Supporting Information

Social status mediates the fitness costs of infection with canine distemper virus in Serengeti spotted hyenas

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This document is structured as following:

1. Study background

(a) *The canine distemper virus epidemic in the Serengeti National Park*

In 1993-1994, a CDV epidemic unexpectedly caused the death of an estimated 30% of the lion population in the Serengeti National Park (NP) (Roelke-Parker *et al.* 1996) and increased mortality and morbidity among infected juvenile spotted hyenas (Haas *et al.* 1996). Clinical disease was also observed in a few bat-eared foxes *Otocyon megalotis*, silver-backed jackals *Canis mesomelas* and golden jackals *Canis aureus* in the park (Roelke-Parker *et al.* 1996).

This epidemic was assumed to be caused by the 'spill-over' of a virulent CDV genotype from the domestic dog population outside the Serengeti NP (Roelke-Parker *et al.* 1996), and the spread of the epidemic within the lion population was posited to require repeated introduction of the virus in terms of jackal-or spotted hyena-to-lion transmission (Craft *et al.* 2008; Craft *et al.* 2009).The assumed 'spill-over' of virulent CDV from domestic dogs is inconsistent with genetic evidence demonstrating that lions and spotted hyenas (i.e. non-canid species) during the 1993/1994 epidemic were infected with a distinct CDV strain from those that infected canids (domestic dogs and bat-eared fox) (Nikolin *et al.* 2017). The frequent re-introduction of CDV from hyenas to lions is unlikely because the vast majority of the widely ranging adult population in the Serengeti NP of approximately 7200 hyenas (Hofer & East 1995) showed no symptoms and hence were not shedding virus (Nikolin *et al.* 2017). Cubs < 12 months restricted in range to clan communal dens were the main hyena age category that shed virus.

During the 1993/1994 epidemic all 'Serengeti' strains from lions and spotted hyenas encoded a rare amino acid combination at two sites under positive selection in the region of the CDV-H protein that binds to SLAM (CD 150) host cell receptors, plus one novel substitution in the CDV-V protein, which suppresses host cytokines and affects the outcome of infection. All 'Serengeti' strains from wild canids and a local domestic dog did not encode this rare amino acid combination in the CDV-H protein or the novel substitution in the CDV-V protein. Rather, 'Serengeti' canid strains encoded amino acid combinations in the CDV-H protein typical for strains from domestic dogs and wild carnivores worldwide, and in the CDV-V protein the same amino acid described in all other CDV strains for which genetic information is available (Nikolin *et al.* 2017).

(b) *Ecology of spotted hyenas in the Serengeti National Park*

In the Serengeti NP, hyenas live in fission-fusion societies (clans) that defend a territory. They mainly feed on migratory ungulates, whose seasonal movements cause large

fluctuations in food abundance within spotted hyena clan territories (Hofer & East 1993a; Hofer & East 2003). This presents lactating females with the problem of fulfilling the increased nutritional demands of lactation during periods when prey abundance in the clan territory is not sufficient to feed all clan members (East *et al.* 2015; Hofer *et al.* 2016). Females breed throughout the year, producing litters contain one or two cubs (rare litters of three cubs never survive) (Hofer & East 2008), hence mothers always encounter periods when migratory prey are absent from their territories when lactating.

When migratory ungulates are abundant in a clan's territory, all mothers feed locally and nurse their offspring daily but when absent, they undertake 'commuting' round-trips of approximately 80 to 140 km between the communal den and foraging areas. When their mothers commute, dependent cubs may fast for up to 9 days (Hofer & East 1993b). During periods between these two extremes, a mother's commuting frequency is determined by her social status, with high-ranking females commuting less often to forage and hence nursing their offspring significantly more frequently than low-ranking ones on average (Hofer & East 1993b; Hofer & East 2003; Hofer *et al.* 2016). In the first 6-12 months of life offspring are stationed at the clan's communal den and when approximately 12 months of age they start to accompany their mothers on commuting trips.

2. Supplementary information on the methods

Overview of our methods:

	Step	Procedure	Established methods	Document(s)
Fieldwork	Step 1a	Identifying, aging and sexing individuals in the field	East & Hofer 2001 Hofer & East 2003 Frank, Glickman & Licht 1991 Hofer & East 1993a Hofer & East 2003	Main text: Study design and data collection Appendix: 2.(a)
	Step 1b	Monitoring clinical signs of CDV	Haas et al. 1996	Main text: Study design and data collection Appendix: 2.(a)
	Step 1c	Collecting biological samples (faeces, blood, tissues)	Hofer, East & Campbell 1993	Main text: Study design and data collection Appendix: 2.(a)
Labwork	Step 2a	Serological analyses	Developped in this study	Appendix: 2.(b)
	Step 2b	RT-PCR screening	Hall 1999 Nikolin et al. 2017	Appendix: 2.(b)
Data preparation	Step 3a	Creating encounter histories	Developped in this study	Main text: Definition of demographic, social and infection states:
	Step 3b	Assigning demographic states	Developped in this study	Main text: Definition of demographic, social and infection states, *Demographic states*
	Step 3c	Assessing standardized social status and assigning social states	East et al. 2003 Hofer & East 2003 Goymann et al. 2001 East et al. 2003 East et al. 2009	Main text: Definition of demographic, social and infection states, *Social states*
	Step 3d	Assigning infection states	Developped in this study	Main text: Definition of demographic, social and infection states, *Infection states* Appendix: 2.(c)
Modelling	Step 4a	Building the model	Pradel, Gimenez & Lebreton 2005 Choquet & Nogue 2011 Gimenez et al. 2012	Main text: Multi-event capture-mark-recapture model
	Step 4b	Goodness of fit	Grosbois et al. 2008	Main text: Multi-event capture-mark-recapture model, *Goodness of fit* Appendix: 2.(c)
	Step 4c	Model selection	Culina et al. 2013 Hannan & Quinn 1979	Main text: Multi-event capture-mark-recapture model, *Model selection* Appendix: 2.(g)

(a) *Data collection*

We recorded the identity of all hyenas within a radius of about 100 m of communal dens for approximately three hours at dawn and dusk (Hofer & East 2003). Cubs may be born at the communal den or at private birth dens, in which case they are usually brought to the communal den when about 7-14 days of age (East, Hofer & Turk 1989). We excluded observations of individuals encountered in locations other than at or close to communal dens and birth dens to avoid heterogeneity in the sampling frame. Animals were well habituated to the presence of observers in vehicles, which enabled detailed observations of social interactions and clinical signs indicative of CDV, even in very young individuals.

