Supplementary information

Dynamics of a morbillivirus at the domestic-wildlife interface: Canine Distemper Virus in domestic dogs and lions

Mafalda Viana*, Sarah Cleaveland, Jason Matthiopoulos, Jo Halliday, Craig Packer, Meggan E. Craft, Katie Hampson, Anna Czupryna, Andrew P. Dobson, Edward J. Dubovi, Eblate Ernest, Robert Fyumagwa, Richard Hoare, J. Grant C. Hopcraft, Daniel L. Horton, Magai T. Kaare, Felix Lankester, Christine Mentzel, Titus Mlengeya, Imam Mzimbiri, Emi Takahashi, Brian Willet, Daniel T. Haydon & Tiziana Lembo*

**Corresponding authors:* mafalda.viana@glasgow.ac.uk and tiziana.lembo@glasgow.ac.uk

Boyd Orr Centre for Population and Ecosystem Health, Institute of Biodiversity, Animal Health and Comparative Medicine, College of Medical, Veterinary and Life Sciences, University of Glasgow, G12 8QQ, UK

S1. Data

Lion data

Lion populations in the Serengeti National Park and Ngorongoro Conservation Area have been continuously monitored by the Serengeti Lion Project since 1966. Most lions included in this study have been observed since birth and are recognised from natural markings and whisker-spot patterns (1-3). Consequently, their precise date of birth is known. For unknown lions, sampled as part of other interventions (such as snare removal), ages were estimated on the basis of nose coloration (4).

Serum samples for serological testing were opportunistically collected as part of the Serengeti National Park management or research interventions (e.g. fitting/removing radio-collars, snare removals and wound treatment; [5]) led by Tanzania National Parks and Tanzania Wildlife Research Institute and Ngorongoro Conservation Area Authority. To maximise samples and ensure independence of observations, if a lion was sampled multiple times and all samples tested positive for canine distemper virus (CDV) antibodies, only the first sample was included in the analyses. Conversely, if all samples were seronegative, only the last sample was included. Finally, if the first sample was seronegative but the second was seropositive, we included both samples in the analysis but the birth year corresponding to the second sample was considered to be the sample year of the first sample. Figure S1 (left panel) shows the individual infection profile of lions from birth to sampling and Table S1 contains the number of samples included in the analyses.

Dog data

Domestic dog populations surrounding the Serengeti National Park have been intensively studied since the early 1990s and serological surveys have been conducted since 1992. Dogs are sampled during central-point and house-to-house vaccination campaigns (6) and, in unvaccinated areas, during randomised household surveys. At the time of sampling, information on the age of each dog sampled is obtained by questioning the owner. Previous longitudinal studies in East Africa demonstrated that owner-reported ages are reliable

when evaluated as part of specific research studies (7-9). The reliability of ages recorded during routine vaccination campaigns is likely to be less certain. The dog sampling protocol is described in (10).

To ensure that only data from unvaccinated dogs were analysed, we included only dogs from villages that had never been vaccinated and dogs from vaccinated villages that were i) sampled before the onset of vaccination in a given village; or ii) born after the previous vaccination campaign. Given that dogs in vaccinated areas receive vaccination also against rabies and canine parvovirus (CPV), for dogs older than 1 year and originating from vaccinated areas, sera were also tested for antibodies against rabies and CPV. Dogs that had a negative antibody titre against at least one of the three pathogens were considered unvaccinated and included in the analyses. Very young pups (0 - 3 months) were excluded from the analyses to avoid misclassification of CDV status due to possible maternal antibodies. Figure S1 (right panel) shows the individual infection profile of dogs from birth to sampling and Table S1 contains the number of samples included in the analyses.

Serological assays

All CDV serology was carried out using neutralisations assays. Sera were analysed at Intervet UK, Animal Health Diagnostic Center at Cornell (New York, USA) and University of Glasgow (UK). Protocols were broadly similar in all laboratories, including viral strains used for the assay. Previous studies demonstrated comparable results between the Intervet and Cornell tests (10). We used a cut-off titer value equivalent to a 1:16 dilution to evaluate prior exposure. This value was the minimum dilution consistently used across all samples and is consistent with other studies of CDV exposure in wild carnivore species (11-13). Sera from older dogs originating from vaccinated areas and with uncertain vaccination status were additionally tested for antibodies against rabies and CPV (see above). Antibody titres for rabies were determined by fluorescent antibody virus neutralisation assay (FAVN) (14), and for CPV by hemagglutination inhibition (HAI) testing (15). FAVN testing was performed at the Animal Health and Veterinary Laboratories Agency (UK). HAI testing was conducted at Intervet and the Cornell Laboratory.

Table S1: Number of serum samples from Ngorongoro and Serengeti lions and unvaccinated domestic dogs sampled in areas surrounding the Serengeti National Park available for canine distemper virus serology. Dog sample sizes are also subdivided depending on whether the dog originated from an unvaccinated or vaccinated village.

		Non-vaccinated domestic dogs		
Year	**Lions**	**Total**	**Non-vaccinated villages**	**Vaccinated villages**
1984	24	-	-	-
1985	112	-	-	-
1986	24	-	-	-
1987	75	-	-	-
1988	-	-	-	-
1989	16	-	-	-
1990	3	-	-	-
1991	17	-	-	-
1992	5	223	223	-
1993	9	155	155	-
1994	70	240	240	-
1995	-	-	-	-
1996	-	181	180	1
1997	11	643	458	185
1998	25	921	658	263
1999	6	486	455	31
2000	11	90	23	67
2001	21	250	134	116
2002	8	151	19	132
2003	16	550	508	42
2004	14	1073	586	487
2005	19	403	35	368
2006	23	672	188	484
2007	1	307	12	295
2008	5	84	-	84
2009	3	157	-	157
2010	6	133	-	133
2011	10	72	-	72
2012	1	75	-	75
Total	535	6866	3874	2992

