambda_{r,i,t})S_{r,i,a=2,t-\tau} + \delta(1-\gamma_{a=1})(1-\lambda_{r,i,t})S_{r,i,a=1,t-\tau} - \mathbf{1}_{r,i,t} \\ I_{r,i,a=2,t} &= (1-\gamma_{a=2})\lambda_{r,i,t}I_{r,i,a=2,t-\tau} + \delta(1-\gamma_{a=1})\lambda_{r,i,t}I_{r,i,a=1,t-\tau} + \mathbf{1}_{r,i,t} \\ R_{r,i,a=2,t} &= (1-\gamma_{a=2})R_{r,i,a=2,t-\tau} + \delta(1-\gamma_{a=1})R_{r,i,a=1,t-\tau} + (1-\rho)[(1-\gamma_{a=2})I_{r,i,a=2,t-\tau} \\ &\quad + \delta(1-\gamma_{a=1})I_{r,i,a=1,t-\tau}]. \end{aligned}$$

The subscripts  $a = 1$  and  $a = 2$  referred to the first (young) and second (adult) age categories.  $\mathbf{1}_{r,i,t}$  referred to viral incursion (see below).  $\delta$  referred to the rate at which young small ruminant became adults.  $\rho$  was the PPR case fatality rate. In the absence of disease, all villages from a given region  $r$  were composed by  $N_r$  small ruminants, and their birth rate was constant  $b_{r,i,t} = b_r$ . As PPR caused abortion and mortality (1, 2), the birth rate was reduced during an epidemic:  $b_{r,i,t} = b_r(S_{r,i,a=2,t-\tau} + R_{r,i,a=2,t-\tau}) / (\kappa N_r)$ , with  $\kappa$  being the proportion of adult small ruminants. Once the epidemic faded out in village  $i$ ,  $b_{r,i,t} = b_r$ , ensuring the progressive replenishment of the village population. Finally,  $\lambda_{r,i,t}$  was the risk of infection for a susceptible small ruminant due to contacts with infected small ruminants in the same village  $i$ :  $\lambda_{r,i,t} = 1 - \exp(-\beta_r^w \sum_a I_{r,i,a,t-\tau} / N_{r,i,t-\tau})$ . Production being extensive, the contact process was assumed to be frequency-dependent, with  $N_{r,i,t}$  being the total number of small ruminants in village  $i$  at time  $t$ . Therefore, the within-village basic reproduction number was defined as follows:  $R_{0,r}^w = \beta_r^w / ((1-\gamma_{a=1})(1-\kappa) + (1-\gamma_{a=2})\kappa)$ .

**PPRV transmission between villages.** While the village component was deterministic, intervillage transmission was stochastic. Homogeneous mixing was assumed, with respect to the region of origin. At time  $t$ , the risk  $\pi_{r,i,t}$  of having at least one susceptible small ruminant in a noninfected village  $i$  in region  $r$  becoming infected due to infected small ruminants in other villages was computed as follows:  $\pi_{r,i,t} = 1 - \exp[-(\sum_a S_{r,i,a,t-\tau} \sum_k \beta_k^w \sum_{j \neq i} \sum_a I_{r,j,a,t-\tau} / \sum_{j \neq i} N_{k,j,t-\tau})]$ . A random number was generated between 0 and 1. If it was lower than  $\pi_{r,i,t}$ , PPRV was introduced in village  $i$ ,  $\mathbf{1}_{r,i,t} = 1$ ; if not,  $\mathbf{1}_{r,i,t} = 0$ . Note that, if  $\sum_a I_{r,i,a,t-\tau} > 0$ ,  $\mathbf{1}_{r,i,t} = 0$ .

**Parameter estimation.** All prior distributions were uniform with wide ranges (Table 1). The joint posterior distribution was estimated by repeated stochastic simulations using ABC-SMC. A simulation matched the data if distances between summary statistics computed for simulated and observed datasets were below given thresholds. The summary statistics were as follows: (i) the observed ( $o_{1,r}$ ) and simulated ( $s_{1,r}$ ) number of positive animals in region  $r$ , and (ii) the observed ( $o_{2,r,i}$ ) and simulated ( $s_{2,r,i}$ ) proportions of sampled kebeles in region  $r$  with an apparent seroprevalence falling within a range  $l$ : [0–5%],

[5–10%], [10–20%], [20–30%], [30–40%], [40–50%], and [50–100%]. As there were two regions, there were thus four pairs of summary statistics, compared using the distance functions  $d_{1,r} = |o_{1,r} - s_{1,r}| / o_{1,r}$  and  $d_{2,r} = \sum_i o_{2,r,i} \log(o_{2,r,i} / s_{2,r,i}) + s_{2,r,i} \log(s_{2,r,i} / o_{2,r,i})$ . The latter was based on the relative entropy (55), following ref. 23. To ensure that this function was defined (i.e., all  $o_{2,r,i}$  and  $s_{2,r,i} > 0$ ), 1 was added to every bin (i.e., to the number of observed and simulated kebeles falling in each of the seven seroprevalence ranges). The algorithm is further detailed in *SI Appendix*. Other parameters were fixed, and their values assessed based on a review of the literature (*SI Appendix, Table S2*). The impact of variations in these values on transmission parameter estimates was explored (*SI Appendix*).

**Simulations and outcomes.** The duration of a simulation was drawn from a uniform distribution ranging from 20 to 25 y, as PPRV was assumed to have circulated in Ethiopia for at least 20 y before the 1999 survey (12, 13, 56). The infection was

introduced at the start of a simulation, and every 500 d for the first 8 y of a simulation. Based on the posterior distribution, we computed the posterior predictive values of the village-level reproduction numbers  $r_{kr}^b$ , defined as the expected number of villages in region  $r$  infected by a single infected village in region  $k$ , in an initially fully susceptible metapopulation. It measured the potential of PPRV to be sustained within a region ( $k = r$ ) and from a region to another ( $k \neq r$ ) (*SI Appendix*). The within-village and village-level immunity levels preventing PPRV transmission were assessed, as well as the annual vaccination coverage required to maintain immunity levels above these thresholds. The mathematical model was coded in the C language, and the ABC-SMC algorithm was implemented in R, version 3.2.2 (57). Scripts are available at <https://bit.ly/2MxQD7d>.

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