Transmission and elimination of Peste des Petits Ruminants in Ethiopia: insights from a dynamic model

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Supporting Information

Further information are provided about the data, the model and its parameter values. The results of a sensitivity analysis exploring the impact of variation in fixed parameter values on transmission parameter estimates are also presented.

Data

Small ruminant population. The number of sheep and goats in the highlands were derived from the 2001-2002 Agricultural census (1). However, this census was incomplete for Afar and Somali regions, for which the small ruminant population was estimated as described by Behnke et al (2010) (2). According to the 2006 Livestock Development Master Plan Study, Afar and Somali, accounted altogether for 34.6% and 43.2% of the national sheep and goat populations, respectively. Assuming that these figures remained stable from 2002 to 2006, the number of sheep and goats in the lowlands was estimated by multiplying the respective highland population sizes by 0.346/(1-0.346) and 0.432/(1-0.432), respectively. The numbers of sheep and goats were estimated to be 14.2 and 12.9 million in the highlands, respectively, and 7.5 and 9.8 million in the lowlands, respectively. There were thus 27.2 million small ruminants in the highlands and 17.4 million in the lowlands.

Number of villages. We first estimated the number of villages in pastoral and sedentary areas by quantifying the number of kebeles, and the number of villages per kebele, in each area. Kebeles, or sub-districts, are Ethiopia's lowest administrative division level. They have

www.pnas.org/cgi/doi/10.1073/pnas.1711646115

been described as comprising of about 5000 people (3, 4). In (5), the number of kebeles in 2007 was provided for seven regions (Tigray, Afar, Amhara, Oromia, Benishangul-Gumuz, SNNPR and Gambella). Using the 2007 census data, the average number of people per kebele was 4263. The census estimated the population to be about 53.5 million in 1994 and 73.75 million in 2007 (6). Through linear extrapolation, the human population in 1999, the year of the PPR serological survey, was estimated to be 61.3 million. An average of 4000-5000 people per kebele would then mean 12,000-15,000 kebeles in the whole country. As 84% of the human population was rural, the number of rural kebeles would then range between 10,000-13,000. Other references suggested higher numbers of kebeles: about 30,000 for the whole country (i.e. 2043 people/kebele) (4), thus 25,000 in rural areas. The figure of 21,000 rural kebeles was mentioned (i.e. 2452 people/kebele) in another reference (7). The average number of villages per kebele varied from 10 to 30 according to different sources (3, 4, 8). Assuming that the number of kebeles in rural Ethiopia ranged from 10,000 to 25,000, and the average number of villages per kebele from 10 to 30, then the actual number of villages would range from 100,000 to 750,000. The average village population size would then range between 69 and 515 inhabitants.

In addition, we estimated the number of villages based on surveys which estimated village population sizes. A census conducted in 39 villages in Oromia region, estimated an average of 1660 people per village (9). The 1994 Ethiopian Rural Household Survey covered 1477 households in 15 villages across Ethiopia (10). Assuming an average household size of 6.6 people (11), this would mean an average of at least 650 people per village. With an average of 650-1500 people per village, the number of villages in rural Ethiopia would range between 34,000 and 80,000. Discrepancies between these estimates may result from the definition of a "village". Indeed, a got, a kebele sub-structure, can be described either as a village (5) or as a

group of 3-5 villages, potentially creating confusion, as the distinction between both units is unclear in the field (12).

Eight percent of the rural Ethiopian population were in Afar and Somali regions according to the 2007 census. Therefore, assuming that the average size of a village would be the same in highlands as in lowlands, there would 11 times as many villages in highlands as villages in lowlands (6). However, the size of households and villages may vary according to regions (11). For instance, while an average 4.9 people formed a rural household in Ethiopia according to the 2007 census, this figure reached 6.4 for Afar and Somali regions according to the same census (6), and it was even estimated to be 8.1 in Somali according to another survey (11). Given the uncertainty about the number of villages to be considered, several scenarios were explored, assuming different numbers of villages and different ratios between the number of villages in lowland and highland areas (Table S3).

Serological survey. The sampling was multistage, with regions, weredas (third administrative division level), kebeles (fourth administrative division level) and villages as the first, second, third and fourth sampling units, respectively. Within each village, 20 small ruminants were supposed to be sampled. Sera were analysed using a competitive ELISA test. The survey has been described in more details by Waret-Szkuta et al. (12). A total of 13,651 animals were sampled. As 99.1% of the 4648 samples for which the animal age was recorded were from adults (i.e. >1yo), we assumed that all other samples were also from adults, and discarded the 41 samples collected from young animals. The classification of adults into further age categories was not performed reliably, therefore, serological age profiles could not be assessed. We also discarded 38 samples for which their geographical origin was unknown. This left 13,572 samples for the analysis. The kebele of origin was specified for 68.4% (9287/13572) of samples. As an average of 40 samples were collected in identified kebeles, and 82.3% (191/232) of identified kebeles counted 40 or fewer samples, samples for

which the kebele was unknown were randomly grouped, with respect to their wereda of origin, so that each group included 40, or fewer, samples. For instance, if 115 samples were collected in a given wereda and their kebele of origin was unknown, 40 samples were randomly attributed to kebele A, 40 samples to kebele B, and the remaining 35 samples to kebele C. These simulations were repeated 1000 times, and resulted in very small variation in the distribution of kebeles according to their seroprevalence (Table S1). The village of origin was specified for only 7.2% (980/13572) of samples. Within a given kebele, the number of sampled villages, and, within these villages, the number of sampled animals were obtained by dividing the number of samples by 20, as 20 samples were supposed to be collected in each village. The quotient of the Euclidian division corresponded to the number of villages within which 20 samples were collected, and the remainder to the number of animals (<20) sampled in an additional village. For instance, if 108 samples were collected from a given kebele, 20 animals were sampled in 5 villages, and 8 animals in an additional sixth village. Overall, 91.6% (643/702) of villages had serological results for 20 animals. The sample sizes for each surveyed village are reported in Tables S6-7.

Model

PPRV transmission within a village. The viral dynamics within a village was explored using a stochastic model. Transitions between compartments were modelled as binomial processes. For instance, the number of young small ruminants being infected between time t and $t+\tau$ was given by a binomial process with the number of young small ruminants that survived between time t and $t+\tau$ as the number of trials, and the risk of infection $\lambda_{r,i,t}$ as the probability of success. The number of young animals entering into the village population at time t was generated through a Poisson process with parameter $b_{r,i,t}$. Using baseline parameters (Table S2), a PPRV incursion caused an epidemic followed by extinction for all

possible values of β_r^w, N_r and ρ. In other words, PPRV dynamics in a village was epidemic, and did not result in endemicity. Under a scenario assuming a much higher turn-over of the village population (κ = 0.49, Table S2), viral endemicity within a village was only possible for the maximal value of N_r (N_r = 3473), $\rho < 0.8$ and β_r^w approaching the upper bound of its prior distribution, 10. However, the probability of an epidemic resulting in viral endemicity within a village was very low, only peaking at 0.7% for N_r = 3473 and $\beta_r^w = 10$. When accounting for a refractory period – i.e. adults recovering from infection only contributed again to new births after a fixed period of time as PPR caused abortions – PPRV could not become endemic within a village under any parameter scenario.

Simulation of the serological survey. During the field serological survey, a total number of K_r kebeles, $K_{r=L} = 79$ and $K_{r=H} = 268$, and W_r villages, $W_{r=L} = 120$ and $W_{r=H} = 582$ (Table S1), were selected in each region r. Let's $V_{r,k}$ be the number of villages surveyed in a selected kebele k (with $k \in \{1,...,K_r\}$), and $A_{r,k,v}$ the number of animals sampled in a village v (with $v \in \{1,...,V_{r,k}\}$) in kebele k in region r. At the end of each simulation, the serological survey was reproduced in each region r through the following algorithm:

(1) Initialise a vector M of integers, of size n_r, $M = \{1,...,n_r\}$, with n_r being the number of villages in region r.

(2) Set the kebele indicator $k = 1$ and village indicator $v = 1$.

(3) Randomly select a value i from M. i now refers to the *simulated* village which is paired with the *surveyed* village v in kebele k.

(4) Simulate the sampling of $A_{r,k,v}$ animals in the simulated village i, and count the number a of immune animals among them. This is achieved through a hypergeometric process $H\left(A_{r,k,v}, R_{r,i,a=2}, N_{r,i,a=2}\right)$, with $N_{r,i,a=2}$ and $R_{r,i,a=2}$ being the number of adults and immune adults in the simulated village.