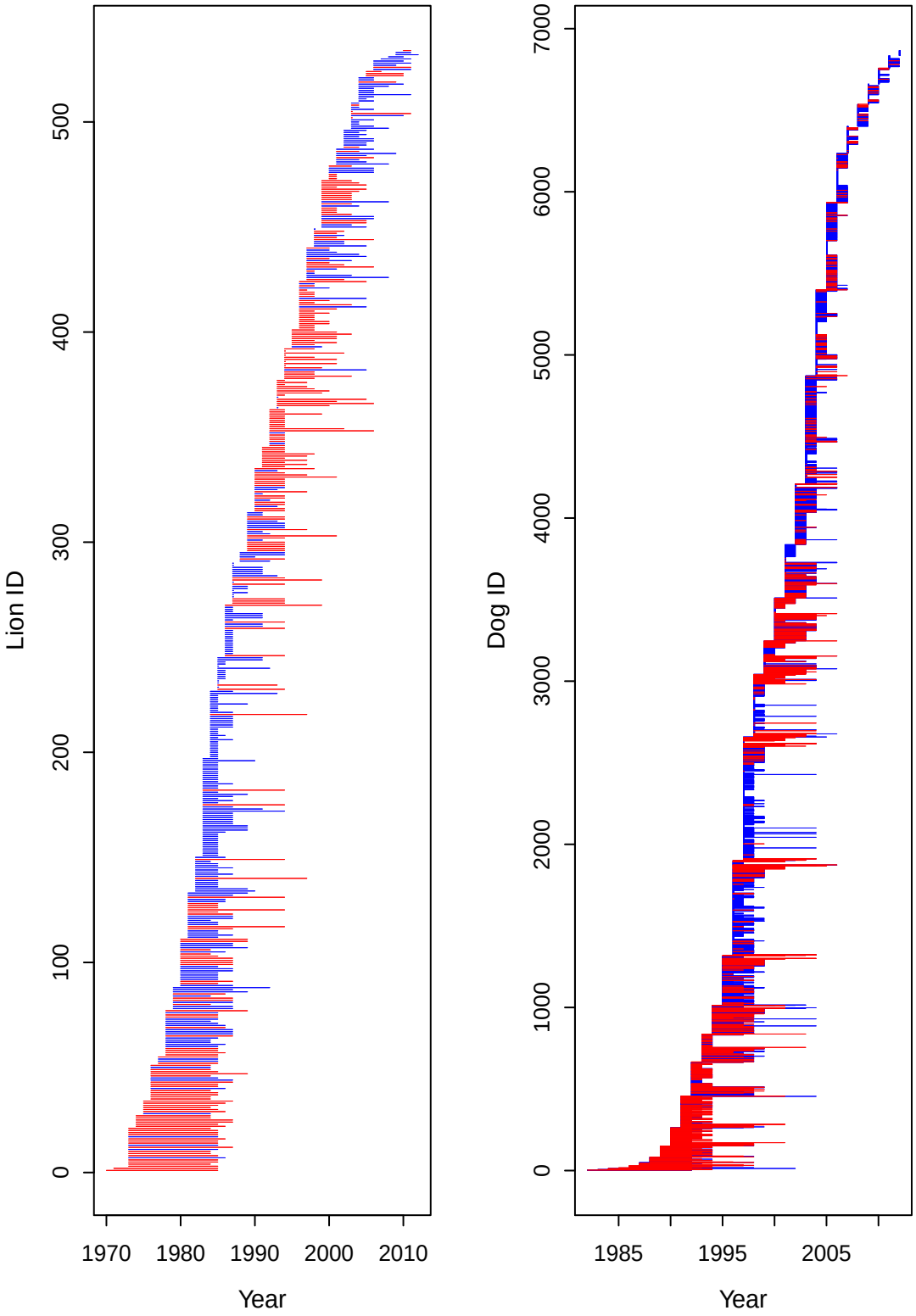

Figure S1: Canine distemper virus serology test result for lions (left) and dogs (right). Each horizontal line corresponds to a seropositve (red) or seronegative (blue) individual and starts in the year of birth and finishes in the year of sampling.

S2. CDV serology model

We used a Bayesian state-space model to estimate the annual force of CDV infection of each species from serology data and vaccination history of each individual. Briefly, the model is composed of two parts: i) a *Biological process* which characterises the mechanism of infection; and ii) an *Observation process* which confronts the processes underlying the generation of the observed data (mostly at the population level) with the individual level data. Ultimately, the state-space model describes the combination of stochastic processes giving rise to the data in Figure S1.

We chose this approach over a more mechanistic model, which typically comprises epidemiological compartmental models (e.g. susceptible-infected-recovered type models), for two main reasons. First, because, while there are examples of the use of serology data to estimate seroprevalence, it is extremely challenging to reconstruct disease dynamics from this type of data given a number of data limitations, such as the unknown timing of exposure and misclassification of seropositivity. Second, fitting mechanistic models to populations for which key information (e.g. number of susceptible and infected individuals, uneven sampling of age-structure, etc.) is unknown would not be more powerful than our chosen approach. Although our modelling approach is mainly phenomenological, it (1) enables integration of multiple types of data, such as serology and vaccination, (2) captures and characterises the main features of the disease dynamics (e.g. periodic peaks of infection, cross-species transmission) and (3) distinguishes between those features acting at the population and/or individual level, while estimating the timing of infection from serological assays that are never 100% accurate.

Biological process

Ultimately we are interested in estimating, $h_s(t)$, the proportion of infected individuals of species s at year t, where t ranges from 1 to 43 and corresponds to a time series from 1984 to 2012. Here, this proportion is defined through a logit transformation such that:

$$h_s(t) = \frac{exp(H_s(t))}{1 + exp(H_s(t))} \tag{1}$$

where $H_s(t)$ is the predictor of $h_s(t)$, defined as a stochastic realisation from a Gaussian

process:

$$H_s(t) \sim N(\bar{H}_s(t), \sigma_{s,t}) \tag{2}$$

The variability implied by $\sigma_{s,t}$ is the annual variation around the expectation $\bar{H}_s(t)$. The prior distribution for $\sigma_{s,t}$ is defined in Table S2. $\bar{H}_s(t)$ is formulated as a linear function of covariates that describe the CDV transmission process in lions and domestic dogs. In their most parameterised forms, the expected predictors for dogs and lions were:

$$\bar{H}_{lions,t} = \beta_0 + \beta_1 t + \beta_2 H_{lions,t-1} + \beta_3 H_{lions,t-2} + \beta_4 H_{dogs,t-1} \tag{3}$$

$$\bar{H}_{dogs,t} = \omega_0 + \omega_1 t + \omega_2 H_{dogs,t-1} + \omega_3 H_{dogs,t-2} + \omega_4 H_{lions,t-1} - \omega_5 C_{t-1} - \omega_6 C_{t-2} \tag{4}$$

The parameters β_0 and ω_0 correspond to the intercept, while β_1 and ω_1 correspond to the coefficients of a linear trend on time, for lions and dogs respectively. β_2 and β_3 for lions and ω_2 and ω_3 for dogs, correspond to the coefficients of an autoregressive component (AR) with lag 1 and 2, respectively. The AR terms were used to emulate the disease dynamics. Specifically, the AR defines the probability of infection in a given year as dependent on the previous year (lag 1) and the year before that (lag 2). With the addition of stochasticity, particular combinations of parameter values for the AR(2) process can generate persistent cycles in the infection dynamics. The epidemiological process behind CDV dynamics is thought to last 2-6 weeks, however, given the substantial logistical and financial constraints involved in obtaining serum samples at very close intervals (particularly from lions), the serology dataset used in this study only allowed us to explore exposure patterns at longer time intervals, and does not provide sufficient resolution and sample sizes to efficiently inform a model specified to a time-scale smaller than a year. Further, given that the questions we ask are at an annual scale, the magnitude of these AR components are thought to be appropriate. The parameters β_4 and ω_4 correspond to the cross-species transmission coefficients, and are defined as an autoregression of lag 1 on the probability of infection of the other species (i.e. β_4 corresponds to the transmission from dogs to lions and $\omega 4$ to the transmission from lions to dogs). The difference between the lion and dog linear predictors is that the dogs might be directly affected by vaccination. The impact of regional vaccination coverage (C_t) on the domestic dog annual CDV seroprevalence was then included as a lagged covariate with coefficients ω_5 and ω_6. The effect of village-level vaccination on dog seroprevalence (rather than regional-level

vaccination coverage) was investigated at a later stage when estimating the probability of an individual becoming infected (see equation 8). The priors for all these coefficients are defined in Table S2. When needed we constrained the priors to be biologically plausible. For example, the priors for the transmission and vaccination parameters were described as strictly positive since there is no cross-species immunity and vaccination cannot increase infectivity. Owing to the AR lags, priors drawn from a normal distribution with mean β_0 or ω_0 for lions and dogs respectively, and variance 10^3 were allocated to the first two time points, $\bar{H}_{t=1,2}$.

We note that the effect of domestic dog vaccination on lion CDV dynamics was not directly included in the description of $\bar{H}_{lions,t}$ since the cross-species transmission term of dog to lions (β_4) already takes this effect into account. This term uses the dog infection hazard, which is estimated taking into account the vaccination history of the dog (see below) and the overall vaccination coverage of the region. Hence, if directly included in the lion predictor, vaccination would be accounted for twice. To investigate the impact of dog vaccination in lions, we developed a prediction model (see subsection *Prediction model*).

At the individual level, the probability of an individual i of species s becoming infected at time t, denoted $u_{i,s}(t)$, is defined as:

$$u_{i,s}(t) = \frac{exp(U_{i,s}(t)}{1 + exp(U_{i,s}(t))} \tag{5}$$

where $U_{i,s}(t)$ is the predictor of $u_{i,s}(t)$ and is modelled as a Gaussian term such that:

$$U_{i,s}(t) \sim N(\bar{U}_{i,s}(t), \psi_{i,s,t}) \tag{6}$$

The stochasticity generated by ψ is the expression of individual variation which increases with the duration of the time interval between the years of birth and sampling; i.e. the uncertainty in the time of exposure of an individual with a greater time interval between birth and sampling will be higher than one with a short interval. The prior distribution for $\psi_{i,s,t}$ is defined in Table S2. $\bar{U}_{i,s}(t)$ is defined for each species as:

$$\bar{U}_{i,lions}(t) = H_{i,lions}(t) \tag{7}$$

$$\bar{U}_{i,dogs}(t) = H_{i,dogs}(t) + v_1 H_{i,dogs,t-1} V_{i,t-1} + v_2 H_{i,dogs,t-2} V_{i,t-2} \quad (8)$$

For dogs, $\bar{U}_{i,s}(t)$ also takes into account the effect of village-level vaccination (V). The parameters v_1 and v_2 are the coefficients for this covariate which investigates the impact of whether dog i comes from a village that was vaccinated in the previous year or previous two years, respectively. The priors for these parameters are defined in Table S2.