As part of our health monitoring of study clans, we recorded the start and end of clinical signs consistent with (but not exclusive to) clinical signs of CDV infection (blue sheen on eyes, respiratory distress, ataxia, poor limb coordination, bloody faeces and listlessness) and of secondary infections know to typically occur in spotted hyenas infected with CDV (nasal and or ocular discharge) (Haas *et al.* 1996). Some spotted hyenas suspected to have contracted CDV showed several signs. Individuals with such signs were particularly closely monitored to record their potential date of death or disappearance.

We screened for the presence of CDV by RT-PCR, in faecal (n = 61) and saliva samples (n = 2) collected non-invasively, and blood (n = 27), or tissue (n = 16) samples collected from known hyenas that died from natural causes, were hit by vehicles on roads or were immobilized mostly for the removal of wire snares set by bushmeat hunters (Hofer, East & Campbell 1993).

(b) *Laboratory analyses*

Genetic analysis for assessing relatedness: Microsatellite DNA profiling was used to confirm the identity of the mother and sire of cubs born in study clans using the methods fully detailed elsewhere (East *et al.* 2003; Wilhelm *et al.* 2003; East *et al.* 2009).

RT-PCR screening of samples for the presence of CDV RNA: Following collection, faecal samples were thoroughly mixed and divided into aliquots. Faecal, tissue and blood samples prior to 2004 were frozen in liquid nitrogen and then stored and transported at -80 °C. Samples collected after 2004 were preserved in RNAlater (Sigma–Aldrich Co, St. Louis, MO, USA) following the manufacturer's instructions, stored and transported at -10 °C. All samples were later stored at -80 °C until further use.

Unless stated otherwise, all kits were used according to manufacturer's instructions. Viral RNA was isolated from samples using the MinElute virus spin Kit (Qiagen, Hilden, Germany). RNA from whole blood or WBC was isolated using TRI Reagent® and the Direct-zol™ RNA MiniPrep (Zymo Research Europe, Freiburg, Germany) following manufacturer's instructions and including an in-column DNase I treatment. Reverse transcriptase-polymerase chain reaction (RT-PCR) was performed using SuperScript™ III One-Step RT-PCR System (Life Technologies GmbH, Darmstadt, Germany) using primers 5 (5'- TGCTGGAGATGGTTTAATTCAATC-3') and 12 (5'-TAGAGGAGACCAGGTCACTGT-3') targeting a 514-nt fragment of the 3'-end of the haemagglutinin (H) gene. Amplicons were purified using Qiagen PCR purification Kit (Qiagen, Hilden, Germany). Sequencing was bidirectional and conducted using the fluorescent Big Dye Terminator Cycle sequencing Kit 3.1 (ABI, Darmstadt, Germany). Sequences were analyzed on an ABI model 3130xl Genetic Analyzer (ABI). Editing of the sequences was carried out with BIOEDIT v.7.0.9.0 (Hall 1999). A BLAST search at the NCBI server (http://blast.ncbi.nlm.nih.gov/Blast.cgi) was performed to find out whether the sequence fragments obtained matched known CDV sequences.

All RT-PCR positive samples were screened more than once to confirm their positive status.

Serological assays: CDV antibody titers in sera were measured using ELISAs established at four laboratories between 1994 and 2015 (Department of Infectious Diseases, Institute of Virology, Veterinary University Hannover, Germany; Animal Health Diagnostic Centre, College of Veterinary Medicine, Cornell University, Ithaca, NY 14853, USA; Leibniz Institute for Zoo and Wildlife Research, Berlin, Germany; Diagnostic Laboratory, Institute of Virology, University of Giessen, Giessen, Germany). The titer level considered to indicate significant exposure to CDV was set by each laboratory.

(c) *Assigning infection states and sample sizes*

Individuals were assigned to one of three infection states (susceptible, infected and recovered) using three criteria: 1) the presence or absence of clinical signs of CDV infection; 2) RT-PCR screening results for the presence (viropositive) or absence (vironegative) of the virus, and 3) CDV antibody titers indicative of exposure to the virus (seropositive) or indicative of no exposure to the virus (seronegative). Information from these three citeria were combined as tabulated in Table S1 to define the state of an individual within a particular year. Sample sizes are presented in terms of number of individuals and number of states. The reason why the number of susceptible and the recovered state exceeds the number of

individuals is because (as mentioned in the methods) any individual classified as (i) ‘susceptible’ in a given year was further classified as ‘susceptible’ during all previous years when the individual was detected; (ii) ‘infected’ in a given year was classified as ‘susceptible’ during all previous years, and ‘recovered ‘during all subsequent years when the individual was detected; (iii) ‘recovered’ in a given year was classified as ‘recovered’ during all subsequent years when the individual was detected.

Table S1. How infection states were assigned. Sample sizes in terms of number of individuals (*N* individuals) and number of (infection) states (*N* states) are provided for females and males.

	Serology (CDV antibodies)	**Clinical sign**	**Virology (PCR screening)**	♀			♂		
				***N* individuals**		***N* states**	***N* individuals**		***N* states**
Susceptible	Seronegative	No sign	Vironegative	5	38	195	6	59	115
	Seronegative	No sign	Unknown	8			6		
	Unknown	No sign	Vironegative (cubs)	25			47		
Infected	Seropositive or Unknown	Sign observed	Unknown	52	54	54	35	38	38
	Seropositive or Unknown	Sign observed	Viropositive	0			1		
	Seropositive or Unknown	No sign	Viropositive	2			1		
Recovered	Seropositive	No sign	Unknown	4	12	314	3	8	88
	Seropositive	No sign	Vironegative	8			5		

(d) *Sample sizes for combinations of demographic, social and infection states*

We considered 24 known states for the females and 18 ones for the males, representing the different combinations of demographic, social and infection states (the 'dead' state cannot appear as an initial state). The possible combination states in females are: cub-low-ranking-susceptible (C.L.S), cub-low-ranking-infected (C.L.I), cub-low-ranking-recovered (C.L.R), cub-high-ranking-susceptible (C.H.S), cub-high-ranking-infected (C.H.I), cub-high-ranking-recovered (C.H.R), subadult-low-ranking-susceptible (SA.L.S), subadult-low-ranking-infected (SA.L.I), subadult-low-ranking-recovered (SA.L.R), subadult-high-ranking-susceptible (SA.H.S), subadult-high-ranking-infected (SA.H.I), subadult-high-ranking-recovered (SA.H.R), breeder-low-ranking-susceptible (B.L.S), breeder-low-ranking-infected (B.L.I), breeder-low-ranking-recovered (B.L.R), breeder-high-ranking-susceptible (B.H.S), breeder-high-ranking-infected (B.H.I), breeder-high-ranking-recovered (B.H.R), non-breeder-low-ranking-susceptible (NB.L.S), non-breeder-low-ranking-infected (NB.L.I), non-breeder-low-ranking-recovered (NB.L.R), non-breeder-high-ranking-susceptible (NB.H.S), non-breeder-high-ranking-infected (NB.H.I), non-breeder-high-ranking-recovered (NB.H.R). Females with an unknown infection state (U) could be assigned to one of 8 combinations of known demographic and social states (C.L.U, C.H.U, SA.L.U, SA.H.U, B.L.U, B.H.U, NB.L.U, NB.H.U).