(5) Simulate the number of animals *p* which tested positive. This is the sum of the number of true and false positives generated through the respective binomial processes $B(a, Se)$ and $B(A_{r,k,v} - a, 1 - Sp)$, with *Se* and *Sp* being the laboratory test sensitivity and specificity.

(6) Compute the cumulated number of tested (*T*) and positive (*P*) animals in kebele *k*:

   a. If $v=1$, $T = A_{r,k,v}$ and $P = p$

   b. If $v>1$, set *T* to $T + A_{r,k,v}$ and *P* to $P + p$

(7) Remove the value *i* from vector *M*.

(8) Depending on the value of *v*:

   a. If $v = V_{r,k}$, compute the simulated apparent seroprevalence for kebele *k*: P/T, set the kebele indicator *k* to $k+1$ and village indicator $v=1$.

   b. If $v < V_{r,k}$, set the village indicator *v* to $v+1$.

(9) If $k = K_r$ and $v = V_{r,k}$, go to (10), if not, return to (3).

(10) Compute the proportion of kebeles for which the simulated apparent seroprevalence falls within the following ranges: [0%-5%[, [5%-10%[, [10%-20%[, [20%-30%[, [30%-40%[, [40%-50%[, [50%-100%].

ABC-SMC algorithm. The algorithm started with drawing a set of parameter values – or particle – from the prior distributions. The particle served as model input to generate a simulated dataset, which was then compared to the observed dataset using a set of summary statistics, as detailed in the manuscript. If all the distances $d_{1,L}$, $d_{1,H}$, $d_{2,L}$ and $d_{2,H}$ between the simulated and observed summary statistics were below pre-defined tolerance thresholds $\varepsilon_{1,L}$, $\varepsilon_{1,H}$, $\varepsilon_{2,L}$ and $\varepsilon_{2,H}$, the particle was accepted, otherwise it was rejected. This procedure was repeated until 2000 particles were accepted. These 2000 particles formed an intermediate

distribution, in which each particle was weighted according to its probability of having been sampled, as defined in (13). A new sequence then started by drawing a particle from this intermediate distribution. Each of its parameter values was perturbed by adding to each of them a value randomly drawn from a uniform distribution $U\left[-\varphi\chi_i, \varphi\chi_i\right]$, where $\varphi = 0.2$ was the intensity of the perturbation and χ_i was the range of the marginal distribution of the parameter *i* in the intermediate distribution. Instead of having a fixed value for φ, an alternative would be to define the value of φ for each ABC-SMC sequence and parameter in order to ensure that the variance of the perturbation kernel was always twice as high as the variance of the intermediate marginal posterior distribution, as suggested by Beaumont (38). This approach and its results are presented in "Intensity of the perturbation" paragraph in the Sensitivity analysis section. If the prior probability of the perturbed particle was zero (i.e. at least one of its perturbed parameter values fell outside the range of their respective prior distributions), the process was repeated (i.e. drawing a particle and perturbing it) until obtaining a perturbed particle with a non-zero prior probability. Each tolerance threshold ε was automatically lowered to the sixtieth ($\varepsilon_{1,L}$ and $\varepsilon_{2,L}$) or eightieth ($\varepsilon_{1,H}$ and $\varepsilon_{2,H}$) percentiles of its distribution at the previous sequence. This new sequence ended when 2000 particles were accepted. New sequences were thus repeated, until predictive distributions generated by simulations reached an acceptable agreement with the observations (14), and further sequences did not further improve the model goodness-of-fit. The output of the final sequence was an approximation of the joint posterior distribution (13). Convergence of the posterior distribution and convergence towards the observed summary statistics were checked visually (15).

For any sequence *s*, the weight $w_s^{(i)}$ of a particle $\theta_s^{(i)}$ was defined as (13):

$$w_s^{(i)} = \frac{\pi\left(\theta_s^{(i)}\right)}{\sum_{j=1}^{n=2000} w_{s-1}^{(j)} K\left(\theta_{s-1}^{(j)}, \theta_s^{(i)}\right)}$$

With $\pi\left(\theta_s^{(i)}\right)$ being the prior probability of a given particle, and $K\left(\theta_{s-1}^{(j)}, \theta_s^{(i)}\right)$ the probability density for generating $\theta_s^{(i)}$ from a particle $\theta_{s-1}^{(j)}$. Given that all priors and perturbation kernels were uniform distributions, weights could be calculated as:

$$w_s^{(i)} = \frac{1}{\sum_{j=1}^{n=2000} w_{s-1}^{(j)} \mathbf{1}\left(\theta_{s-1}^{(j)}, \theta_s^{(i)}\right)}$$

With $\mathbf{1}\left(\theta_{s-1}^{(j)}, \theta_s^{(i)}\right)$ being an indicator function equal to 1 if $\theta_s^{(i)}$ could have been generated by $\theta_{s-1}^{(j)}$ (i.e. each parameter value of $\theta_s^{(i)}$ was included within the range defined by the uniform perturbation kernel around the corresponding parameter value of $\theta_{s-1}^{(j)}$), and 0 if not.

The weights were normalised, and the effective sample size (ESS) computed as follows (15):

$$\overline{w_s^{(i)}} = \frac{w_s^{(i)}}{\sum_{j=1}^{n=2000} w_s^{(j)}}$$

$$ESS = \frac{1}{\sum_{i=1}^{n=2000} \left(\overline{w_s^{(i)}}\right)^2}$$

For all intermediate sequences, the ESS was higher than 900 for 2000 particles.

Village-level reproduction number. The posterior predictive values of the village-level reproduction numbers were computed as follows. Consider a village in region *r*. It was infected at time *t=0*, causing an epidemic within the village. $N_{a,t}$ and $I_{a,t}$ were the total number of animals and the number of infected animals in age category *a* at time *t* in this village, respectively. The probability that this village did not infect any animal in another village within the same region *r* at time *t* was:

$$\exp\left[-\beta_{rr}^{b}\frac{N_r\sum_a I_{a,t}}{(n_r-2)N_r+\sum_a N_{a,t}}\right]$$

Where n_r was the number of villages in region *r*, and N_r the number of animals in a village in the absence of disease (i.e. all animals were susceptible). The probability that at least one animal was infected in a given village within the same region throughout the course of the epidemic in the primary infected village was, therefore:

$$1-\int_{t=0}^{t=\infty}\exp\left[-\beta_{rr}^{b}\frac{N_r\sum_a I_{a,t}}{(n_r-2)N_r+\sum_a N_{a,t}}\right]dt$$

The expected number of villages in region *r* infected by an infected village in the same region was given by:

$$r_{rr}=(n_r-1)\left(1-\int_{t=0}^{t=\infty}\exp\left[-\beta_{rr}^{b}\frac{N_r\sum_a I_{a,t}}{(n_r-2)N_r+\sum_a N_{a,t}}\right]dt\right)$$

The expected number of villages in region *r* infected by an infected village in region *k* (with $k\neq r$) was given by:

$$r_{kr}=(n_r-1)\left(1-\int_{t=0}^{t=\infty}\exp\left[-\beta_{kr}^{b}\frac{N_r\sum_a I_{a,t}}{(n_k-1)N_k+\sum_a N_{a,t}}\right]dt\right)$$

In the lowlands, a proportion p_v of villages was selected for vaccination, and, within each of these villages, a proportion p_a of animals were immunised. We assessed the values of p_v and p_a required for the lowland village-level reproduction number to be below 1, i.e. PPRV could not then be sustained in the region. It was assessed by calculating the dominant eigenvalue of the next generation matrix (16):

$$\begin{pmatrix} r_{VV}^{V} & r_{VU}^{V} \\ r_{UV}^{V} & r_{UU}^{V} \end{pmatrix}$$

with the following four elements:

(1) the expected number of vaccinated villages infected by an infected vaccinated village:

$$r_{VV}^{V} = \left(p_v n_L - 1 \right) \left(1 - \int_{t=0}^{t=\infty} \exp\left[-\beta_{LL}^{b} \frac{\left(1-p_a\right) N_L \sum_a I_{a,t}^{V}}{\left(n_L - 2\right) N_L + \sum_a N_{a,t}^{V}} \right] dt \right)$$