Table S2: Prior distributions for the parameters used to model the lion and domestic dog populations annual probability of canine distemper virus infection. AR corresponds to the autoregression.

Species	**Variable**	**Parameter**	**Distribution**	**Prior**
Lions	Correct detection	q^+	Beta	$\sim beta(25, 0.5)$
	False detection	q^-	Beta	$\sim beta(0.5, 25)$
	Variance	$\psi \vert \sigma$	Normal	$\sim N(0, \tau^{2^{-1}})$
	Standard deviation	τ	Uniform	$\sim U(0, 5)$
	Intercept	β_0	Normal	$\sim N(0, 0.001)$
	Linear trend	β_1	Normal	$\sim N(0, 0.001)$
	AR(1)	β_2	Normal	$\sim N(0, 0.1)$
	AR(2)	β_3	Normal	$\sim N(0, 0.1)$
	Dog-to-lion transmission	β_4	Exponential	$\sim exp(0.5)$
Dogs	Correct detection	q^+	Beta	$\sim beta(25, 0.5)$
	False detection	q^-	Beta	$\sim beta(0.5, 25)$
	Variance	$\psi \vert \sigma$	Normal	$\sim N(0, \tau^{2^{-1}})$
	Standard deviation	τ	Uniform	$\sim U(0, 5)$
	Village status (lag 1)	v_1	Exponential	$\sim exp(0.5)$
	Village status (lag 2)	v_2	Exponential	$\sim exp(0.5)$
	Intercept	ω_0	Normal	$\sim N(0, 0.001)$
	Linear trend	ω_1	Normal	$\sim N(0, 0.001)$
	AR(1)	ω_2	Normal	$\sim N(0, 0.1)$
	AR(2)	ω_3	Normal	$\sim N(0, 0.1)$
	Lion-to-dog transmission	ω_4	Exponential	$\sim exp(0.5)$
	Regional vacc. (lag 1)	ω_5	Exponential	$\sim exp(0.5)$
	Regional vacc. (lag 2)	ω_6	Exponential	$\sim exp(0.5)$

*Normal distributions are expressed in terms of mean and precision.

Observation process

Assuming that the i^{th} individual of species s was born in year t_i and was sampled in year T_i, the probability $r_{i,s}(T_i)$ that, at sampling, it was in fact seropositive is:

$$r_{i,s}(T_i) = 1 - \prod_{t=t_i}^{T_i}(1 - u_{i,s}(t)) \tag{9}$$

where, $u_{i,s}(t)$ is the probability of individual i, of species s, becoming infected at time t as described above in equation (5). This probability links the observation process at the individual level with the biological process at the population level. We estimate the likelihood of being infected between birth and sampling years because it is impossible to identify the exact time of exposure from a serology test. Once an individual is infected and recovers from CDV it gains life-long immunity, hence, after infection, the individual will always test positive in the serology assay. This means that the time-series investigated starts in the year when the first individual from both species was born, i.e. 1970; and ends in the last year for which data are available, i.e. 2012. As a consequence, larger uncertainty should be expected in the initial years of the time-series when no samples, hence no serological data, were available, but the first lions were already born.

In addition to the inherent difficulty in detecting antibodies, test results from serological assays are typically sensitive to cut-off thresholds (16). This means that the true disease status of an individual does not always correspond to the test result. In order to account for this potential misclassification of the animal disease status, in Table S3 we introduce probabilities of Type I and Type II errors.

Table S3: Probabilities (q) associated with canine distemper virus serological misclassification.

		True state	
		+	-
Test	+	q^+	$1-q^-$
result	-	$1-q^+$	q^-

The likelihood that an individual i is detected as seropositive ($P(X_i = 1)$) or seronegative ($P(X_i = 0)$) is based on serology data X and was defined in our model as:

$$P(X_i = 1) = r_{i,s}(T_i)q^+ + (1 - r_{i,s})(1 - q^-) \tag{10}$$

$$P(X_i = 0) = r_{i,s}(T_i)(1 - q^+) + (1 - r_{i,s})q^- \tag{11}$$

Based on serological literature, we expect high correct detection (q) and low false detection ($1 - q$). See Table S2 for further details on these priors.

The total likelihood of the data X under the model and parameters was:

$$P(X_{i,s}) = \prod_{i=1}^{n} x_{i,s}P(X_{i,s} = 1) + (1 - x_{i,s})P(X_{i,s} = 0) \tag{12}$$

where n corresponds to the number of samples taken from each species and $x_{i,s}$ corresponds to individual draws from the data X. The likelihood of the data X from individual i and species s was generated from a Bernoulli distribution with success probability P, i.e. probability of getting a seropositive individual upon testing, such that:

$$X_{i,s} \sim Bernoulli(P(X_{i,s} = 1)) \tag{13}$$

Where $X_s = 1$ corresponds to a CDV positive titer and $X_s = 0$ to a CDV negative titer. If both realisations are equally likely, $P(X_s = 1) = P(X_s = 0) = 0.5$.

Prior sensitivity

We explored the sensitivity of the model results to the prior distributions by constraining and widening the allocated distribution range. The posterior distributions for most parameters and the annual proportion of infected individuals $h_s(t)$ remained similar, suggesting that the parameter estimates are not sensitive to the priors chosen (Table S2). Wider priors for q^+ and q^- generated convergence issues, but (1) given that these were highly constrained to account for the knowledge that there is a low probability of false detection and high probability of correct detection; and (2) given that the overall $h_s(t)$ pattern remained largely unchanged; we find our results robust. The posterior distributions for the cross-species transmission terms were sensitive to wide priors (e.g. $\sim exp(10)$) but these are not thought to be plausible values for these parameters. These posterior

distributions were not sensitive to the small changes in the priors (e.g. mean 0.2 to 2).

Model selection and model fit

The model from which we draw inferences is the one that addresses our full set of scientific questions, but also converges well, generates validated fits, and is not identified by the Deviance Information Criterion (DIC) as obviously overparameterised. The model that is biologically plausible and addresses our full set of questions (i.e. model described above and shown as model A.1 in Table S4) is our preferred model [17]. However, in order to ensure this model performs well, we further evaluate i) numerical robustness, in the form of convergence and mixing criteria (e.g. visual inspection of the chains, and Gelman-Rubin statistics [18,19] shown in Table S5), ii) goodness-of-fit, by, for example, validating the posteriors against the priors, iii) parsimony, in the form of the DIC [see below, 20], and iv) parameter posteriors, allowing coefficients of non-significant terms to shrink to zero (see section *Sensitivity analysis* and associated results).

To evaluate the parsimony, we compared the DIC of several variations of the linear predictor $\bar{H}_s(t)$ presented in Eqs. 3 and 4 (Table S4). Model A.1 in Table S4, corresponds to the fully parameterised model and is described as explained in Eqs. 3 and 4 for lions and dogs, respectively. We compare small changes to this model (A.1 - A.7), such as excluding lags for the village-level vaccination (e.g. model A.2), one-way cross-species transmission (model A.3) or lags in the vaccination coverage effect (model A.7). However, we also compare substantial changes (models A-K), such as removing cross-species transmission (models B), or investigating alternative disease dynamics, e.g. by replacing the autoregressive components with sinusoidal dynamics (models J-K). Table S4 shows all formulations investigated for the predictor $\bar{H}_s(t)$ and whether it included the effect of village-level vaccination on domestic dogs as in Eq. 8. In Table S4, we use 'Yes' and 'No' to denote presence or absence of village-level vaccination, and 'Lag 1' or 'Lag 2' to denote the presence of that specific lag only.