The possible combinations states in males are: cub-low-ranking-susceptible (C.L.S), cub-low-ranking-infected (C.L.I), cub-low-ranking-recovered (C.L.R), cub-high-ranking-susceptible (C.H.S), cub-high-ranking-infected (C.H.I), cub-high-ranking-recovered (C.H.R), Pre-breeder-low-ranking-susceptible (PB.L.S), Pre-breeder-low-ranking-infected (PB.L.I), Pre-breeder-low-ranking-recovered (PB.L.R), Pre-breeder-high-ranking-susceptible (PB.H.S), Pre-breeder-high-ranking-infected (PB.H.I), Pre-breeder-high-ranking-recovered (PB.H.R), breeder-low-ranking-susceptible (B.L.S), breeder-low-ranking-infected (B.L.I), breeder-low-ranking-recovered (B.L.R), breeder-high-ranking-susceptible (B.H.S), breeder-high-ranking-infected (B.H.I), breeder-high-ranking-recovered (B.H.R). Males with an unknown infection states could be assigned one of 6 combinations of known demographic and social states (C.L.U, C.H.U, PB.L.U, PB.H.U, B.L.U, B.H.U). In Table S2 we summarize and provide sample sizes for all combinations of states used in the female and male capture-mark-recapture (CMR) data sets.

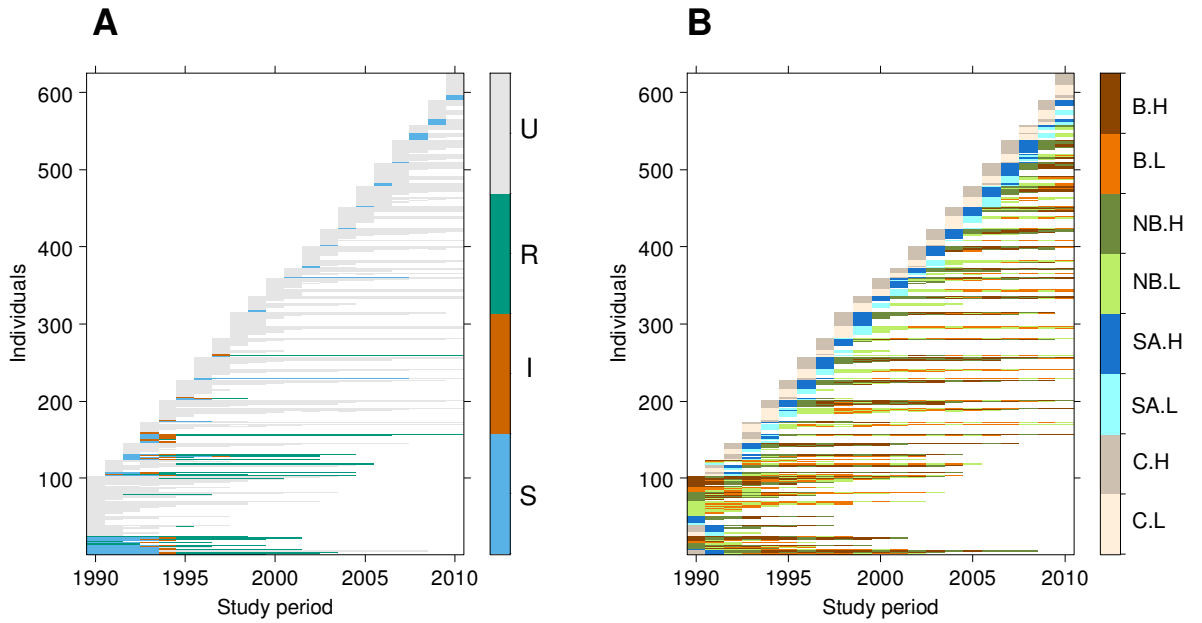
Table S2. State definition, abbreviation and sample sizes for each combination of a given demographic, social and infection state, as used in the female and male CMR data sets. The infection state was assigned with uncertainty and thus included an unknown infection state. The grey shaded rows indicate that the sample size for the given combination of demographic, social and infection states was < 5. These combinations of states were not tested separately during the model selection procedure; instead they were pooled with other social or infection states to increase the global sample size.

Demographic states	Social states	Infection states	Abbreviated state combination	*N* ♀	*N* ♂
Cub (C): 0−365 days	Low (L)	Susceptible (S)	C.L.S	31	26
		Infected (I)	C.L.I	5	7
		Recovered (R)	C.L.R	0	0
		Unknown (U)	C.L.U	201	240
	High (H)	Susceptible (S)	C.H.S	45	45
		Infected (I)	C.H.I	8	8
		Recovered (R)	C.H.R	0	0
		Unknown (U)	C.H.U	249	256
Subadult (SA): 366−720 days	Low (L)	Susceptible (S)	SA.L.S	5	-
		Infected (I)	SA.L.I	6	-
		Recovered (R)	SA.L.R	3	-
		Unknown (U)	SA.L.U	160	-
	High (H)	Susceptible (S)	SA.H.S	19	-
		Infected (I)	SA.H.I	8	-
		Recovered (R)	SA.H.R	1	-
		Unknown (U)	SA.H.U	223	-
Non-Breeder (NB): have not given birth to a litter	Low (L)	Susceptible (S)	NB.L.S	23	-
		Infected (I)	NB.L.I	3	-
		Recovered (R)	NB.L.R	53	-
		Unknown (U)	NB.L.U	409	-
	High (H)	Susceptible (S)	NB.H.S	26	-
		Infected (I)	NB.H.I	9	-
		Recovered (R)	NB.H.R	79	-
		Unknown (U)	NB.H.U	383	-
Pre-breeder (PB): 366 days – dispersal or first reproduction	Low (L)	Susceptible (S)	PB.L.S	-	1
		Infected (I)	PB.L.I	-	3
		Recovered (R)	PB.L.R	-	1
		Unknown (U)	PB.L.U	-	220
	High (H)	Susceptible (S)	PB.H.S	-	13
		Infected (I)	PB.H.I	-	9
		Recovered (R)	PB.H.R	-	6
		Unknown (U)	PB.H.U	-	223
Breeder (B): ♀: have given birth to a litter ♂: reproductive natal or immigrant male	Low (L)	Susceptible (S)	B.L.S	21	13
		Infected (I)	B.L.I	7	0
		Recovered (R)	B.L.R	77	9
		Unknown (U)	B.L.U	302	644
	High (H)	Susceptible (S)	B.H.S	25	17
		Infected (I)	B.H.I	8	11
		Recovered (R)	B.H.R	101	72
		Unknown (U)	B.H.U	358	802

(e) *Graphical representation of demographic, social and infection states*

Fig. S1 illustrates the CMR data sets for both females and males for the study period from 1990 to 2010.