(2) the expected number of unvaccinated villages infected by an infected vaccinated village:

$$r_{VU}^{V} = \left(1-p_v\right) n_L \left(1 - \int_{t=0}^{t=\infty} \exp\left[-\beta_{LL}^{b} \frac{N_L \sum_a I_{a,t}^{V}}{\left(n_L - 2\right) N_L + \sum_a N_{a,t}^{V}} \right] dt \right)$$

(3) the expected number of vaccinated villages infected by an infected unvaccinated village:

$$r_{UV}^{V} = p_v n_L \left(1 - \int_{t=0}^{t=\infty} \exp\left[-\beta_{LL}^{b} \frac{\left(1-p_a\right) N_L \sum_a I_{a,t}^{U}}{\left(n_L - 2\right) N_L + \sum_a N_{a,t}^{U}} \right] dt \right)$$

(4) the expected number of unvaccinated villages infected by an infected unvaccinated village:

$$r_{UU}^{V} = \left[\left(1-p_v\right) n_L - 1 \right] \left(1 - \int_{t=0}^{t=\infty} \exp\left[-\beta_{LL}^{b} \frac{N_L \sum_a I_{a,t}^{U}}{\left(n_L - 2\right) N_L + \sum_a N_{a,t}^{U}} \right] dt \right)$$

The superscripts *U* and *V* applied to $N_{a,t}$ and $I_{a,t}$ referred to the unvaccinated and vaccinated status of the primary infected village.

Vaccination campaigns and temporal evolution of the immunity level. The immunity level (i.e. proportion of immunised animals) in a village a year after the first round of vaccination, during which $q_{y,r}$ young and $q_{a,r}$ adult small ruminants were vaccinated, was given by:

$$V_1 = \left(1-\kappa\right) q_{y,1} \sigma\varphi \frac{\int_{t=0}^{t=T-1} \left(1-\gamma_{a=2}\right)^t dt}{T} + \kappa q_{a,1} \sigma \left(1-\gamma_{a=2}\right)^T$$

With T=365 days, κ the proportion of adults in the population, σ the vaccine effectiveness (i.e. proportion of vaccinated animals developing lifelong immunity), $\gamma_{a=2}$ the non-PPR

mortality rate in adults, and φ the probability of a young animal becoming an adult. Parameter values were the same as in the PPRV transmission model (Table S2), except that $\gamma_{a=2}$ was expressed per day. The first part of the equation referred to the probability of a young animal remaining in the population a year after the vaccination campaign. $(1-\kappa)q_{y,r}\sigma$ was the proportion of the population that was young and effectively immunised. As births occurred all year long, $1/T$ was the probability of a young animal to be born on any day in the year preceding its vaccination. $\int_{t=0}^{t=T-1}(1-\gamma_{a=2})^t dt$ was the probability of an animal which became an adult (with probability φ) 365-*t* days after having been vaccinated to still be in the population *t* days later (i.e. 365 days after its vaccination). The second part of the equation referred to the probability of immunised adults remaining in the population. In subsequent rounds, only young animals were vaccinated, and the immunity level, a year after the round *r*, with $r \geq 2$, was expressed as:

$$V_{r\geq 2} = (1-\kappa)q_{y,1}\sigma\varphi\frac{\int_{t=0}^{t=T-1}(1-\gamma_{a=2})^t dt}{T} + V_{r-1}(1-\gamma_{a=2})^T$$

Parameters

Prior distributions. All prior distributions were uniform. β_L^w and β_H^w had the same prior distribution. Attempts have been made to estimate the basic reproduction number within a flock or a village. R_0 was thus estimated as 2.8 in Senegal (17)[1] and around 4 in Tanzania (18). These estimates should, however, be considered with caution. Final epidemic sizes were used to estimate R_0 in the Senegalese study. The population sizes were very small and estimates were

[1] Note that R_0 was reported to be equal to 6.3. However, based on the reference provided in support of R_0 calculation (36), *f* is the probability of surviving infection, and not the probability of dying of infection (f = 2/3). Also, the authors did not account for the seronegative animals when estimating the population size, which should not have been equal to 148, but to 154. After correction, y = 6/154 (the fraction that did not become infected) and x = 105/154 (the fraction that survived the epidemic), verifying $(1-f)(1-y) = (1-x)$. $R_0 = (1-f)\,log(y) / log(x) = 2.8$.

thus highly influenced by uncertainty in the number of animals reported to have survived or died of the disease. In the Tanzanian study, the estimation of R_0 was based on the age at sampling as a proxy for age of seroconversion. R_0 for rinderpest virus was estimated at 1.2-1.9 and 4, for two different viral strains and settings in East Africa (19). While this information is not directly transferrable to our study, it seemed reasonable to constrain R_0^w to be less than 10. In the absence of any information about the transmission potential of PPRV between villages, wide distributions were chosen for β_{LL}^b, β_{LH}^b and β_{HH}^b. The lower bounds of the prior distributions included 0, meaning that the corresponding inter-village reproduction numbers could be below 1. As mentioned in the Method section, r_{HL} was fixed to 0. Results with $r_{HL} = 0.5$ are presented below.

Fixed parameter values. Parameter values are shown in Table S2. Based on the estimated lengths of incubation and symptomatic periods (17, 20), the average length of the infection period τ – which was also the length of a timestep – was assumed to be equal to 10 days. The PPR case fatality rate ρ was the probability of an infected small ruminant dying of the disease. ρ is generally described to range from 20% to 80-90%, even approaching 100%, depending on the viral strain, the population at-risk, and the epidemic or endemic nature of the infection within the respective population (20-23). Field outbreak investigations conducted in affected sheep and goat flocks estimated variable case fatality rates: 18-27% in Iran (24), 25% in India (25), 40% in Egypt (26), 47% in Nigeria (27), 70% in Saudi Arabia (28). As a baseline, ρ was fixed to 50%, and the impact of variations in this parameter value on the estimation of other parameters was assessed (see below).

The parameters $b_r, \delta, \gamma_{a=1}, \gamma_{a=2}$ were computed based on:

- The number of small ruminants per village, N_r
- The proportion of adults in the population, κ

- The probability of a young becoming an adult, φ

In the absence of disease, the population dynamics in village *i* was given by:

$$N_{r,i,a=1,t} = b_{r,i,t} + (1-\delta)(1-\gamma_{a=1}) N_{r,i,a=1,t-\tau}$$

$$N_{r,i,a=2,t} = (1-\gamma_{a=2}) N_{r,i,a=2,t-\tau} + \delta(1-\gamma_{a=1}) N_{r,i,a=1,t-\tau}$$

With $N_{r,i,a=1,t}$ and $N_{r,i,a=2,t}$ being the number of young and adult small ruminants at time *t*.

From time *t* to $t+\tau$, the rate at which small ruminants left the young compartment was $\gamma_{a=1} + \delta(1-\gamma_{a=1})$, the first element referring to young animals exiting the population (e.g. harvested animals, animals dying from another disease) and the second to young animals ageing (i.e. becoming adults). The probability of a young becoming adult, φ, was, therefore, expressed as:

$$\varphi = \frac{\delta(1-\gamma_{a=1})}{\gamma_{a=1} + \delta(1-\gamma_{a=1})}$$

In order for the average time spent by animals in the young compartment to be one year, the following constraint was applied:

$$\gamma_{a=1} + \delta(1-\gamma_{a=1}) = \tau/365$$

The parameters $b_r, \delta, \gamma_{a=1}, \gamma_{a=2}$ could therefore be expressed as a function of N_r, τ, p_A:

$$b_r = \frac{\tau}{365}\frac{(1-\kappa)}{\kappa} N_r$$

$$\delta = \frac{\tau\varphi}{365 - \tau(1-\varphi)}$$

$$\gamma_{a=1} = \frac{\tau(1-\varphi)}{365}$$

$$\gamma_{a=2} = \frac{\tau}{365}\frac{(1-\kappa)}{\kappa}\varphi$$

Domestic small ruminant populations are characterised by a low adult males:females (m:f) sex ratio (*ASR*) resulting from a sex-biased harvesting of young animals. The mortality rate of young animals differed between the sexes, while the mortality rate of adults was the same for males and females. The probability of a young becoming an adult was φ_M for males and φ_F for females. The adult m:f sex ratio could thus be expressed as:

$$ASR = \frac{\varphi_M}{\varphi_F}$$

With

$$\varphi = \frac{\varphi_M}{2} + \frac{\varphi_F}{2}$$

φ_M and φ_F were given by:

$$\varphi_F = \frac{2\varphi}{(1+ASR)}$$

$$\varphi_M = \frac{2\varphi ASR}{(1+ASR)}$$

Based on the 2001-2002 agricultural census (1), the proportion of adults was κ=0.6, and the adult m:f sex ratio *ASR*=0.25 (i.e. 80% of adults were female). This meant that the annual kidding rate *k*, i.e. the number of kids per adult female and per year (i.e. $365(ASR+1)b_r/\tau$), was 0.83. While the average litter size for sheep and goats was reported to range between 1-1.5, the kidding interval was around 300 days and the survival rate of kids generally ranged between 0.5-0.9 (29-35). Annual kidding rates reported in the literature, therefore, ranged from 0.6 to 1.6. In order to explore the impact of an increase in the population turn-over on the estimation of infection parameters, we also considered κ=0.49, which corresponded to an annual kidding rate of 1.3.