The deviance is a measure of overall fit, whilst the penalty measures the complexity of the model by identifying the effective number of parameters in the model (20,21). A parsi-

monious model is expected to have relatively few parameters, thus a lower DIC indicates a better model. As such, our biologically preferred model is also the most parsimonious as it was separated from the second best model by 12 DIC values. However, although the posterior distributions for parameters with unconstrained priors are approximately normally distributed (e.g. β_1 and ω_1), for parameters whose priors were constrained to accommodate biological realism (e.g. β_4, ω_4), the posterior distributions tended to be unimodal, but skewed (Figure S2), violating normality assumptions required for estimating DIC. As such, given that the model suggested by the DIC is the one containing all of the candidate covariates and interactions, it is possible that it suffers from a degree of overparameterisation. Nonetheless, even if model terms are not structurally dropped from the classic data-dredging approach, their importance may still be allowed to shrink parametrically via our sensitivity analysis that quantifies the effect size of each model term on the basis of prediction models (see section *Sensitivity analysis*). As such, considering this multifaceted approach to model selection (i.e. biologically plausible and scientifically interesting, and criteria i-iv), our choice of preferred model is deemed robust and reliable.

All models were fitted using JAGS software (22) which uses Gibbs sampling to generate posterior distributions of the parameters given the likelihood, prior distributions and the data itself. We ran our models for $50 \cdot 10^4$ iterations with burn-in of $30 \cdot 10^4$ to achieve convergence. Since the estimation of the number of effective parameters used to estimate the DIC is typically slower to converge than the model parameters, we recompiled each model for further $20 \cdot 10^4$ iterations before estimating the DIC using 2000 iterations.

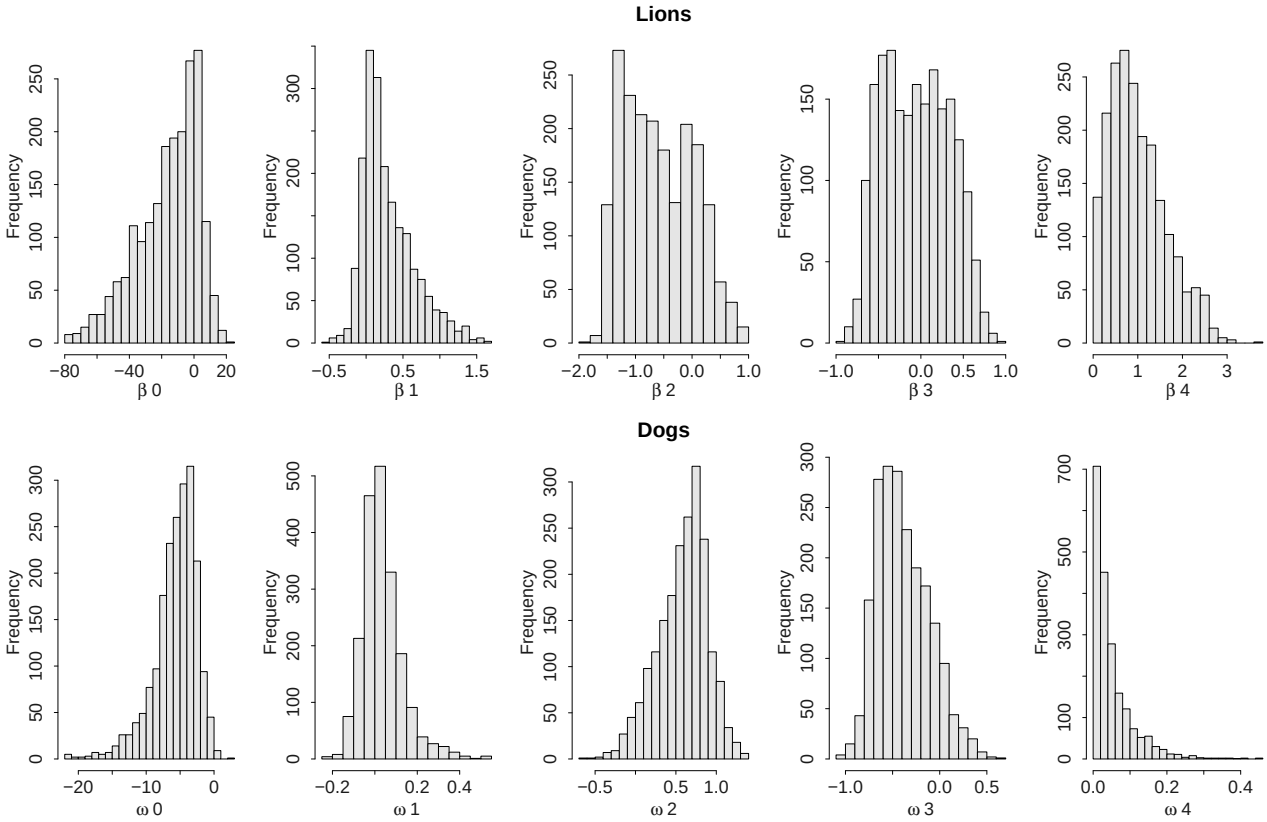

Figure S2: Histograms of the posterior distributions of the best model, i.e. model A.1.

Table S4: Model selection. Description of the linear predictors $\bar{H}_s(t)$ of all models fitted with and without village-level vaccination (V), and resultant delta-Deviance Information Criterion (ΔDIC). ΔDIC in red corresponds to the best model and bold values correspond to the subsequent two best models. See description of parameters in Table S2.

Model	**V**	**Linear predictor**	**ΔDIC**
A.1	Yes	$\beta_0 + \beta_1 t + \beta_2 H_{lions,t-1} + \beta_3 H_{lions,t-2} + \beta_4 H_{dogs,t-1}$ $\omega_0 + \omega_1 t + \omega_2 H_{dogs,t-1} + \omega_3 H_{dogs,t-2} + \omega_4 H_{lions,t-1} - \omega_5 C_{t-1} - \omega_6 C_{t-2}$	**0**
A.2	No	$\beta_0 + \beta_1 t + \beta_2 H_{lions,t-1} + \beta_3 H_{lions,t-2} + \beta_4 H_{dogs,t-1}$ $\omega_0 + \omega_1 t + \omega_2 H_{dogs,t-1} + \omega_3 H_{dogs,t-2} + \omega_4 H_{lions,t-1} - \omega_5 C_{t-1}$	1436
A.3	Yes	$\beta_0 + \beta_1 t + \beta_2 H_{lions,t-1} + \beta_3 H_{lions,t-2} + \beta_4 H_{dogs,t-1}$ $\omega_0 + \omega_1 t + \omega_2 H_{dogs,t-1} + \omega_3 H_{dogs,t-2} - \omega_5 C_{t-1}$	583
A.4	Lag 1	$\beta_0 + \beta_1 t + \beta_2 H_{lions,t-1} + \beta_3 H_{lions,t-2} + \beta_4 H_{dogs,t-1}$ $\omega_0 + \omega_1 t + \omega_2 H_{dogs,t-1} + \omega_3 H_{dogs,t-2} + \omega_4 H_{lions,t-1} - \omega_5 C_{t-1}$	1256
A.5	Lag 2	$\beta_0 + \beta_1 t + \beta_2 H_{lions,t-1} + \beta_3 H_{lions,t-2} + \beta_4 H_{dogs,t-1}$ $\omega_0 + \omega_1 t + \omega_2 H_{dogs,t-1} + \omega_3 H_{dogs,t-2} + \omega_4 H_{lions,t-1} - \omega_5 C_{t-1}$	1027
A.6	Lag 1	$\beta_0 + \beta_1 t + \beta_2 H_{lions,t-1} + \beta_3 H_{lions,t-2} + \beta_4 H_{dogs,t-1}$ $\omega_0 + \omega_1 t + \omega_2 H_{dogs,t-1} + \omega_3 H_{dogs,t-2} - \omega_5 C_{t-1}$	**47**
A.7	Yes	$\beta_0 + \beta_1 t + \beta_2 H_{lions,t-1} + \beta_3 H_{lions,t-2} + \beta_4 H_{dogs,t-1}$ $\omega_0 + \omega_1 t + \omega_2 H_{dogs,t-1} + \omega_3 H_{dogs,t-2} + \omega_4 H_{lions,t-1} - \omega_5 C_{t-1}$	1454
A.8	No	$\beta_0 + \beta_1 t + \beta_2 H_{lions,t-1} + \beta_3 H_{lions,t-2} + \beta_4 H_{dogs,t-1}$ $\omega_0 + \omega_1 t + \omega_2 H_{dogs,t-1} + \omega_3 H_{dogs,t-2} + \omega_4 H_{lions,t-1} - \omega_5 C_{t-1} - \omega_6 C_{t-2}$	226
B.1	Yes	$\beta_0 + \beta_1 t + \beta_2 H_{lions,t-1} + \beta_3 H_{lions,t-2} + \beta_4 H_{dogs,t-1}$ $\omega_0 + \omega_1 t + \omega_2 H_{dogs,t-1} + \omega_3 H_{dogs,t-2} + \omega_4 H_{lions,t-1}$	834
B.2	No	$\beta_0 + \beta_1 t + \beta_2 H_{lions,t-1} + \beta_3 H_{lions,t-2} + \beta_4 H_{dogs,t-1}$ $\omega_0 + \omega_1 t + \omega_2 H_{dogs,t-1} + \omega_3 H_{dogs,t-2} + \omega_4 H_{lions,t-1}$	447
B.3	Yes	$\beta_0 + \beta_1 t + \beta_2 H_{lions,t-1} + \beta_3 H_{lions,t-2} + \beta_4 H_{dogs,t-1}$ $\omega_0 + \omega_1 t + \omega_2 H_{dogs,t-1} + \omega_3 H_{dogs,t-2}$	424
B.4	Lag 1	$\beta_0 + \beta_1 t + \beta_2 H_{lions,t-1} + \beta_3 H_{lions,t-2} + \beta_4 H_{dogs,t-1}$ $\omega_0 + \omega_1 t + \omega_2 H_{dogs,t-1} + \omega_3 H_{dogs,t-2} + \omega_4 H_{lions,t-1}$	556
B.5	Lag 2	$\beta_0 + \beta_1 t + \beta_2 H_{lions,t-1} + \beta_3 H_{lions,t-2} + \beta_4 H_{dogs,t-1}$ $\omega_0 + \omega_1 t + \omega_2 H_{dogs,t-1} + \omega_3 H_{dogs,t-2} + \omega_4 H_{lions,t-1}$	229
B.6	Lag 1	$\beta_0 + \beta_1 t + \beta_2 H_{lions,t-1} + \beta_3 H_{lions,t-2} + \beta_4 H_{dogs,t-1}$ $\omega_0 + \omega_1 t + \omega_2 H_{dogs,t-1} + \omega_3 H_{dogs,t-2}$	**12**