Females:



Males:

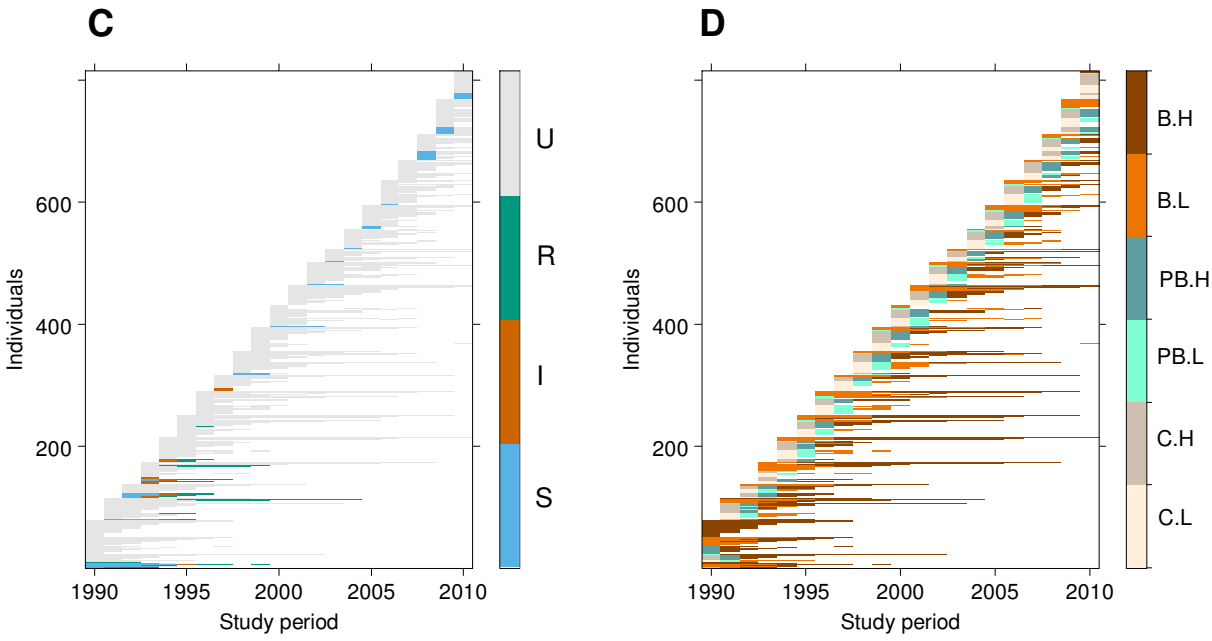


Figure S1. Graphical visualization of the female and male CMR data sets. The study period is represented on the x-axis and ranges from 1990 to 2010. Each 'row' on the y-axis corresponds to one individual from its year of first observation to its year of disappearance. Plots (**A**) and (**C**) focus on infection and show all females ($N = 625$) and all males ($N = 816$), respectively, in 'susceptible' (blue), 'infected' (orange), 'recovered' (green) and 'unknown' (grey) states during each year of the study period. Plots (**B**) and (**D**) focus on demographic and social states. Starting from the bottom of each plot, for (B) females: cub-low-ranking (C.L), cub-high-ranking (C.H), subadult-low-ranking (SA.L), subadult-high-ranking (SA.H), non-breeder-low-ranking (NB.L), non-breeder-high-ranking (NB.H), breeder-low-ranking (B.L), breeder-high-ranking (B.H) and for (**D**) males: cub-low-ranking (C.L), cub-high-ranking (C.H), pre-breeder-low-ranking (PB.L), pre-breeder-high-ranking (PB.H), breeder-low-ranking (B.L), breeder-high-ranking (B.H). The legend on the right side of each plot shows the color code for the states considered.

(f) *Modelling: Multi-event capture-mark-recapture approach*

Goodness-of-fit (model validation): The use of CMR and MECMR models relies on several important assumptions, which we detail and test in this section, using a goodness-of-fit (GOF) test. This test allows us to check the validity of the model, by verifying that the "detected section of the population" is representative of the section of the population that is undetected. More specifically, this test allows to verify that the fate and the detection of a given individual is (1) independent from the fate and the detection of any other individual, and that it is not influenced by (2) the monitoring design or the (3) behaviour of that individual (Pradel, Gimenez & Lebreton 2005; Choquet *et al.* 2009). Any violation in these assumptions can lead to the underestimation of the variance of parameters and result in the selection of potentially biased (over-parameterized) models (Pradel, Gimenez & Lebreton; Choquet *et al.* 2009).

To verify that we fulfilled these assumptions, we conducted a preliminary assessment of the GOF of the most parameterized model to the data, by calculating chi-square values in contingency tables using the software U-CARE 2.3.2 (Choquet *et al.* 2009).

The validation of our model was first done with the test '3G.SM'. This overall composite test is based on the conjunction of several null hypotheses depicting the main assumptions stated above. We did not find any significant departure from those assumptions, meaning that the

fully parameterized model adequately fitted the data (χ^2 = 75.69, df=143 p-value = 0.7 for females and χ^2 = 31.3, df = 37, p-value = 0.7 for males).

We then performed more specific tests. The test '3G.SR' is aimed at detecting a potential lack-of-fit due to heterogeneity in individual detection or the presence of individuals that temporarily move beyond the core study area. If the presence of such transient individuals on the study site is overlooked, survival will be underestimated (Choquet *et al.* 2009). The null hypothesis states that "there is no difference in the probability of being later re-detected between "new" and "old" individuals detected simultaneously." (Choquet *et al.* 2009). This test was not significant for both females (χ^2= 13.7, df = 32, p-value = 0.99) and males (χ^2 = 31.3, df = 37, p-value = 0.7), showing that our estimations are not biased by potential heterogeneity in individual detection or temporal absence from the study area.

The test 'M.ITEC' for 'trap-dependence' is aimed at detecting a potential lack-of-fit due to the monitoring design resulting in some individuals responding positively or negatively to the capturing/detection method (e.g.. when some individuals are attracted to baited camera traps or afraid of flashing cameras.). It tests the null hypothesis that "there is no difference in the probabilities of being re-encountered in the different states at the occasion $_{i+1}$ between the animals in the same state at occasion $_{i}$ whether encountered or not encountered at this date, conditional on presence at both occasions" (Choquet *et al.* 2009). Here we could not run this test because a great majority of females and males were re-detected the year following their first detection, resulting in insufficient variation in the re-detection probability to run this test. However, our close observations of thousands of hyenas at communal dens during the past thirty years do not suggest in any way that some clan members would have avoided the vicinity of communal dens owing to the presence of the research vehicle. We are confident that our monitoring design did not influence the probability of re-detecting an individual after its first detection.