The baseline probability of a young becoming an adult was φ = 0.5, which meant that 20% and 80% of young females and males were harvested before reaching one year of age (i.e.

$\varphi_F = 0.2$, $\varphi_M = 0.8$). Another value φ = 0.625 was also considered, implying $\varphi_F = 1$ and $\varphi_M = 0.25$. The range of explored values of κ and φ, resulted in a turn-over rate of 40%-51% (proportion of the population being renewed within a year), and in an average life expectancy of 2.5-4 years for adults, depending on the parameter combination.

The competitive ELISA test sensitivity and specificity were 0.945 and 0.994, respectively (37).

Sensitivity analysis

Prior distribution. The marginal posterior distributions of β_L^w could be described as a combination of two probability density functions. A unimodal Gaussian-like distribution peaking for low β_L^w values, and a uniform distribution over high β_L^w values truncated by the upper bound of the prior distribution. For high values of β_L^w, the model was insensitive to further increases in the parameter value, as it did not affect village population infectiousness and simulated seroprevalence patterns (Figure S2). While β_L^w and β_{LL}^b were strongly negatively correlated for low values of β_L^w, this was not the case for high values of β_L^w: as β_L^w increased, β_{LL}^b remained constant (Figure S5).

As the upper bound of the prior distribution of β_L^w increased, the upper bound of its marginal posterior distribution also increased, resulting in higher β_L^w posterior median and credible intervals, and lower β_{LL}^b posterior median. However, the maximums a posteriori (MAPs) and the shape of the marginal posterior distributions of β_L^w and β_{LL}^b were unchanged. Likewise, the threshold for PPRV elimination remained the same.

Number of villages and village population size. Given the uncertainty about the number of villages within which lowland and highland small ruminant populations were divided, we

explored different scenarios, varying the overall number of villages and the ratio L:H between the number of villages in lowlands and highlands (Table S3). As the overall highland and lowland populations remained constant, changing the number of villages also altered village population sizes (Table S3). As the ratio L:H doubled, r_{LH}^{b} was divided by two. The upper bound of the credible interval of r_{HH}^{b} remained lower than 1, and the vaccination coverage required for PPRV elimination remained similar across scenarios, as immunising $p_a \in [0.37, 0.39]$ animals in $p_v \in [0.71, 0.73]$ villages would prevent viral circulation in the lowlands.

PPR case fatality rate and demographic profiles. To explore the impact of these parameters on the model outcome, the model was fitted to the serological results in lowlands only. The number n_L of lowland villages was fixed to 5000. Changes in the values of these parameters altered the posterior median estimates of β_L^w and β_{LL}^b (Table S4) in comparison to the baseline scenario (Table 1). However, the shapes of the marginal posterior distributions were unchanged: the highest posterior density of the level of within-village transmission in the lowlands was concentrated at low β_L^w values, and a second, low probability and almost uniform mode was located at high β_L^w values. The maximum a posteriori (MAP) was about the same as for the baseline scenario, $\text{MAP} \in [1.33, 1.37]$, only the relative densities of both posterior components were modified. The vaccination coverage required for PPRV elimination remained similar across scenarios, as immunising $p_a \in [0.38, 0.41]$ animals in $p_v \in [0.69, 0.73]$ villages would prevent viral circulation in lowlands.

Relative strength of mixing between highlands and lowlands. As mentioned in the main text, β_{HL}^b was expressed as $\beta_{HL}^b = r_{HL}\beta_{LH}^b \left(P_H / P_L\right)$, with r_{HL}, the relative strength of mixing. In the baseline scenario, r_{HL} was fixed to 0, i.e. highland animals could not infect lowland

animals. When fixing r_{HL} to 0.5 (with n_L = 10,000 and n_H = 100,000), r_{LL}^b decreased, from 1.49 (95%CrI:1.27-2.01) for $r_{HL} = 0$, to 1.38 (95%CrI:1.05-1.89). Due to the lower value of r_{LL}^b, the overall proportion of lowland small ruminants that would need to be immunised to prevent viral circulation was reduced by 10%, compared to a scenario for which $r_{HL} = 0$.

Vaccine effectiveness. Reducing vaccine effectiveness σ (i.e. the probability of a vaccinated animal to develop lifelong protective immunity) increased the number of small ruminants that would need to be vaccinated over the 4 rounds of vaccination by 11% for σ = 0.9, and 25% for σ = 0.8 (Table S5).

Intensity of the perturbation. With φ = 0.2, the variance of the perturbation kernels was lower than the variance of the intermediate marginal posterior distributions (the ratio of the two variances ≤ 0.8). We re-estimated parameters by defining the value of φ for each ABC-SMC sequence and parameter in order to ensure that the variance of the perturbation kernel was always twice as high as the variance of the intermediate marginal posterior distribution (38). This resulted in a higher intensity of the perturbation, φ then ranging from 0.3 to 0.8 depending on the parameter and sequence, but it did not affect the parameter estimates (Table S8).

Table S1: Serological survey results according to two partitioning criteria. Afar and Somali: villages in Afar and Somali (or with an altitude lower than 1000m) are pastoral, others are sedentary; p_k: seroprevalence within a kebele; as the attribution of some samples to kebeles was simulated, the mean and the range of the proportion of kebeles with a given seroprevalence are shown.

	Pastoral, lowlands		Sedentary, highlands	
	Afar, Somali	<1000m	Others than Afar, Somali	>1000m
no. samples	2115	2548	11457	11024
no. positives (%)	352 (16.6%)	439 (17.2%)	522 (4.6%)	435 (3.9%)
no. villages	120	142	582	560
no. kebeles	79	86	268	261
Prop. kebeles				
$0\% \leq p_k \leq 5\%$	25.3% (25.3%-25.3%)	24.4% (24.4%-24.4%)	72.1% (70.9%-73.5%)	73.6% (72.4%-74.7%)
$6\% \leq p_k \leq 10\%$	10.1% (10.1%-10.1%)	10.5% (10.5%-10.5%)	12.4% (10.4%-14.2%)	12.3% (10%-13.8%)
$11\% \leq p_k \leq 20\%$	25.3% (25.3%-25.3%)	25.6% (25.6%-25.6%)	7.3% (6.3%-8.6%)	6.7% (5.7%-8%)
$21\% \leq p_k \leq 30\%$	20.3% (20.3%-20.3%)	19.8% (19.8%-19.8%)	3.5% (3.4%-3.7%)	3.2% (3.1%-3.4%)
$31\% \leq p_k \leq 40\%$	8.9% (8.9%-8.9%)	9.3% (9.3%-9.3%)	0.4% (0.4%-0.4%)	0% (0%-0%)
$41\% \leq p_k \leq 50\%$	2.5% (2.5%-2.5%)	3.5% (3.5%-3.5%)	2.6% (2.6%-2.6%)	2.3% (2.3%-2.3%)
$p_k \geq 51\%$	7.6% (7.6%-7.6%)	7% (7%-7%)	1.9% (1.9%-1.9%)	1.9% (1.9%-1.9%)

Table S2: Parameter values. $\gamma_{a=1}$, $\gamma_{a=2}$, δ, k and l were computed based on values of τ, κ and φ. References and justification of parameter values are provided in the supplementary text; the turn-over rate refers to the proportion of the population which is renewed in a year.