Model	V	Linear predictor	ΔDIC
C.1	Yes	$\beta_0 + \beta_2 H_{lions,t-1} + \beta_3 H_{lions,t-2} + \beta_4 H_{dogs,t-1}$ $\omega_0 + \omega_2 H_{dogs,t-1} + \omega_3 H_{dogs,t-2} + \omega_4 H_{lions,t-1} - \omega_5 C_{t-1}$	129
D.1	Yes	$\beta_0 + \beta_1 t + \beta_2 H_{lions,t-1} + \beta_3 H_{lions,t-2}$ $\omega_0 + \omega_1 t + \omega_2 H_{dogs,t-1} + \omega_3 H_{dogs,t-2} - \omega_5 C_{t-1}$	126
E.1	Yes	$\beta_0 + \beta_1 t + \beta_2 H_{lions,t-1} + \beta_3 H_{lions,t-2}$ $\omega_0 + \omega_1 t + \omega_2 H_{dogs,t-1} + \omega_3 H_{dogs,t-2}$	55
E.2	No	$\beta_0 + \beta_1 t + \beta_2 H_{lions,t-1} + \beta_3 H_{lions,t-2}$ $\omega_0 + \omega_1 t + \omega_2 H_{dogs,t-1} + \omega_3 H_{dogs,t-2}$	692
F.1	Yes	$\beta_0 + \beta_2 H_{lions,t-1} + \beta_3 H_{lions,t-2} + \beta_4 H_{dogs,t-1}$ $\omega_0 + \omega_2 H_{dogs,t-1} + \omega_3 H_{dogs,t-2} + \omega_4 H_{lions,t-1}$	753
F.2	No	$\beta_0 + \beta_2 H_{lions,t-1} + \beta_3 H_{lions,t-2} + \beta_4 H_{dogs,t-1}$ $\omega_0 + \omega_2 H_{dogs,t-1} + \omega_3 H_{dogs,t-2} + \omega_4 H_{lions,t-1}$	84
G.1	Yes	$\beta_0 + \beta_2 H_{lions,t-1} + \beta_3 H_{lions,t-2}$ $\omega_0 + \omega_2 H_{dogs,t-1} + \omega_3 H_{dogs,t-2} - \omega_5 C_{t-1}$	842
H.1	Yes	$\beta_0 + \beta_2 H_{lions,t-1} + \beta_3 H_{lions,t-2}$ $\omega_0 + \omega_2 H_{dogs,t-1} + \omega_3 H_{dogs,t-2}$	833
I.1	Yes	$\beta_0 + \beta_2 H_{lions,t-1} + \beta_4 H_{dogs,t-1}$ $\omega_0 + \omega_2 H_{dogs,t-1} + \omega_4 H_{lions,t-1}$	169
J.1	Yes	$\beta_0 + \beta_1 t + \beta_2 cos(\frac{2\pi t}{F}) + \beta_3 sin(\frac{2\pi t}{F}) + \beta_4 H_{dogs,t-1}$ * $\omega_0 + \omega_1 t + \omega_2 cos(\frac{2\pi t}{F}) + \omega_3 sin(\frac{2\pi t}{F}) + \omega_4 H_{lions,t-1} - \omega_5 C_{t-1}$	122
J.2	Yes	$\beta_0 + \beta_1 t + \beta_2 cos(\frac{2\pi t}{F}) + \beta_3 sin(\frac{2\pi t}{F}) + \beta_4 H_{dogs,t-1}$ * $\omega_0 + \omega_1 t + \omega_2 cos(\frac{2\pi t}{F}) + \omega_3 sin(\frac{2\pi t}{F}) + \omega_4 H_{lions,t-1}$	102
J.3	No	$\beta_0 + \beta_1 t + \beta_2 cos(\frac{2\pi t}{F}) + \beta_3 sin(\frac{2\pi t}{F}) + \beta_4 H_{dogs,t-1}$ * $\omega_0 + \omega_1 t + \omega_2 cos(\frac{2\pi t}{F}) + \omega_3 sin(\frac{2\pi t}{F}) + \omega_4 H_{lions,t-1}$	488
K.1	Yes	$\beta_0 + \beta_1 t + \sum_{n=1}^{N} (\beta_{2_n} cos(\frac{2\pi t}{F_n}) + \beta_{3_n} sin(\frac{2\pi t}{F_n})) + \beta_4 H_{dogs,t-1}$ * $\omega_0 + \omega_1 t + \sum_{n=1}^{N} (\omega_{2_n} cos(\frac{2\pi t}{F_n}) + \omega_{3_n} sin(\frac{2\pi t}{F_n})) + \omega_4 H_{lions,t-1} - \omega_5 C_{t-1}$	1369
K.2	Yes	$\beta_0 + \beta_1 t + \sum_{n=1}^{N} (\beta_{2_n} cos(\frac{2\pi t}{F_n}) + \beta_{3_n} sin(\frac{2\pi t}{F_n})) + \beta_4 H_{dogs,t-1}$ * $\omega_0 + \omega_1 t + \sum_{n=1}^{N} (\omega_{2_n} cos(\frac{2\pi t}{F_n}) + \omega_{3_n} sin(\frac{2\pi t}{F_n})) + \omega_4 H_{lions,t-1}$	115
K.3	No	$\beta_0 + \beta_1 t + \sum_{n=1}^{N} (\beta_{2_n} cos(\frac{2\pi t}{F_n}) + \beta_{3_n} sin(\frac{2\pi t}{F_n})) + \beta_4 H_{dogs,t-1}$ * $\omega_0 + \omega_1 t + \sum_{n=1}^{N} (\omega_{2_n} cos(\frac{2\pi t}{F_n}) + \omega_{3_n} sin(\frac{2\pi t}{F_n})) + \omega_4 H_{lions,t-1}$	181

*N corresponds to the number of trigonometric curves, here N=2, and F corresponds to the sinusoidal frequency which was drawn from a uniform distribution between 1 and 42, the full length of the time-series.

