Vaccination strategies to conserve the endangered African wild dog (*Lycaon pictus*)

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**A B S T R A C T**

Pathogens such as rabies virus and canine distemper virus present a significant risk to the long-term survival of some endangered African wild dog (*Lycaon pictus*) populations. Vaccination of wild dogs is one approach that might reduce population extinction risks; however, clear guidelines for how best to do this are lacking. Hence, we developed a mathematical model of wild dog population dynamics that integrates a pathogen transmission model and a vaccination process. Simulations indicated that the most effective vaccination strategy includes all age classes (when pups are 2-months old), prioritizes the breeding pair, and vaccinates at least 4 animals per pack included in the vaccination effort. In addition, populations for which an Allee effect, high rates of pathogen introduction, or low rates of recovery and immunity were simulated, required greater vaccination coverage (dogs/pack), to protect an equivalent number of packs compared to populations for which no Allee effect, low rates of pathogen introduction, or high rates of recovery and immunity were simulated. For populations in which some packs (high-risk) have a greater risk of pathogen exposure than others (low-risk), vaccinating both high- and low-risk packs, or only low-risk packs, is more effective than vaccinating only high-risk packs when pathogen introduction rates are high. Finally, model results suggest that vaccination of wild dog populations against pathogens, such as canine distemper virus, that do not cause 100% mortality and against which some wild dogs develop acquired immunity, may be unnecessary.

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1. Introduction

The African wild dog (*Lycaon pictus*) is one of the world’s most endangered carnivores with fewer than 8000 individuals, in fewer than 800 packs, remaining in the wild (summarized in KWS, 2010). Habitat loss and persecution are the major threats to wild dog conservation (Woodroffe et al., 2007), with many wild populations reduced to such small numbers that pathogens such as rabies virus and canine distemper virus (CDV) may pose a significant risk to their long-term persistence (Woodroffe et al., 1997). Hence, strategies for wild dog conservation call for management to reduce this risk (IUCN/SSC, 2007, 2008).

African wild dog populations occur at low densities and it is unlikely that they are capable of maintaining pathogens such as CDV and rabies in the absence of other host species. Although transmission rates within-packs are high, contact between packs is relatively infrequent, such that all animals within a pack may recover or die before they transmit infection to another pack (Mills, 1993; Woodroffe and Donnelly, in preparation). However, these viruses can be introduced through spillover from more abundant maintenance or reservoir hosts, such as domestic dogs (*Canis familiaris*). In principle, the risk that these pathogens pose to wild dog populations might be reduced either by preventing pathogen transmission from a reservoir, or by protecting individual wild dogs through immunization.

Addressing disease risks to wild dogs by managing pathogens in the reservoir is appealing because it avoids potential risks associated with handling an endangered species. However, several factors complicate this approach. First, there is uncertainty about which species are the reservoir host(s). In most of Africa, evidence suggests that domestic dogs are the principle reservoir responsible for the persistence of rabies (Leombo et al., 2008) and CDV (Alexander et al., 1996; Cleaveland et al., 2000; Gowtage-Sequeira et al., 2009); however, wild carnivore populations may also play a role in pathogen transmission dynamics (Craft et al., 2008; Pfükenyi et al., 2009; Prager et al., in preparation; Sabeta et al., 2007; Zulu et al., 2009). Second, wild dogs’ low population densities and wide-ranging behaviour mean that any management of reservoir hosts would need to be conducted over vast areas, entailing considerable effort and expense. Third, unless management directed exclusively at reservoir hosts completely eliminates the pathogen, spillover events can still occur and endanger wildlife health. For example, even with a domestic dog rabies vaccination...
program in place, a rabies outbreak occurred in highly endangered Ethiopian wolves (Canis simensis, Randall et al., 2006).

Given the potential difficulties of managing disease in reservoir hosts, it is worth considering vaccination of wild dogs themselves, to reduce the risks of disease-related population extinction. Captive studies suggest that vaccines against CDV and parvovirus are likely protective in wild dogs (van Heerden et al., 2002). In free-ranging wild dogs, administration of a single dose of inactivated rabies vaccine has failed to prevent deaths (Woodroffe, 2001); however, multiple doses appear to be protective (Hofmeyr et al., 2004) and modified live rabies vaccines, delivered orally, prompted a stronger immune response than did inactivated vaccines (Knobel et al., 2003). Hence, protection of free-ranging wild dogs by vaccination appears achievable in principle.