Parameters	Description (unit)	Values					
		Baseline	*Scenarios*				
Infection parameters							
τ	Infection period (days)	10					
ρ	PPR case fatality rate	0.5	*0.5*	*0.5*	*0.5*	*0.2*	*0.8*
Demographic parameters							
κ	Proportion of adults	0.6	*0.6*	*0.49*	*0.49*	*0.6*	*0.6*
φ	probability of a young becoming adult	0.5	*0.625*	*0.5*	*0.625*	*0.5*	*0.5*
$\gamma_{a=1}$	Mortality rate, young ($x10^{-2}$)	1.37	*1.03*	*1.37*	*1.03*	*1.37*	*1.37*
$\gamma_{a=2}$	Mortality rate, adults ($x10^{-2}$)	0.91	*1.14*	*1.43*	*1.78*	*0.91*	*0.91*
δ	Ageing parameter ($x10^{-2}$)	1.39	*1.73*	*1.39*	*1.73*	*1.39*	*1.39*
k	Annual kidding rate	0.83	*0.83*	*1.30*	*1.30*	*0.83*	*0.83*
l	Life expectancy of adults (years)	4	*3.4*	*2.9*	*2.5*	*4*	*4*
	Turn-over rate	40%	*40%*	*51%*	*51%*	*40%*	*40%*

Table S3: Impact of variations in the number of villages on parameter posterior estimates and reproduction number posterior predictive values. n_L and n_H refer to the number of villages in the lowlands and highlands; the human rural populations in the lowlands and highlands were estimated to 4.12 and 47.37 million, and the small ruminant population sizes to 17.4 and 27.2 million in both regions; posterior median and 95% credible intervals are shown; posterior predictive values of animal- and village-level reproduction numbers were calculated based on the inferred posterior distribution.

	Number of villages			
n_L	5000	5000	10,000	10,000
n_H	25,000	50,000	50,000	100,000
$n_L : n_H$	1:5	1:10	1:5	1:10
Number of people/village				
Lowland	824	824	412	412
Highland	1895	947	947	474
Number of small ruminants/village				
Lowland	3473	3473	1736	1736
Highland	1087	543	543	272
β_L^w	1.54 (1.28-9.32)	1.53 (1.27-9.34)	1.55 (1.28-9.27)	1.56 (1.26-9.45)
β_H^w	5.70 (1.88-9.70)	5.91 (1.85-9.87)	6.01 (1.92-9.78)	6.19 (1.85-9.73)
β_{LL}^b (x10^{-3})	0.54 (0.37-0.97)	0.54 (0.37-0.97)	1.07 (0.74-1.94)	1.08 (0.75-1.94)
β_{LH}^b (x10^{-3})	0.13 (0.03-0.27)	0.26 (0.07-0.51)	0.25 (0.05-0.49)	0.50 (0.08-1.05)
β_{HH}^b (x10^{-3})	0.28 (0.01-0.84)	0.57 (0.03-1.61)	0.58 (0.03-1.73)	1.19 (0.06-3.47)
$R_{0,L}^w$	1.52 (1.26-9.22)	1.51 (1.26-9.23)	1.53 (1.26-9.17)	1.54 (1.24-9.35)
$R_{0,H}^w$	5.63 (1.86-9.60)	5.84 (1.83-9.76)	5.94 (1.90-9.68)	6.11 (1.83-9.63)
r_{LL}^b	1.51 (1.25-2.03)	1.50 (1.27-2.03)	1.49 (1.27-2.01)	1.49 (1.27-2.01)
r_{LH}^b	0.57 (0.14-1.25)	1.13 (0.27-2.46)	0.55 (0.12-1.12)	1.09 (0.20-2.36)
r_{HH}^b	0.30 (0.01-0.88)	0.31 (0.01-0.86)	0.31 (0.02-0.92)	0.32 (0.01-0.91)

Table S4: Impact of variations in the case fatality rate on parameter posterior estimates and reproduction number posterior predictive values. Only lowland transmission parameters were estimated, using 5000 villages; posterior median and 95% credible intervals are shown; posterior predictive values of animal- and village-level reproduction numbers were calculated based on the inferred posterior distribution; ρ: PPR case fatality rate; κ: proportion of adults; φ: probability of a young becoming adult.

	PPR case fatality rate and demographic parameters					
ρ	0.5	0.2	0.8	0.5	0.5	0.5
κ	0.6	0.6	0.6	0.6	0.49	0.49
φ	0.5	0.5	0.5	0.625	0.5	0.625
β_L^w	1.88 (1.28-9.76)	1.53 (1.28-9.74)	3.33 (1.26-9.58)	1.85 (1.28-9.79)	1.40 (1.24-9.21)	1.40 (1.26-9.17)
β_{LL}^b ($\times 10^{-3}$)	0.47 (0.37-0.94)	0.64 (0.38-1.18)	0.44 (0.36-0.80)	0.47 (0.36-0.96)	0.64 (0.36-1.11)	0.67 (0.37-1.08)
$R_{0,L}^w$	1.86 (1.26-9.65)	1.51 (1.26-9.63)	3.30 (1.25-9.47)	1.83 (1.26-9.68)	1.38 (1.23-9.10)	1.38 (1.25-9.07)
r_{LL}^b	1.48 (1.26-2.00)	1.52 (1.31-1.97)	1.51 (1.24-2.24)	1.48 (1.26-2.10)	1.58 (1.26-2.32)	1.64 (1.28-2.26)

Table S5: Within-village vaccination coverage and number of vaccine doses required to eliminate PPRV from the lowlands. The number of animals to be vaccinated within a village and within the entire lowland region was computed to ensure that 37% animals were immunised a year after a vaccination round, in 70.7% villages. σ: vaccine effectiveness; υ: within-village vaccination coverage; doses: overall number of vaccine doses for the lowlands; P_c: ratio between the number of vaccinated animals suggested here and assuming a full vaccination coverage (round 1: all young and adult animals, round 2-4: all young animals).

	Turn-over: 40%			Turn-over: 51%		
	σ = 1	σ = 0.9	σ = 0.8	σ = 1	σ = 0.9	σ = 0.8
Round 1						
υ, young	61.7%	68.5%	77.1%	75.5%	83.9%	94.4%
υ, adults	61.7%	68.5%	77.1%	75.5%	83.9%	94.4%
υ, all	61.7%	68.5%	77.1%	75.5%	83.9%	94.4%
Doses (x10^6)	7.6	8.4	9.5	9.3	10.3	11.6
Round 2						
υ, young	61.6%	68.6%	77.0%	75.5%	83.9%	94.4%
υ, adults	0%	0%	0%	0%	0%	0%
υ, all	24.6%	27.4%	30.8%	38.5%	42.8%	48.1%
Doses (x10^6)	3.0	3.4	3.8	4.7	5.3	5.9
Round 3						
υ, young	61.7%	68.5%	77.1%	75.5%	83.9%	94.4%
υ, adults	0%	0%	0%	0%	0%	0%
υ, all	24.7%	27.4%	30.8%	38.5%	42.8%	48.1%
Doses (x10^6)	3.0	3.4	3.8	4.7	5.3	5.9
Round 4						
υ, young	61.6%	68.5%	77.1%	75.5%	83.9%	94.4%
υ, adults	0%	0%	0%	0%	0%	0%
υ, all	24.6%	27.4%	30.8%	38.5%	42.8%	48.1%
Doses (x10^6)	3.0	3.4	3.8	4.7	5.3	5.9
All rounds						
Doses (x10^6)	16.6	18.5	20.8	23.5	26.1	29.3
P_c	43.6%	48.4%	54.5%	53.4%	59.3%	66.7%

Table S6: Sample sizes in lowland villages. Each row refers to a surveyed village, *n*: the number of animals sampled in each village, ID: the ID of the corresponding kebele.