Sensitivity analysis

We used a forward prediction approach to investigate the effect of village- and regional-level vaccination on the annual proportion of infected domestic dogs and of the presence of cross-species transmission from lions-to-dogs and dogs-to-lions on their respective annual proportion of infected individuals ($h_s(t)$). All predictions were then made using Eqs. 1-4 and generated from the parameters estimated in each iteration of the best model (model A.7), i.e. a new prediction is generated in each iteration from which we can calculate the mean prediction values and associated credible intervals.

First, to ascertain the predictive power of the model, we compared the estimates from equation 4 (i.e. infection hazard of dogs at time t) with its prediction estimates (Figure S3). Figure S3 shows that in the first ∼10 years, the median and credible intervals of the estimate (grey in Figure S3) and prediction (red in Figure S3) are very similar. This suggests that the model's predictive power is of approximately 10 years.

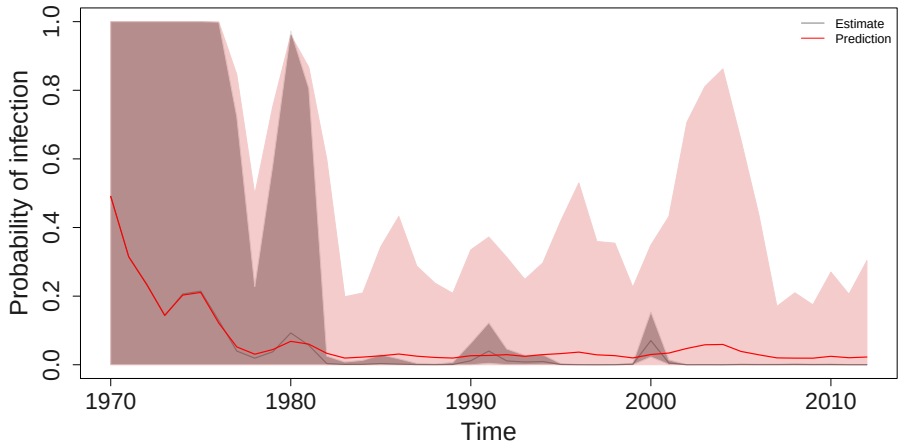

Figure S3: Comparison between the mean and 95% credible intervals of the estimated (grey) and predicted dog probability of infection (red) from the best model.

To investigate the effect of village-level vaccination, we compared predictions from models A.7 and A.8 (A.8 is similar to A.7 but without village-level vaccination). The mean and credible intervals of these models were very similar (see Figure 5 in main text), indicating that there is little or no impact of village-level vaccination on the overall disease dynamics. The similarity between these two prediction models also show that one can be used as a proxy for the other. Following this result, we used the predictions based on model

A.8 to investigate the impact of region-level vaccination. Using model A.7 as the base model for these predictions would bias the results as the effect of village-level vaccination is carried through to the other parameters of the model, and it would be difficult to distinguish their effect. A prediction was then generated based on model A.8 and by setting the region-level vaccination parameter to zero. Finally, to investigate the effect of cross-species transmission, we generated another prediction model by setting both region-level vaccination and lion-to-dog, and dog-to-lion transmission parameters to zero.

S3. JAGS code

```
model{
  for(t in 1:Ntime){

    # Hazard predictor for lions
    H.lions[t] ~ dnorm(Hbar.lions[t], prec.lions)
    Hbar.lions[t]<- beta0 + beta1*t + beta2*H.lions[t-1] + beta3*H.lions[t-2] + beta4*H.dogs[t-1]

    # Hazard predictor for dogs
    H.dogs[t] ~ dnorm(Hbar.dogs[t], prec.dogs)
    Hbar.dogs[t]<- omega0 + omega1*t + omega2*H.dogs[t-1] + omega3*H.dogs[t-2] +
                   omega4*H.lions[t-1] - omega5*Coverage[t-1] - omega6*Coverage[t-2] }

  #Hazards for t=1 & t=2 (AR time lags)
  H.lions[1] ~ dnorm(beta0, 0.001)
  H.lions[2] ~ dnorm(beta0, 0.001)
  H.dogs[1] ~ dnorm(omega0, 0.001)
  H.dogs[2] ~ dnorm(omega0, 0.001)

  #Lion probability of infection
  for(i in 1:Nlions){
    for(t in 1:Ntime){
      U.lions[i,t]<- H.lions[t] + phi.lions[i,t]
      phi.lions[i,t] ~ dnorm(0, tau.phi.lions)
      logit(u.lions[i,t])<- U.lions[i,t]
      P.lions[i,t]<- 1-u.lions[i,t] }

    # probability of infection between birth and sample year
    r.lions[i]<- 1-prod(P.lions[i,Birth.lions[i]:Sample.lions[i]])
    # probability of getting a seropositive result upon testing
    p.lions[i]<- qpos.lions*r.lions[i] + qneg.lions*(1-r.lions[i])

    # Likelihood of data (titre binary results)
    X.lions[i] ~ dbern(p.lions[i]) }

  #Dog probability of infection given vaccinated status of its village
  for(i in 1:Ndogs){
    for(t in 3:Ntime){
      U.dogs[i,t]<- H.dogs[t] - v1*H.dogs[t-1]*Vvacc[(i-1)*Ntime+(t-1)] - v2*H.dogs[t-2]*Vvacc[(i-1)*
                    Ntime+(t-2)] + phi.dogs[i,t]
      phi.dogs[i,t] ~ dnorm(0, tau.phi.dogs)
      logit(u.dogs[i,t])<- U.dogs[i,t]
      P.dogs[i,t]<- 1-u.dogs[i,t] }

    # Probability of infection at t=1 & t=2 (AR time lags)
    U.dogs[i,1] <- H.dogs[1]
    logit(u.dogs[i,1])<- U.dogs[i,1]
    P.dogs[i,1]<- 1-u.dogs[i,1]
    U.dogs[i,2] <- H.dogs[2]
    logit(u.dogs[i,2])<- U.dogs[i,2]
    P.dogs[i,2]<- 1-u.dogs[i,2]

    # probability of infection between birth and sample year
    r.dogs[i]<- 1-prod(P.dogs[i,Birth.dogs[i]:Sample.dogs[i]])
    # probability of getting a seropositive result upon testing
    p.dogs[i]<- qpos.dogs*r.dogs[i] + qneg.dogs*(1-r.dogs[i])

    # Likelihood of data (titre binary results)
    X.dogs[i] ~ dbern(p.dogs[i]) }
```

```
  #Priors

    prec.lions<- 1/(sigma.lions*sigma.lions)
    sigma.lions ~ dunif(0,5)
    prec.dogs<- 1/(sigma.dogs*sigma.dogs)
    sigma.dogs ~ dunif(0,5)

    tau.phi.lions<- 1/(sigma.phi.lions*sigma.phi.lions)
    sigma.phi.lions ~ dunif(0,5)
    tau.phi.dogs<- 1/(sigma.phi.dogs*sigma.phi.dogs)
    sigma.phi.dogs ~ dunif(0,5)

    qneg.lions ~ dbeta(0.5,25)
    qneg.dogs ~ dbeta(0.5,25)
    qpos.lions ~ dbeta(25,0.5)
    qpos.dogs ~ dbeta(25,0.5)

    lambda<- 1/2

    beta0 ~ dnorm(0,0.001)
    beta1 ~ dnorm(0,0.001)
    beta2 ~ dnorm(0,0.1)
    beta3 ~ dnorm(0,0.1)
    beta4 ~ dexp(lambda)

    omega0 ~ dnorm(0,0.001)
    omega1 ~ dnorm(0,0.001)
    omega2 ~ dnorm(0,0.1)
    omega3 ~ dnorm(0,0.1)
    omega4 ~ dexp(lambda)
    omega5 ~ dexp(lambda)
    omega6 ~ dexp(lambda)
    v1 ~ dexp(lambda)
    v2 ~ dexp(lambda)

} #end model
```

$Ntime$ (total number of years in time-series), $Ndogs$ and $Nlions$ (total of number of dogs and lions sampled respectively), $Coverage$ (regional-level vaccination coverage; i.e. proportion of vaccinated villages per year), $Vvacc$ (binary variable indicating whether the dog i came from a vaccinated village), $Birth.$ (year of birth of each individual i, $Sample.$ (year when the serum sample of individual i was collected), and X (titre binary result where 1 indicates positive for CDV infection and 0 indicates negative) correspond to available data.