Finally, the test 'WBWA' (for WhereBeforeWhereAfter) is aimed at verifying that the movements of animals are not based on the knowledge of previously visited sites (Pradel, Gimenez & Lebreton 2005). This test is typically used in multisite CMR models that record encounters with marked animals (very often bird species that establish breeding colonies) over different sites. In multistate CMR models, 'sites' correspond to 'states'. Such 'memory effect' would constitute a violation of the assumptions of CMR models because it can lead to different behaviours (and hence biological states) for individuals belonging to the same set of

animals detected at given occasion, depending on which states they had been previously (Pradel, Gimenez & Lebreton 2005). This test is based on the null hypothesis that 'there is no difference in the expected state of next reencounter among individuals previously encountered in the different states" (Pradel, Wintrebert & Gimenez 2003, Choquet *et al.* 2009). In our case, a non-significant test would confirm that the state transition of an individual detected at a given occasion does not depend on its state at the previous occasion. This test was not significant for both females (χ^2 =165.5, df = 172, p-value = 0.62) and males (χ^2 = 26.8, df = 28, p-value = 0.53), showing that there was no effect of past state.

True and apparent survival probability: In the CMR modelling context, the estimated survival probability is not always the "true" survival because mortality cannot be distinguished, but the "apparent" survival, which combines both the effect of mortality and emigration (Schaub *et al.* 2004). In the female analysis, we estimated the true survival of adults since females were philopatric whereas in the male analysis the survival of adults was likely underestimated because immigrant males can re-disperse (Höner *et al.* 2007, M.L. East personal obs.) and join clans outside the study area. Because female and male cubs, female subadults and a great majority of male pre-breeders are also philopatric, the estimate of their survival is also an estimate of true survival.

Supplementary information on model structure:

(i) Modeling the initial state probabilities: For females the matrix $\boldsymbol{I_S}$ is the matrix of the initial state probabilities, composed of a single row with 24 states, representing the different combinations of demographic, social and infection states (the 'dead' state cannot appear as an initial state). This vector provides information about the proportion of females in different combinations of demographic, social and infection states in the female CMR data set.

$$\boldsymbol{Is}=[\pi\ \pi\ \pi\ \pi\ \pi\ \pi\ \pi\ \pi\ \pi\ \pi\ \pi\ \pi\ \pi\ \pi\ \pi\ \pi\ \pi\ \pi\ \pi\ \pi\ \pi\ \pi\ (1-\pi)] \quad (1)$$

with π being the probability of being in a given initial demographic, social and infection state. The entries in I_s correspond to the probabilities (from left to right) of being in a combination of states described in order in the section above (2.d)

A similar matrix was constructed for males with 18 states corresponding to the initial state probabilities as cubs, pre-breeders, or breeders in each social and infection state.

ii) Modeling the biological processes for males:

As the social and infection processes occurring in males were modeled similarly as for females, we only show here how we modelled male demographic transitions and survival probabilities.

The transitions between the demographic states of males can be represented by the matrix $\boldsymbol{Demo}_{\boldsymbol{males}}$ that considers the transitions of males, to different demographic states, that is to the pre-breeder (PB) and breeder (B) states, as defined in the main text and summarized in Table S2. Surviving cubs (C) become PB with a probability of 1.

$$\boldsymbol{Demo}_{\boldsymbol{males}} = \begin{matrix} C \\ PB \\ B \end{matrix} \begin{bmatrix} 0 & 1 & 0 \\ 0 & 1-\psi & \psi \\ 0 & 0 & 1 \end{bmatrix} \quad (2)$$

with Ψ being the transition probability to the B state accessible from PB males and 1- Ψ its complement. Each entry in $Demo_{males}$ is the probability of transition from a 'starting' demographic state, three rows corresponding to the demographic states C, PB, B on the left side of the matrix**,** to an 'ending' demographic state – three columns corresponding to the demographic states C, PB, B (not shown for simplicity).

Survival: The matrix $\boldsymbol{Survival}_{\boldsymbol{males}}$ account for the annual survival probabilities of males:

$$\boldsymbol{Survival}_{\boldsymbol{males}} = \begin{matrix} C \\ PB \\ B \\ Dd \end{matrix} \begin{bmatrix} \phi & 0 & 0 & 1-\phi \\ 0 & \phi & 0 & 1-\phi \\ 0 & 0 & \phi & 1-\phi \\ 0 & 0 & 0 & 1 \end{bmatrix} \quad (3)$$

Each entry in $Survival_{males}$ is the probability of survival of a given demographic state. For males in this matrix these survival probabilities comprise three rows corresponding to the demographic states C, PB, B on the left side of the matrix. Dd represents the transition to the dead state.

iii) Combining all biological processes for males:

The matrix ***Infection /Social*** (similar for both females and males) then combines the infection matrix with the social matrix, and displays the possible transitions to the different infection states within and between the two social states:

$$\boldsymbol{Infection/Social} = \begin{array}{c} \\ L.S \\ H.S \\ L.I \\ H.I \\ L.R \\ H.R \end{array} \begin{array}{c} \begin{array}{cccccc} L.S & H.S & L.I & H.I & L.R & H.R \end{array} \\ \begin{bmatrix} (1-\beta)*r & (1-\beta)*(1-r) & \beta*r & \beta*(1-r) & 0 & 0 \\ (1-\beta)*(1-r) & (1-\beta)*r & \beta*(1-r) & \beta*r & 0 & 0 \\ 0 & 0 & 0 & 0 & r & (1-r) \\ 0 & 0 & 0 & 0 & (1-r) & r \\ 0 & 0 & 0 & 0 & r & (1-r) \\ 0 & 0 & 0 & 0 & (1-r) & r \end{bmatrix} \end{array} \quad (4)$$

Each entry in ***Infection/Social*** is the probability of transition from a 'starting' combination of a social (L and H) and an infection (S, I and R) state, with six rows corresponding to the combinations L.S, H.S, L.I, H.I, L.R, H.R on the left side of the matrix, to an 'ending' combination of social and infection states, with six columns corresponding to the combinations L.S, H.S, L.I, H.I, L.R, H.R (not shown for simplicity). The * symbol represents the multiplication of parameters.