	n	ID		*n*	ID
v1	20	1	v61	20	38
v2	20	1	v62	20	38
v3	20	2	v63	20	39
v4	20	2	v64	20	40
v5	20	3	v65	20	40
v6	20	3	v66	20	41
v7	20	4	v67	20	42
v8	20	4	v68	7	42
v9	20	5	v69	10	43
v10	20	5	v70	18	44
v11	20	6	v71	18	45
v12	20	6	v72	18	46
v13	20	7	v73	13	47
v14	20	7	v74	6	48
v15	20	8	v75	10	49
v16	20	8	v76	7	50
v17	20	9	v77	12	51
v18	20	9	v78	9	52
v19	20	10	v79	8	53
v20	20	10	v80	10	54
v21	11	11	v81	11	55
v22	20	12	v82	7	56
v23	20	12	v83	20	57
v24	20	13	v84	20	57
v25	20	13	v85	20	58
v26	20	14	v86	20	58
v27	20	14	v87	20	59
v28	20	15	v88	20	59
v29	18	15	v89	20	60
v30	20	16	v90	20	60
v31	20	16	v91	20	61
v32	20	17	v92	20	61
v33	4	17	v93	18	62
v34	20	18	v94	20	63
v35	16	18	v95	20	63
v36	20	19	v96	20	64
v37	20	19	v97	20	64
v38	20	20	v98	20	65
v39	20	21	v99	20	65
v40	20	21	v100	20	66
v41	20	22	v101	20	66
v42	20	23	v102	10	67
v43	20	23	v103	20	68
v44	9	24	v104	20	69
v45	17	25	v105	20	69
v46	20	26	v106	15	70
v47	4	26	v107	20	71
v48	12	27	v108	20	72
v49	18	28	v109	20	73
v50	20	29	v110	20	74
v51	4	29	v111	20	74
v52	10	30	v112	20	75
v53	14	31	v113	20	75
v54	10	32	v114	20	76
v55	20	33	v115	20	76
v56	2	33	v116	20	77
v57	20	34	v117	20	77
v58	20	35	v118	20	78
v59	20	36	v119	20	78
v60	20	37	v120	19	79

Table S7: Sample sizes in highland villages. Each row refers to a surveyed village, *n*: the number of animals sampled in each village, ID: the ID of the corresponding kebele.

	n	ID		*n*	ID		*n*	ID		*n*	ID		*n*	ID
v1	20	1	v71	20	41	v141	20	76	v211	20	110	v281	20	144
v2	13	1	v72	20	42	v142	20	77	v212	20	110	v282	20	144
v3	20	2	v73	20	42	v143	20	77	v213	20	111	v283	20	144
v4	13	2	v74	20	42	v144	20	78	v214	20	111	v284	20	144
v5	20	3	v75	20	42	v145	20	78	v215	20	112	v285	20	145
v6	11	3	v76	20	43	v146	20	79	v216	20	112	v286	20	145
v7	20	4	v77	20	43	v147	20	79	v217	20	113	v287	20	146
v8	13	4	v78	20	43	v148	20	80	v218	20	113	v288	20	147
v9	20	5	v79	20	44	v149	20	80	v219	20	114	v289	20	147
v10	12	5	v80	20	44	v150	20	81	v220	20	114	v290	20	148
v11	20	6	v81	20	44	v151	20	81	v221	20	115	v291	20	148
v12	20	7	v82	19	44	v152	20	82	v222	20	115	v292	20	149
v13	20	8	v83	20	45	v153	20	82	v223	20	116	v293	20	149
v14	20	9	v84	20	45	v154	20	83	v224	20	116	v294	20	150
v15	20	10	v85	2	45	v155	20	83	v225	20	117	v295	20	150
v16	20	11	v86	20	46	v156	20	84	v226	20	117	v296	20	150
v17	20	12	v87	20	47	v157	20	84	v227	20	118	v297	20	150
v18	20	13	v88	20	48	v158	20	85	v228	20	118	v298	20	151
v19	20	14	v89	20	49	v159	20	85	v229	20	119	v299	20	151
v20	20	15	v90	20	50	v160	20	86	v230	20	119	v300	20	151
v21	20	16	v91	20	51	v161	20	86	v231	20	120	v301	20	151
v22	20	16	v92	20	51	v162	20	87	v232	20	120	v302	20	152
v23	20	17	v93	20	52	v163	20	87	v233	20	121	v303	20	152
v24	18	17	v94	12	52	v164	20	88	v234	20	121	v304	20	152
v25	20	18	v95	20	53	v165	20	88	v235	20	122	v305	20	152
v26	20	19	v96	20	53	v166	20	89	v236	20	122	v306	20	153
v27	20	20	v97	20	54	v167	20	89	v237	20	123	v307	20	153
v28	20	21	v98	20	54	v168	20	90	v238	20	123	v308	20	153
v29	20	22	v99	20	55	v169	20	90	v239	20	124	v309	20	153
v30	20	23	v100	20	55	v170	20	91	v240	20	124	v310	20	154
v31	20	23	v101	20	56	v171	20	91	v241	20	125	v311	20	154
v32	20	24	v102	20	56	v172	20	92	v242	20	126	v312	20	154
v33	20	24	v103	20	57	v173	20	92	v243	20	127	v313	20	154
v34	20	25	v104	20	57	v174	20	93	v244	20	127	v314	20	155
v35	20	25	v105	20	58	v175	20	93	v245	20	128	v315	20	155
v36	20	26	v106	20	58	v176	20	94	v246	20	129	v316	20	155
v37	20	26	v107	20	59	v177	20	94	v247	20	129	v317	20	155
v38	20	27	v108	5	59	v178	20	95	v248	20	130	v318	20	156
v39	20	27	v109	20	60	v179	20	95	v249	20	130	v319	20	156
v40	20	27	v110	20	60	v180	20	95	v250	20	130	v320	20	157
v41	20	27	v111	20	61	v181	20	95	v251	20	130	v321	20	157
v42	20	28	v112	20	61	v182	20	95	v252	20	131	v322	20	158
v43	20	28	v113	20	62	v183	20	95	v253	20	131	v323	20	158
v44	20	28	v114	20	62	v184	20	96	v254	20	132	v324	20	159
v45	20	28	v115	20	63	v185	20	96	v255	20	132	v325	20	159
v46	20	29	v116	20	63	v186	19	97	v256	20	132	v326	20	160
v47	20	29	v117	20	64	v187	20	98	v257	20	132	v327	20	160
v48	20	30	v118	20	64	v188	20	98	v258	20	133	v328	20	161
v49	20	30	v119	20	65	v189	20	99	v259	20	133	v329	20	161
v50	20	31	v120	20	65	v190	20	99	v260	20	134	v330	20	162
v51	20	31	v121	20	66	v191	20	100	v261	20	134	v331	20	162
v52	19	32	v122	20	67	v192	11	100	v262	20	135	v332	20	163
v53	20	33	v123	20	67	v193	20	101	v263	20	135	v333	20	163
v54	11	33	v124	20	68	v194	20	101	v264	20	136	v334	20	164
v55	20	34	v125	14	68	v195	20	102	v265	20	136	v335	2	164
v56	20	34	v126	20	69	v196	20	102	v266	20	136	v336	20	165
v57	20	35	v127	19	69	v197	20	103	v267	20	136	v337	20	165
v58	20	35	v128	20	70	v198	20	103	v268	20	137	v338	20	166
v59	20	36	v129	20	70	v199	20	104	v269	20	137	v339	20	166
v60	20	36	v130	20	71	v200	20	104	v270	20	138	v340	20	167
v61	20	37	v131	19	71	v201	20	105	v271	20	138	v341	20	168
v62	20	37	v132	20	72	v202	20	105	v272	20	139	v342	20	169
v63	20	38	v133	17	72	v203	20	106	v273	20	139	v343	20	169
v64	20	38	v134	20	73	v204	20	106	v274	20	140	v344	20	170
v65	20	39	v135	10	73	v205	20	107	v275	20	140	v345	20	170
v66	20	39	v136	20	74	v206	20	107	v276	20	141	v346	20	171
v67	20	40	v137	13	74	v207	20	108	v277	20	141	v347	20	171
v68	20	40	v138	20	75	v208	20	108	v278	20	142	v348	20	172
v69	18	40	v139	20	75	v209	20	109	v279	20	142	v349	20	172
v70	20	41	v140	20	76	v210	20	109	v280	20	143	v350	20	173

	n	ID
v351	20	173
v352	20	173
v353	20	173
v354	20	173
v355	20	174
v356	20	174
v357	20	174
v358	20	174
v359	20	174
v360	20	175
v361	20	175
v362	20	175
v363	20	175
v364	20	175
v365	20	176
v366	20	176
v367	20	176
v368	20	177
v369	20	178
v370	20	178
v371	20	178
v372	20	178
v373	20	178
v374	20	179
v375	20	179
v376	20	180
v377	20	181
v378	20	182
v379	20	182
v380	20	183
v381	20	183
v382	20	184
v383	20	185
v384	20	186
v385	20	187
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v581	20	268
v582	20	268

Table S8: Parameter posterior estimates and posterior predictive values of reproduction numbers with a fixed or a flexible intensity of the perturbation kernel. When fixed, the intensity φ of the perturbation was 0.2 (results presented in Table 1). When flexible, φ was defined for each parameter and ABC-SMC sequence in order to ensure that the variance of the perturbation kernel was always twice as high as the variance of the intermediate marginal posterior distribution; β: the number of effective contacts per animal over a 10 day-period – the length of the infection period; β_r^w refers to PPRV transmission within a village in region *r*, and β_{kr}^b to inter-village transmission from region *k* to *r*; likewise, $R_{0,r}^w$ is the within-village reproduction number in region *r*, and r_{kr}^b the village-level reproduction number from region *k* to *r*; median and 95% credible intervals were computed.