Because spillover events can occur from the pathogen reservoir, the goal of wild dog vaccination is not to eliminate the pathogen from the endangered population, but rather to protect a sufficient number of individuals from disease-related mortality such that population extinction is avoided (Haydon et al., 2002; USFWS, in press; Vial et al., 2006). The costs of conducting vaccination – in terms of funding, logistics, and (potentially) animal welfare – demand a vaccination program that maximizes population viability while minimizing the number of animals vaccinated.

The efficacy of vaccinating wild dogs might be influenced by the choice of individuals to vaccinate. Wild dogs live in cooperatively-breeding packs with the majority of offspring born to a dominant pair (Malcolm and Marten, 1982). Vaccine could be distributed among many animals in few packs, or few animals in many packs; lower reproductive success in smaller packs (component Allee effects: Coughenour and Macdonald, 2001) might influence this tradeoff. Logistics will also play a role: locating packs for vaccination is likely to be a limiting factor, making it easier to implement strategies which involve vaccinating fewer packs. Pups could be included in, or excluded from, vaccination, and dominant animals could be prioritized. In addition, where some packs within a population face a higher risk of pathogen exposure than others (perhaps because they live closer to domestic dogs, such as along a park boundary), vaccination might target higher- and/or lower-risk packs. The choice of delivery method might also affect the effectiveness of a vaccination strategy. Oral vaccines can only be delivered when packs have small pups in a den and thus can be located repeatedly at a predictable site (Knobel et al., 2002). In contrast, vaccination by injection is the most appropriate method outside the denning period. This difference in timing, and the fact that pups must reach adult size to safely dart for vaccination, would influence the age at which pups might be vaccinated.

We sought to identify the most effective vaccination strategy to protect African wild dogs from pathogen-related extinction events. Building on the work of Vial et al. (2006), we used a mathematical model of wild dog demography, with an integrated epidemiological model, to simulate a variety of vaccination scenarios. Specifically, we investigated the responses of wild dog populations, facing a range of pathogen exposure levels, to vaccination of (i) different numbers of animals per pack; (ii) different numbers of packs per population; (iii) pups of varying age; (iv) dominant animals; and (v) high- and/or low-risk packs. We also investigated the impact of Allee effects, and recovery from infection with pathogen-induced immunity, on the outcome of vaccination programs. In contrast to Vial et al. (2006), we measure vaccination benefits as increases in population size, rather than reductions in extinction probability, because untenable small populations may nevertheless have low extinction probabilities over the 20-years time period over which the populations are simulated.

2. Methods

The model is an individually-based, age-, sex- and pack-structured demographically stochastic representation of African wild dog population dynamics with an integrated epidemiologic model. The process of disease transmission and natural mortality were modeled with demographic stochasticity in continuous-time, but vaccination, birth, and pack fission and fusion occurred at fixed time points in the year. The model is therefore essentially individually-based. The details of the model structure are summarized below but described fully in Vial et al. (2006). The entire model was coded and run using Borland® Delphi 7™. Except where indicated otherwise, demographic and behavioral parameters (summarized in Box 1) were derived from a field study of wild dogs inhabiting rangelands of northern Kenya (Woodroffe, 2011). A sensitivity analysis of key demographic parameters (age-specific mortality rates and maximum pack size) was performed and is reported in Appendix A (Supplementary electronic material). The demographic processes are:

1. Births: If a dominant pair is present in a pack, one litter is produced on the 60th day of each year, with annual probability \( p_b = 0.95 \). The number of newborn pups in each pack is taken from a frequency distribution of litter size, and each pup is designated male or female with equal probability. Immediately before births occur, all animals undergo age-updating: surviving pups from the preceding year become yearlings, and yearlings become adults. The age of first reproduction is set to 2 years.

2. Deaths: Age-specific annual mortality rates, excluding disease-related mortality, are attributed to pups (\( m_p = 0.191 \)), yearlings (\( m_y = 0.093 \)), and adults (\( m_a = 0.192 \)) independent of sex.

3. Within-pack density dependence: If prior to age-updating the number of adults and yearlings exceeds a fixed threshold size (\( p_r = 15 \)), all female yearlings leave the pack as a single sex group (SSG). If the pack still exceeds the threshold, then all male yearlings also leave as an additional SSG. If the total number of SSGs exceeds the maximum number of SSGs allowed (chosen to be 5), additional SSGs are removed from the population.