S4. Results

Table S5 shows the parameter values resulting from the best model (model A.1) and the estimated shrink factor values of the Gelman-Rubin convergence diagnostic. If both the estimated value and the upper credible interval of the shrink factor are close to 1, the chains are considered to have converged. Apart from the precision and dog-to-lion parameters, all other parameters seem to have converged well, as also shown from the visual observation of the trace plots. As a rule of thumb, a model is considered to be appropriate if approximately 80% of the parameter estimates converged well [20]. Given the large amount of samples involved in $H_{s,t}$ (one per time point and species, i.e. 86), we do not provide the Gelman-Rubin statistics for this parameter, however, similar to the parameters in Table S5, $H_{s,t}$ converged well.

Table S5: Median posterior estimates with associated 95% credible intervals and Gelman-Rubin shrink factor (estimated value [upper credible interval]) of the best model (A.7).

Species	**Variable**	**Param.**	**Median (95% CI)**	**Shrink factor**
Lions	Intercept	β_0	-12.127 (-61.32, 10.61)	1.33 [2.45]
	Linear trend	β_1	0.201 (-0.17, 1.19)	1.22 [1.81]
	AR(1)	β_2	-0.666 (-1.47, 0.61)	1.02 [1.04]
	AR(2)	β_3	-0.057 (-0.673, .642)	1.01 [1.01]
	Dog-to-lion transmission	β_4	0.883 (0.08, 2.48)	1.40 [2.27]
	Precision	σ	0.070 (0.04, 0.40)	1.43 [3.63]
	Correct detection	q^+	0.939 (0.86, 1.00)	1.00 [1.00]
	False detection	q^-	$2.264{\cdot}10^{-3}$ ($4.60{\cdot}10^{-6}$, $2.49{\cdot}10^{-2}$)	1.04 [1.12]
Dogs	Intercept	ω_0	-5.072 (-13.85, -0.95)	1.02 [1.08]
	Linear trend	ω_1	0.020 (-0.12, 0.28)	1.00 [1.00]
	AR(1)	ω_2	0.629 (-0.09, 1.13)	1.01 [1.01]
	AR(2)	ω_3	-0.430 (-0.82, 0.23)	1.01 [1.05]
	Lion-to-dog transmission	ω_4	0.032 (0.00, 0.20)	1.14 [1.49]
	Regional vacc. (lag 1)	ω_5	0.862 (0.01, 22.28)	1.01 [1.02]
	Regional vacc. (lag 2)	ω_6	0.955 (0.01, 0.03)	1.03 [1.05]
	Village status (lag 1)	v_1	0.008 (0.00, 0.03)	1.01 [1.02]
	Village status (lag 2)	v_2	0.008 (0.00, 0.04)	1.00 [1.01]
	Precision	σ	0.235 (0.06, 0.98)	1.12 [1.44]
	Correct detection	q^+	0.991 (0.91, 1.00)	1.01 [1.03]
	False detection	q^-	$3.421{\cdot}10^{-4}$ ($6.11{\cdot}10^{-7}$, $4.03{\cdot}10^{-3}$)	1.00 [1.01]

Annual probability of infection

Figure S4 shows $H_{s,t}$ estimated from the best model, i.e. mean (line) annual probability of infection estimated for lions (top panel) and dogs (bottom panel) in the logit scale, and respective credible intervals 50% (dark colour), 75% (medium colour) and 95% (light colour). This figure allows us to resolve differences at low probabilities that may arise from Figure 3 (i.e. annual probability of infection shown in the main text of this paper), including the power of the mean estimates. Although the uncertainty in the first decade is large, from ∼1980 the estimates are robust with tighter credible intervals, especially when viewed at 75% credibility. This figure shows a higher frequency in peaks of infection in lions than dogs, in particular from 1994 onwards. The peaks that have lower uncertainty before 1994 (∼1981, 1994) are preceded by peaks in the dogs. However, the 2000 infection peak occurred concurrently in both species.

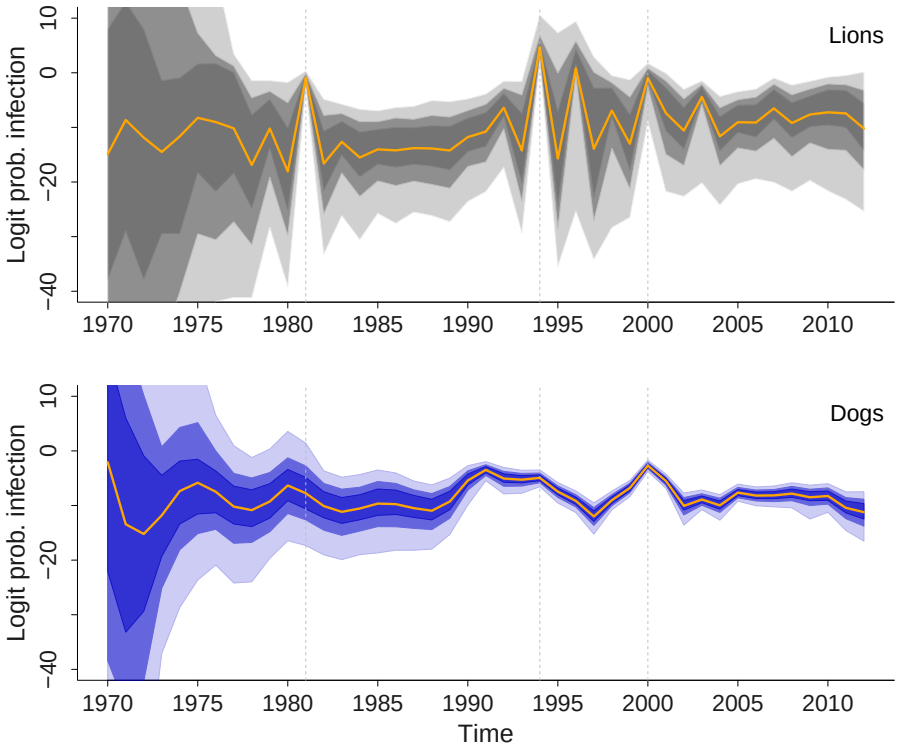

Figure S4: Dog and lions annual probability of infection in the logit scale. Shaded areas around the mean difference (bright line) correspond to 50% (dark), 75% (medium) and 95% (light colour) credible intervals.

In order to compare the probability of infection among species, Figure S5 shows $h_{dogs}(t) - h_{lions}(t)$, i.e. the difference between the probability of infection in dogs with lions estimated

for each draw of the posterior distribution. After the initial first decade of uncertainty, this figure shows an interval of approximately 5 years (∼1976-1981), where the probability of infection in dogs was higher than in lions as seen from the positive values of the difference between the probability of infection in dogs and lions. This is followed by a period of approximately 10 years (∼1982-1991) where the probability of infection in dogs and lions was similar (i.e. the difference is zero), which corresponds to the time where infection is thought to have disappeared form the system (see Figure 3 in main text). However, from mid-1990s there was an increase in the probability of infection in lions compared to dogs, as shown by the negative values in the difference during these times. This also reflects the unstable nature of CDV dynamics in both lions and dogs.

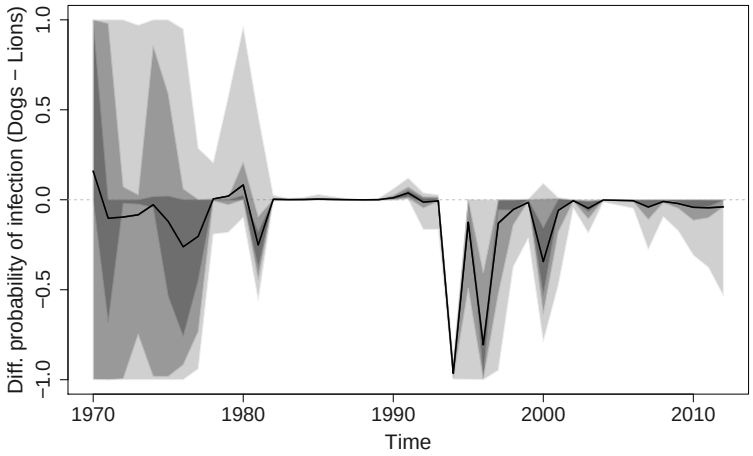

Figure S5: Difference between dog and lion probability of infection. Shaded areas around the mean difference (black line) correspond to 50% (dark grey), 75% (medium grey) and 95% (light grey) credible intervals.