The final matrices $\boldsymbol{Final_{females}}$ and $\boldsymbol{Final_{males}}$ represent the transition probabilities of surviving females and males, respectively, to successive demographic states given their specific social and infection states as formulated in ***Infection/Social*** (see Table S3 below). Each element representing the transition probability of C, SA, B and NB (for females) or C, PB and B (for males) represents a sub-matrix of dimension ***Infection/Social***. $\boldsymbol{Final_{females}}$ and $\boldsymbol{Final_{males}}$ are thus two matrices of total dimensions 25 by 25 and 19 by 19, respectively, for females and males as they consider demographic, social and infection states and the absorbing death state. The × symbol represents a multiplication of matrices. We also implemented a diagonal matrix of 1, denoted as *Diag*(1), with similar dimension as ***Infection/Social*** to better represent the extension of both the survival and demography matrices when combining all matrices into the two final matrices of $\boldsymbol{Final_{females}}$ and $\boldsymbol{Final_{males}}$ (Table S3).

iv) Modeling the observation process:

Detection: The detection matrices accounted for the probability of detection *p* and the probability of non-detection 1-*p* of females and males in different demographic, social and infection states. For simplicity, we only illustrate below the detection matrix of females within their demographic states, $\boldsymbol{Det_{Df.}}$. Each element of $\boldsymbol{Det_{Df}}$ is multiplied by a diagonal matrix of 1, ***Diag*(1)** of dimension ***Infection/Social*** to later extend the $\boldsymbol{Det_{Df}}$ matrix across the social and infection states. Multiplying each transition by the diagonal matrix ***Diag*(1)**, with similar dimension as ***Infection/Social***, reflects the extension of the detection matrix across the social and infection ones:

$$\mathbf{Det}_{Df} = \begin{array}{c} \\ C \\ SA \\ B \\ NB \\ Dd \end{array} \begin{bmatrix} (1-p)*Diag(1) & p*Diag(1) & 0 & 0 & 0 \\ (1-p)*Diag(1) & 0 & p*Diag(1) & 0 & 0 \\ (1-p)*Diag(1) & 0 & 0 & p*Diag(1) & 0 \\ (1-p)*Diag(1) & 0 & 0 & 0 & p*Diag(1) \\ 1 & 0 & 0 & 0 & 0 \end{bmatrix} \quad (5)$$

Assignment of infection states: The partial observation matrix ***PO*** below represents cases where individuals are detected but their infection state is unknown. This is characterized by the last column, whose elements 1-δ denote the probability of failing to assign an infection state to an individual that has been detected. Each element of the matrix is multiplied by a diagonal matrix of 1, ***Diag*(1)** of dimension 8 by 8 for females and of dimension 6 by 6 for males. Such an extension of the ***PO*** matrix represents the partial observation of infection states across the demographic and social states:

$$\boldsymbol{PO} = \begin{array}{c} \\ S \\ I \\ R \end{array} \begin{array}{c} \begin{array}{cccc} \quad & \quad & \quad & \quad \end{array} \\ \begin{bmatrix} \delta * Diag_2(1) & 0 & 0 & (1-\delta) * Diag_2(1) \\ 0 & \delta * Diag_2(1) & 0 & (1-\delta) * Diag_2(1) \\ 0 & 0 & \delta * Diag_2(1) & (1-\delta) * Diag_2(1) \end{bmatrix} \end{array} \quad (6)$$

The multiplication of the detection matrix by the partial observation matrix then provided the final matrix for the observation process (not shown), reflecting the potential incomplete detection of individuals as well as the potential uncertainty on individual infection states.

Table S3 The final matrices $\boldsymbol{Final_{females}}$ and $\boldsymbol{Final_{males}}$ (for the biological process) represent the transition probabilities of surviving females and males, respectively, to successive demographic states given their specific social and infection states as formulated in ***Infection/Social***. ***Diag*(1)** is a diagonal matrix of 1, with similar dimension as ***Infection/Social***. Both matrices are the products of the survival and the demographic matrices of females and males, respectively i.e. $Final_{females} = Survival$ x $Demo$, and $Final_{males} = Survival_{males}$ x $Demo_{males,}$)

$$\boldsymbol{Final_{females}} = \begin{array}{c} \\ C \\ SA \\ B \\ NB \\ Dd \end{array} \begin{bmatrix} \phi \times Infection/Social & 0 & 0 & 0 & 1-\phi \\ 0 & \phi \times Infection/Social & 0 & 0 & 1-\phi \\ 0 & 0 & \phi \times Infection/Social & 0 & 1-\phi \\ 0 & 0 & 0 & \phi \times Infection/Social & 1-\phi \\ 0 & 0 & 0 & 0 & 1 \end{bmatrix} \times \begin{bmatrix} 0 & Diag(1) & 0 & 0 & 0 \\ 0 & 0 & \psi \times Diag(1) & (1-\psi) \times Diag(1) & 0 \\ 0 & 0 & \psi \times Diag(1) & (1-\psi) \times Diag(1) & 0 \\ 0 & 0 & 0 & (1-\psi) \times Diag(1) & 0 \\ 0 & 0 & 0 & 0 & 1 \end{bmatrix}$$

(7)

$$\boldsymbol{Final_{males}} = \begin{array}{c} \\ C \\ PB \\ B \\ Dd \end{array} \begin{bmatrix} \phi \times Infection/Social & 0 & 0 & 0 \\ 0 & \phi \times Infection/Social & 0 & 0 \\ 0 & 0 & \phi \times Infection/Social & 0 \\ 0 & 0 & 0 & 1 \end{bmatrix} \times \begin{bmatrix} 0 & Diag(1) & 0 & 0 \\ 0 & 0 & \psi \times Diag(1) & 0 \\ 0 & 0 & Diag(1) & 0 \\ 0 & 0 & 0 & 1 \end{bmatrix}$$

(8)

$$\boldsymbol{Diag(1)} = \begin{bmatrix} 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 0 & 1 \end{bmatrix} \quad (9)$$

v) Example of encounter history formulation: Finally, we illustrate an observed encounter history, formulated for a fictive female detected for three consecutive years following birth. The fictive female was detected as a high-ranking cub at the first sampling occasion and assigned to be 'susceptible' (denoted as C.H.S), then detected as a high-ranking subadult with an unknown disease state the following year (SA.H.U) and finally detected as low-ranking non-breeder with an unknown disease state, (NB.L.U). The true encounter history of this individual could have been one of three possibilities:

(1) C.H.S → SA.H.S → NB.L.S,

(2) C.H.S → SA.H.S → NB.L.I,

(3) C.H.S → SA.H.I → NB.L.R

The probability of observing the illustrated encounter history is equivalent to the following formulation:

$$
\begin{aligned}
&\mathrm{P(C.H.S, SA.H.U, NB.L.U)} = \\
&\phi_1^C r_1^H (1-\beta_1) p_1^C \delta_1^S \phi_2^{SA} (1-r_2^H)(1-\beta_2)(1-\psi_2^{SA\to B}) p_2^{SA} (1-\delta_2^S) p_3^{NB} (1-\delta_3^S) + \\
&\phi_1^C r_1^H (1-\beta_1) p_1^C \delta_1^S \phi_2^{SA} (1-r_2^H)(\beta_2)(1-\psi_2^{SA\to B}) p_2^{SA} (1-\delta_2^S) p_3^{NB} (1-\delta_3^I) + \\
&\phi_1^C r_1^H (\beta_1) p_1^C \delta_1^S \phi_2^{SA} (1-r_2^H)(1-\psi_2^{SA\to B}) p_2^{SA} (1-\delta_2^I) p_3^{NB} (1-\delta_3^R)
\end{aligned}
\qquad (10)
$$

where the indexed number *1* represents the first sampling occasion (i.e. the first detection occasion), *2* the second and *3* the third, ,ϕ the annual survival probability, *r*, the probability of staying in the same social state, (1- *r* being its complement), β, the infection probability (i.e. the probability of transition: susceptible → infected, with 1- β being its complement), ψ the transition probability to the B state (with the upper index SA→B denoting the specific transition from subadult to breeder, i.e, breeding probability of primiparous females); with 1- ψ being the complement), *p* the detection probability (1-*p* being its complement, i.e. the probability that an individual is not detected) and δ the assignment probability of an individual into an infection state (susceptible, infected or recovered) conditional on its detection (1- δ being its complement, i.e., the probability that an individual has its infection state not assigned, i.e. that it is 'unknown').

(g) *Predictions (models tested)*

This section details the specific predictions corresponding to the models tested for females and males. The expected effects on transition probabilities among demographic and social states and initial state probabilities are detailed below. The expected effects of social status on both the infection and the survival probabilities, based on previous studies, are presented in Table S4.

Transition probabilities among demographic and social states and initial state probabilities
Among females, we tested whether demographic or social states, or the interaction between both terms, influenced the breeding transition probability, expecting NB more likely to breed than B and SA, and high-ranking females more likely to breed than low-ranking ones. We also tested the effect of social status on the probability of transition between social states, with the expectation that the probability of high-ranking females becoming low-ranking is higher than the probability of low-ranking females becoming high-ranking. Among males we tested the effect of demographic and social states, and the interaction between both terms on the breeding transition probability, with the expectation that high-ranking PB were more likely to breed than low-ranking ones. We also controlled for the effect of social status on transition probabilities between social states, expecting the probability of becoming high-ranking to be higher than the probability of becoming low-ranking because of the queuing system. We did not test for effects of infection states on demographic or social transitions to avoid over-parameterizing the models.

For both females and males we modeled initial state probabilities, as preliminary model runs suggested that including this parameter substantially improved model ranking. We did not have specific predictions regarding the effects potentially affecting initial state probabilities. We tested effects of demographic, social and infection states and two-way interactions between these states.

Table S4: Predictions of the impact of social status on individual infection probability with CDV (β) and on survival (ϕ) of infected and recovered spotted hyenas in each sex and demographic state in relation to the two hypotheses (exposure to CDV vs. allocation of body resources to immune processes). In any case, we always expect survival of susceptible to be higher than survival of infected. As we do not expect CDV infection to affect adult survival we did not predict interaction effects between social and infection states among adult females (i.e. breeder and non-breeder categories) and among adult males (breeder category). The abbreviations describe the demographic (C for cubs, SA for subadults, B breeders, NB for non-breeders and PB

for Pre-breeder males), social (H and L for high and low-ranking individuals respectively) and infection states (S for susceptible, I for infected and R for Recovered) and are explained Table S2. The ampersand ('&') indicates that categories are grouped (see legend of Table S2 for details).

Demographic state & sex	**Predictions**	
	Exposure	**Resource allocation to immune processes**
Cub (C) ♀+♂	β: C.H.S > C.L.S ϕ: C.H.I. < C.H.S ϕ: C.L.I. < C.L.S ϕ: C.H.I ≈ C.L.I Exposure to CDV should be similar for high and low-ranking cubs between 0 and 6 months when cubs are den-bound and solely depend on milk. Between 6 and 12 months, high-ranking cubs have higher contact rates with clan members than low-ranking ones (as their mothers provide them with intense behavioral support (East, Hofer & Wickler 1993; East *et al.* 2001; East *et al.* 2009). Thus, high-ranking cubs should globally be more exposed to CDV than low-ranking cubs, between 0 and 12 months. High-ranking cubs have a higher survival rate than low-ranking ones in the absence of CDV infection (Hofer & East 1993b; Hofer & East 2003). Thus, if high-ranking cubs are more exposed than low-ranking ones, all else being equal, the survival rate of high-ranking infected cubs should be reduced, and thus is, for simplicity, expected not to differ too much from that of low-ranking infected cubs.	β: C.H.S < C.L.S ϕ: C.H.I > C.L.I As high-ranking cubs are more often nursed than low-ranking cubs (Hofer & East 1993b; Hofer & East 2003) and receive more milk during their development (Hofer *et al.* 2016), they should have a higher opportunity to allocate body resources to immune processes. Therefore, the outcome of infection should be less severe for high-ranking cubs than for low-ranking ones, both in terms of β and ϕ.
Subadult (SA) ♀	β: SA.H.S > SA.L.S ϕ: SA.H.I ≈ SA.L.I ϕ: SA.H.R ≈ SA.L.R	β: SA.H.S < SA.L.S ϕ: SA.H.I ≤ SA.H.R ϕ: SA.L.I ≤ SA.L.R ϕ: SA.H.I&R > SA.L.I&R

	Exposure to CDV should be higher for high-ranking than low-ranking subadults because the former receive more behavioral support from their mothers and thus have higher contact rates with conspecifics (East, Hofer & Wickler 1993; East & Hofer 2001; East *et al.* 2009 and see East *et al.* 2001 for data on contact rates). In the absence of CDV infection, high-ranking subadults have higher survival rates than low-ranking ones (Hofer & East 1993b; Hofer & East 2003). Thus, if high-ranking subadults are more exposed than low-ranking ones, all else being equal, the survival rate of high-ranking infected subadults should be depressed to a level equivalent to that of low-ranking infected subadults.	As high-ranking subadults are more often nursed than low-ranking cubs (Hofer & East 1993b; Hofer & East 2003), and receive more milk during their development (Hofer *et al.* 2016), they should have a higher opportunity to allocate body resources to immune processes. Therefore, the outcome of infection should be less severe for high-ranking subadults than for low-ranking ones, both in terms of β and ϕ. Because fighting CDV infection is costly to the body, recovered subadults should have a survival rate similar to that of infected ones in each social status category.
Breeder (B) ♀	β: B.H.S > B.L.S Exposure to CDV should be higher for high-ranking than low-ranking breeders because high-ranking breeders have higher contact rates (East *et al.* 2001), including contacts to cubs at the entrance of den holes and aerosol infection while sniffing dens (East, Hofer & Turk 1989).	β: B.H.S < B.L.S Owing to the high energetic cost of both lactation and a low social status (Goymann *et al.* 2001; Hofer *et al.* 2016), low-ranking breeders should have a poorer opportunity to allocate resources for vital body functions and immune processes than high-ranking breeders. Thus, low-ranking breeders should be more infected with CDV (as found in East *et al.* 2015 with a hookworm parasite).
Non-breeder (NB) ♀	β: NB.H.S > NB.L.S Exposure to CDV should be higher for high-ranking than low-ranking non-breeders because high-ranking non-breeders have higher contact rates (East *et al.* 2001), including contacts to cubs at the entrance of den holes and aerosol infection while sniffing dens (East, Hofer & Turk 1989).	β: NB.H.S < NB.L.S High-ranking non-breeders should have a (slightly) higher opportunity to allocate body resources to immune processes than low-ranking ones. Therefore, CDV infection probability should be higher among low-ranking non-breeders than high-ranking ones.