	Post. median (95%CrI) Fixed φ	Post. median (95%CrI) Flexible φ
β_L^w	1.56 (1.26-9.45)	1.58 (1.28-9.57)
β_H^w	6.19 (1.85-9.73)	5.94 (1.92-9.74)
β_{LL}^b (x10^{-3})	1.08 (0.75-1.94)	1.04 (0.74-1.87)
β_{LH}^b (x10^{-3})	0.50 (0.08-1.05)	0.51 (0.09-1.03)
β_{HH}^b (x10^{-3})	1.19 (0.06-3.47)	1.15 (0.06-3.45)
$R_{0,L}^w$	1.54 (1.24-9.35)	1.56 (1.26-9.47)
$R_{0,H}^w$	6.11 (1.83-9.63)	5.88 (1.89-9.63)
r_{LL}^b	1.49 (1.27-2.01)	1.48 (1.26-2.00)
r_{LH}^b	1.09 (0.20-2.36)	1.14 (0.21-2.38)
r_{HH}^b	0.32 (0.01-0.91)	0.31 (0.02-0.93)

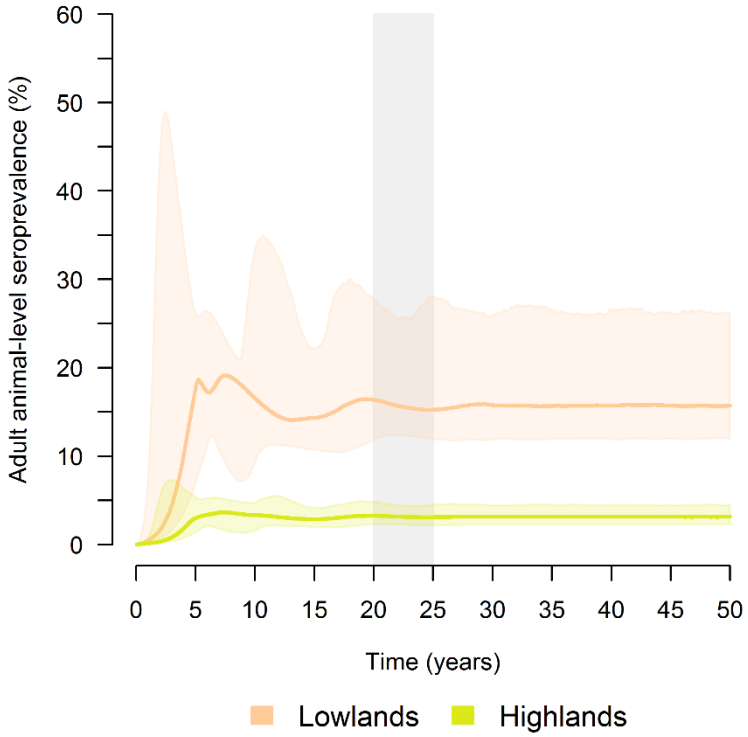

Figure S1: Evolution of the adult animal-level seroprevalence as a function of time. The median proportion of immune adults (solid line), 5th and 95th percentiles (coloured envelop) are shown for both highlands and lowlands; the grey shaded area corresponds to the period during which the serological survey was simulated (20-25 years) following PPRV incursion at t=0.

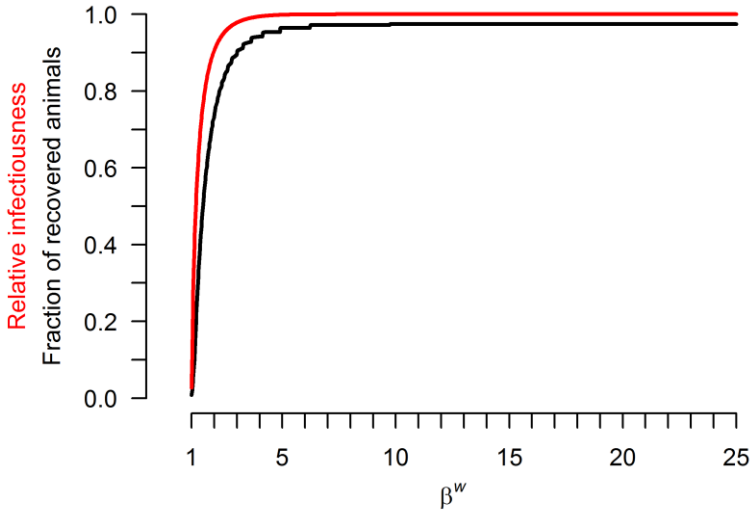

Figure S2: Impact of changes in PPRV transmission on village infectiousness and fraction of recovered animals. β^w : number of effective contacts per unit of time within a village; fraction of recovered animals: proportion of immune animals in the village population at the end of an epidemic which affected an initially fully susceptible population; the infectiousness referred to the potential of a village to infect others.

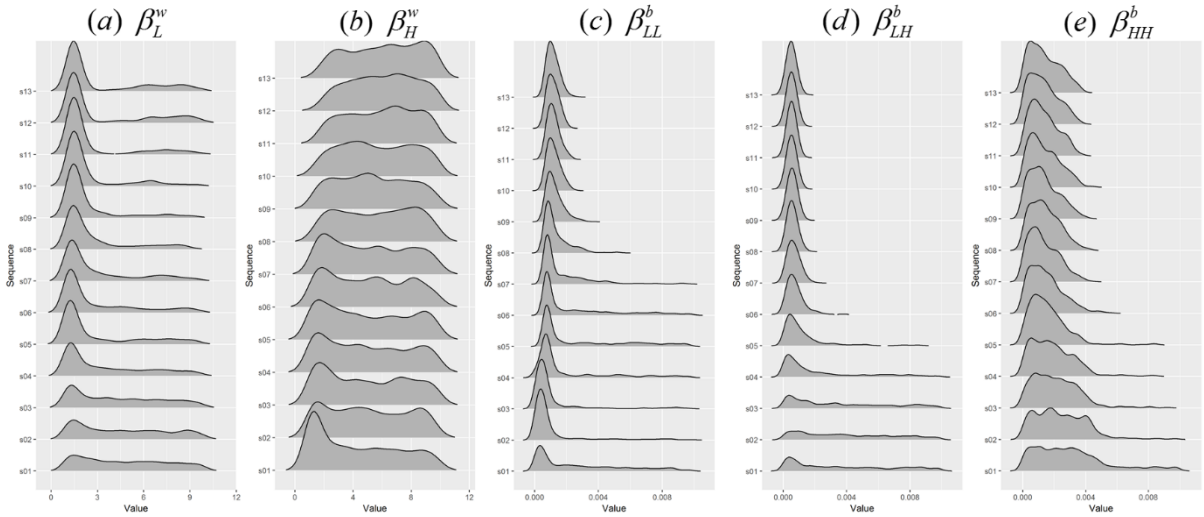

Figure S3: Intermediate marginal posterior distributions. Under the baseline scenario.