Prediction models

To investigate the impact of cross-species transmission on CDV dynamics, we compared the forecast of the annual probability of infection in lions and dogs, with and without the cross-species transmission parameter (Fig.S6). The similarity of the predictions of the mean probability of infection in dogs and associated 95% credible intervals, with (Fig.S6, green) and without (Fig.S6, blue) lion-to-dog transmission, indicates that transmission from lions into dogs is negligible. The lion prediction model was imprecise (see 95% credible intervals in Fig.S6 right panel), likely due to small sample sizes, and was therefore uninformative. However, it is likely that the uncertainty and/or temporal variation in the disease dynamics are masking the effect of this parameter in the prediction models, as

the effect size of the parameters governing dog-to-lion transmission ($\beta 4$=0.283 [0.08-2.48]) resultant from the best model, was ten times larger than that of the lion-to-dog transmission parameter (Fig.S7), providing evidence that cross-species transmission dynamics are dominated by dog-to-lion transmission .

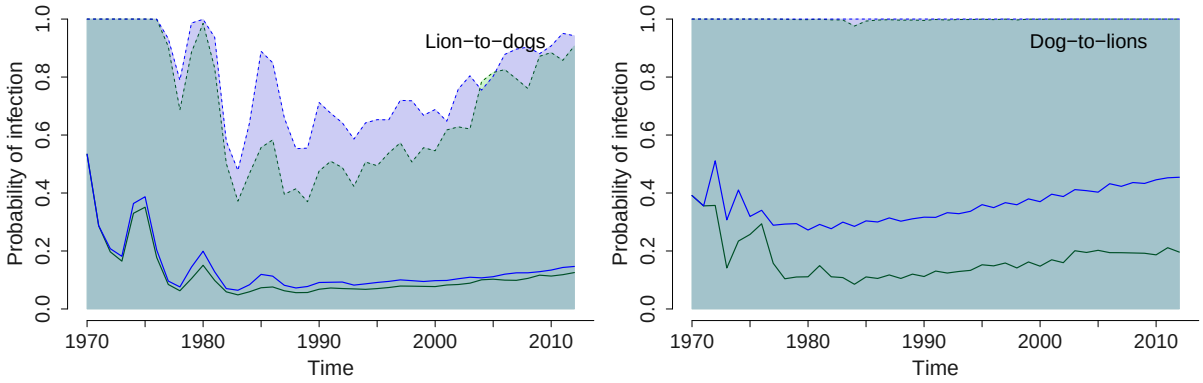

Figure S6: Predictions of the sensitivity model evaluating the effect of cross-species transmission. Left panel) Annual predicted mean probability of dog infection with (green) and without (blue) lion-to-dog transmission; Right panel) Annual predicted mean probability of lion infection with (green) and without (blue) dog-to-lion transmission. Shade corresponds to associated 95% credible interval and dotted line to its upper bound.

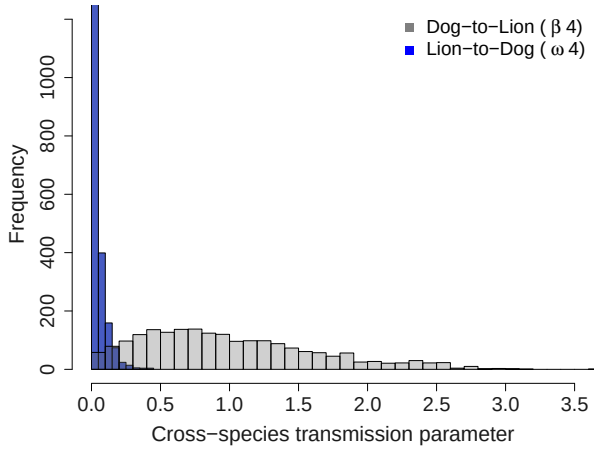

Figure S7: Histogram of the posterior distribution of the cross-species transmission parameters.

In order to investigate the role of dog vaccination on the dynamics of CDV infection in dogs, we compared the forecasts of the dog annual probability of infection with and without vaccination at the village- (Fig.S8, left panel) and regional-levels (Fig.S8, right panel). These figures show that whether a dog originated from a vaccinated or unvaccinated village had a small positive effect on the overall CDV dynamics in domestic dogs

as both the mean and credible intervals of the predicted annual probability of infection, with (Fig.S8, grey) and without (Fig.S8, red) village-level vaccination, were very similar. However, for the period when the regional vaccination coverage was consistently greater than 30%, i.e. after 2003, there was a large marked effect of vaccination on the probability of a dog being infected, as demonstrated by the ∼5% increase in the predicted mean probability of infection from 2003 onwards when this regional-level vaccination effect is set to zero (Fig.S8, green compared to red).

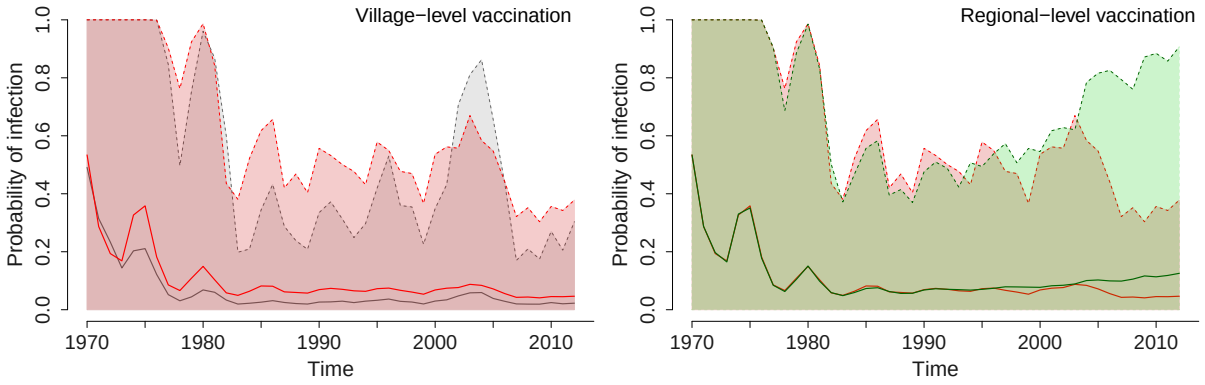

Figure S8: Predictions of the sensitivity evaluating the effect of vaccination. Left panel) Annual predicted mean probability of dog infection with (grey) and without (red) a village-level vaccination parameter; Right panel) Annual predicted mean probability of dog infection with (red) and without (green) a region-level vaccination parameter. Shade corresponds to associated 95% credible interval and dotted line to its upper bound.

Age-seroprevalence

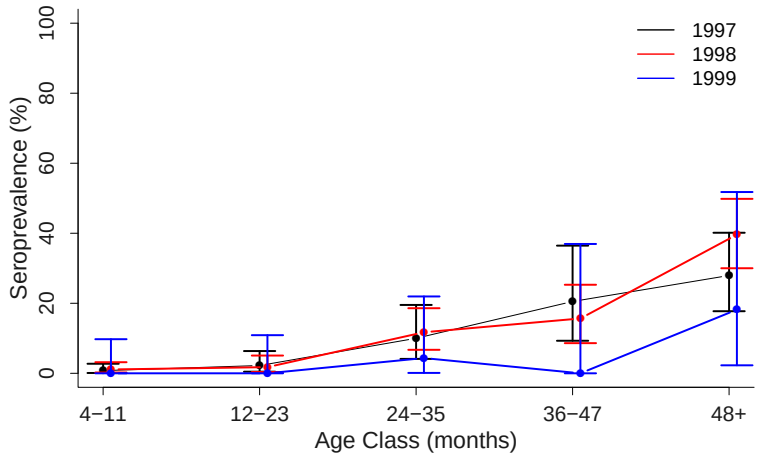

Figure S9: Age seroprevalence of domestic dogs from the districts of Serengeti and Musoma (west of Serengeti National Park) between 1997 and 1999.

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