Pre-breeder (PB) ♂	β: PB.H.S > PB.L.S ϕ: PB.H.I ≈ PB.L.I Exposure to CDV should be higher for high-ranking than low-ranking pre-breeders because the former receive more behavioral support from their mothers and thus have higher contact rates with conspecifics (East, Hofer & Wickler 1993; East *et al.* 2001; East *et al.* 2009 and see East *et al.* 2001 for data on contact rates). In the absence of CDV infection, high-ranking pre-breeders have higher survival rates than low-ranking ones (Hofer & East 1993b; Hofer & East 2003). Thus, if high-ranking pre-breeders are more exposed than low-ranking ones, all else being equal, the survival rate of high-ranking infected pre-breeders should be depressed to a level equivalent to that of low-ranking infected pre-breeders.	β: PB.H.S < PB.L.S ϕ: PB.H.I ≤ PB.H.R ϕ: PB.L.I ≤ PB.L.R ϕ: PB.H.I&R > PB.L.I&R High-ranking pre-breeders receive more maternal milk, have a greater access to resources and thus higher survival rates than low-ranking ones (Hofer & East 1993b; Hofer & East 2003; Höner *et al.* 2007). Thus, the outcome of infection in terms of β and ϕ should be more severe among low-ranking pre-breeders than high-ranking ones, as the former should have a reduced opportunity to invest in immune processes. Because fighting CDV infection is costly to the body, recovered pre-breeders should have a survival similar to that of infected ones in each social status category.
Breeder (B) ♂	β: B.H.S > B.L.S Exposure to CDV should be higher for high-ranking than low-ranking breeders because high-ranking breeders are more tolerated around communal dens (East & Hofer 1991; East, Hofer & Wickler 1993; East & Hofer 2001) and thus have higher contact rates with conspecifics, including potentially infected cubs (see East *et al.* 2009 for data).	β: B.H.S = B.L.S High-ranking and low-ranking breeders should have similar opportunities to allocate body resources to immune processes. High-ranking males, have priority of access to food within their territory (in comparison to low-ranking males), but are more often present than low-ranking ones in their home territories thus probably experience more variation in prey abundance within territories than low-ranking makes, which spend more time feeding on the migratory herds (East & Hofer 1991). Therefore, CDV infection probability should be similar among low-ranking breeders than high-ranking ones.

3. Supplementary results

(a) *Social and breeding transition probabilities in females and males*

Females: High-ranking females were less likely than low-ranking ones to stay in their social status category, irrespective of their demographic or infection state (Table 2). The breeding probability of females depended on an interaction between demographic and social states: Non-breeders had the highest breeding probability, followed by breeders and then subadults and in all these demographic states, high-ranking females had a higher breeding probability than low-ranking ones (Table 2). The initial state probabilities depended on an interaction between demographic and social states ($\pi^{C.H} = 0.48 \pm 0.01$, $\pi^{C.L} = 0.38 \pm 0.01$, $\pi^{SA.H} = 0.02$ (S.E. < 0.01), $\pi^{SA.L} = 0.01$ (S.E. < 0.01), $\pi^{B.H} = 0.04$ (S.E. = 0.01), $\pi^{B.L} = 0.02$ (S.E. < 0.01), $\pi^{NB.H} = 0.03$(S.E. = 0.01), $\pi^{NB.L} = 0.04$ (S.E.< 0.01)). It indicates that at the first detection occasion, there was a predominance of cubs as compared to other demographic states and a pre-dominance of high-ranking females as compared to low-ranking females.

Males: The transition probability of staying in the same social status category for adult males was modeled as a constant as any other effects produced a model with non-identifiable parameters (see Table 2 for the value). The initial state probabilities depended on an interaction between demographic and social states ($\pi^{C.H} = 0.38 \pm 0.01$, $\pi^{C.L} = 0.33 \pm 0.01$, $\pi^{PB.H} = 0.02$ (S.E. < 0.01), $\pi^{PB.L} = 0.05$ (S.E. < 0.01), $\pi^{B.H} = 0.01$ (S.E. < 0.01), $\pi^{B.L} = 0.17$ (S.E. < 0.01). It indicates that at the first occasion, there was a predominance of cubs as compared to other demographic states.

(b) *Observation process (detection and assignment probabilities) for females and males*

Females

(i) The detection probabilities of females varied with their social status, irrespective of their demographic or infection state. Female detection was excellent on average, as expected, owing to the individual-based nature of this long-term study. High-ranking females were slightly more often detected than low-ranking ones (on average: high-ranking: $p^H = 0.99$ (S.E. < 0.01), low-ranking: $p^L = 0.98 \pm 0.01$). The model including demographic states was less well-ranked, indicating that there was no difference in the detection probability of cubs, subadults, breeders and non-breeders.

(ii) The assignment probabilities of S, I and R infection states were best predicted by a model including the effect of infection states (susceptible: δ^{S}: 0.85 ± 0.07; infected: δ^{I}: 0.14 ± 0.01; recovered: δ^{R}: 0.15 ± 0.02).

Males

(i) The detection probabilities of males, as for females, accounted for effects of social states, irrespective of their demographic or infection state. Overall, as for females and as expected, detection probabilities were very high. There was a higher detection of high-ranking males (p^{H} = 0.99, with S.E. < 0.01) than low-ranking ones (p^{L} = 0.86 ± 0.01), probably because high-ranking male breeders are more tolerated and thus more often present at communal dens than low-ranking ones (East & Hofer 1991).

(ii) The assignment probabilities of S, I and R infection states for males depended, as for females, on infection states (susceptible: δ^{S}: 0.37 ± 0.04; infected: δ^{I}: 0.05 < 0.01; recovered: δ^{R}: 0.09 ± 0.01).

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