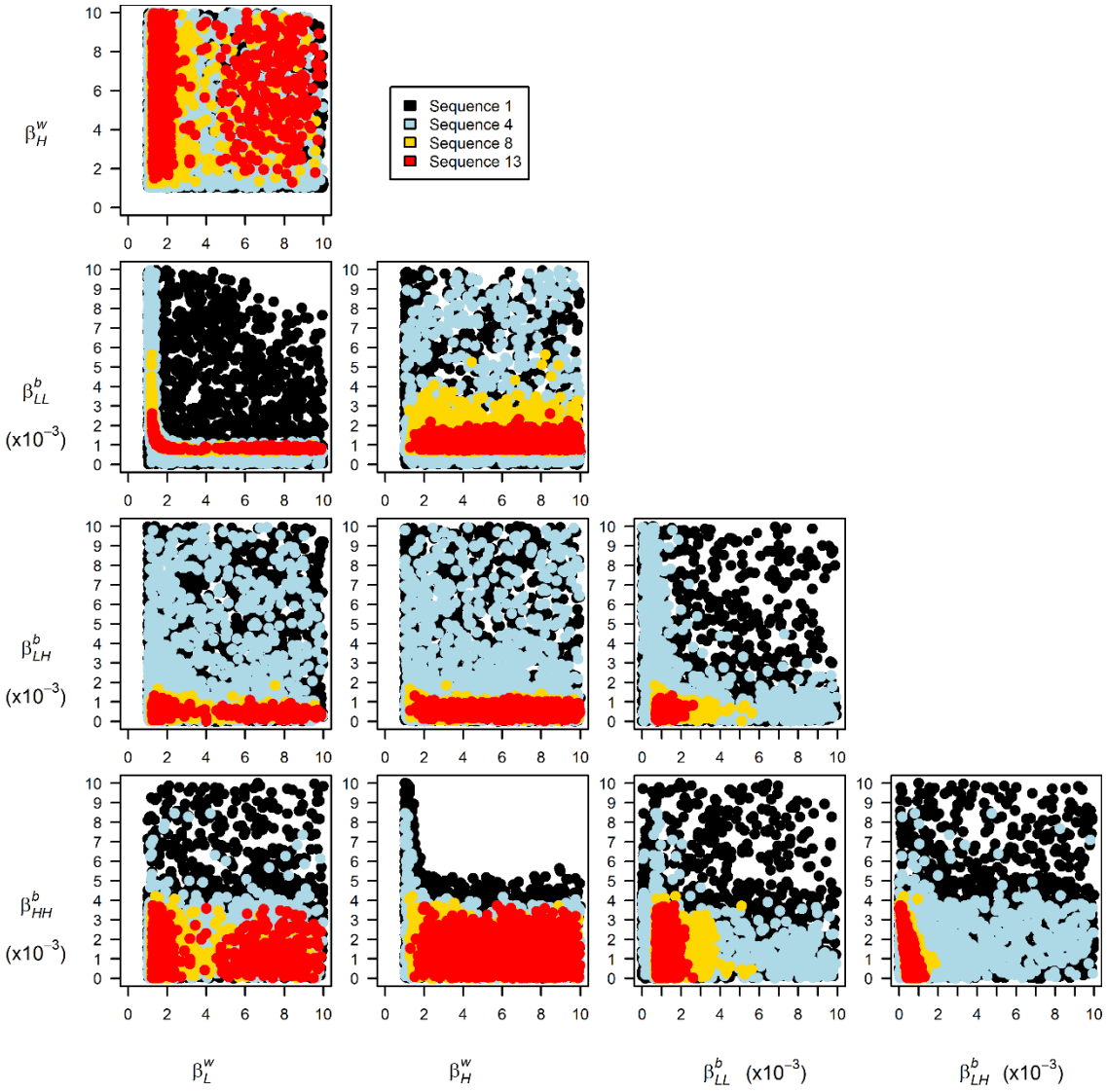

Figure S4: Two-dimensional scatterplots of intermediate marginal posterior distributions. Distributions were estimated under the baseline scenario. The particles from the first sequence are in black, particles from the fourth sequence in blue, particles from the eighth sequence in yellow and those from the thirtieth and last sequence in red.

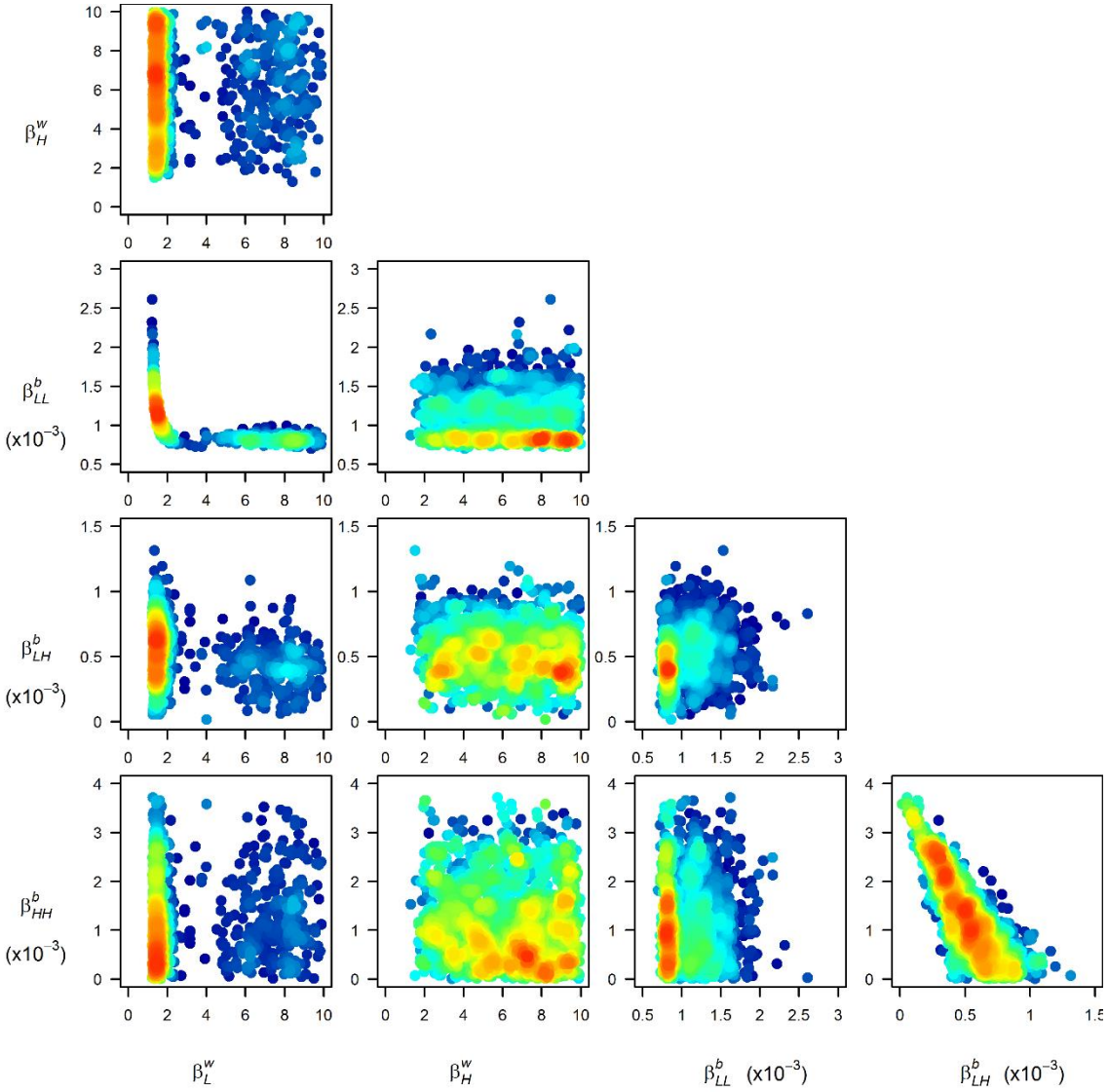

Figure S5: Correlation between marginal posterior density distributions. Density is represented through the colour spectrum from dark blue (low density) to red (high density); the posterior distribution was estimated under the baseline scenario.

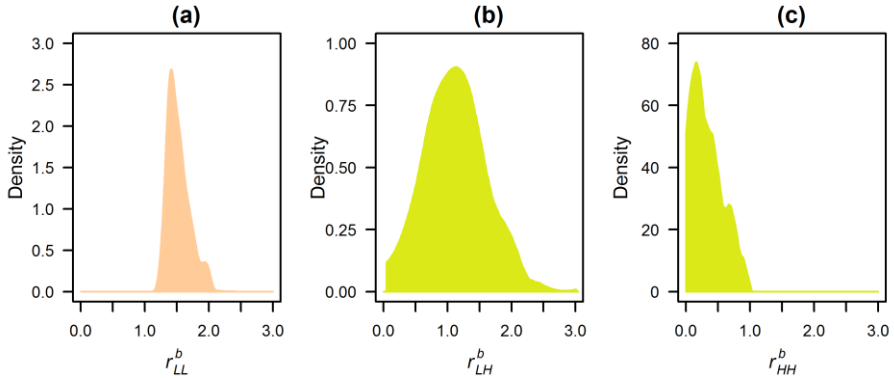

Figure S6: Posterior predictive distribution of village-level reproduction numbers.

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