4. Pack formation: Fusion occurs randomly and annually (on day 60 prior to breeding), between non-sibling SSGs of the opposite sex if the number of packs (excluding SSGs) is below a defined maximum number of packs (10).

Box 1

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age of first possible reproduction</td>
<td>Start of second year</td>
</tr>
<tr>
<td>Sex ratio at birth</td>
<td>1:1</td>
</tr>
<tr>
<td>Probability of pack breeding annually (( p_b ))</td>
<td>0.95</td>
</tr>
<tr>
<td>Birth</td>
<td>Day 60</td>
</tr>
<tr>
<td>Frequency distribution of litter size</td>
<td>1 pup: 0.2; 0.024; 3: 0.071; 4: 0.048; 5: 0.167; 6: 0.071; 7: 0.143; 8: 0.143; 9: 0.095; 10: 0.095; 11: 0.071; 12: 0.048; 13: 0.14; 14: 0.024</td>
</tr>
<tr>
<td>Age-specific annual mortality (excluding disease)</td>
<td>( m_p ), ( m_y ), ( m_a ): 0.191, 0.093, 0.192</td>
</tr>
<tr>
<td>Maximum pack size/dispersal threshold (( P_r ))</td>
<td>15</td>
</tr>
<tr>
<td>Maximum number of single sex groups in a population</td>
<td>5</td>
</tr>
<tr>
<td>Slope of the Allee effect (( \alpha ))</td>
<td>0.5</td>
</tr>
<tr>
<td>Maximum population size</td>
<td>10</td>
</tr>
<tr>
<td>Succession to dominant position (( p_s ))</td>
<td>1</td>
</tr>
<tr>
<td>Rate of pathogen exposure (( d ))</td>
<td>0.015 (low), 0.073 (observed/moderate), 0.22 (high), and 0.73 (very high)</td>
</tr>
<tr>
<td>Per-pack risk of pathogen exposure:</td>
<td>( d ), (low-risk), 3d, (high-risk)</td>
</tr>
<tr>
<td>Rate of recovery from pathogen (( r_f ))</td>
<td>0</td>
</tr>
<tr>
<td>Rate of mortality from pathogen (( \mu_f ))</td>
<td>0.2</td>
</tr>
<tr>
<td>Day of vaccination</td>
<td>Day 300</td>
</tr>
<tr>
<td>Number of wild dogs vaccinated per pack</td>
<td>0–15</td>
</tr>
<tr>
<td>Number of packs vaccinated per population</td>
<td>4</td>
</tr>
</tbody>
</table>
5. **Allee effect:** When an Allee effect is incorporated, litter size is reduced in smaller packs. For a pack containing \( N \) adult and yearling individuals, litter size is reduced by \( \kappa (p_T - N) \), rounded to the nearest integer, and not permitted to become negative. The slope, \( \kappa \), was estimated to be 0.5 based on data in Creel et al. (2004) but the effects of increasing this to 0.75 were also explored. is set to zero when the Allee effect is excluded.

6. **Succession to dominant position:** If, prior to breeding and vaccination, there is no dominant male or female, they may be replaced from within the pack by an adult of the appropriate sex with a probability of \( p_T \), which equals 1, except when examining the benefit of prioritizing vaccination of the breeding pair, when it equals 0.3.

The infectious pathogen processes are as follows:

1. **Pathogen exposure:** Neither pathogen dynamics in the reservoir, nor the population dynamics of the reservoir itself were modeled explicitly. Pathogen introduction from the reservoir into wild dog packs (containing any susceptible individuals) was modeled as a fixed yearly rate \( d_c \). This rate has been estimated as 0.073 introduction events/pack/year (once per pack every 13.5 years) in the Kenya study population (RW unpublished data) and 0.083 events/pack/year in northern Botswana (Mills et al., 1998). Analyses were performed at 0.015 (low), 0.073 (moderate), 0.22 (high), and 0.73 (very high) introductions/pack/year.

2. **Per-pack risk of pathogen exposure:** We examined vaccination strategies for populations in which all packs had the same risk of exposure and for populations where half of the packs (termed “high-risk packs”) were assumed to have a \( d_c \) three times that of the other packs (termed “low-risk packs”).

3. **Pathogen dynamics:** Once infection was present in a wild dog pack, individuals were subjected to a continuous-time, susceptible-infected-recovered/vaccinated (SIR-V) process (Anderson and May, 1991). The infection rates (both from reservoir to pack and within-pack) were used to compute stochastic orders and times of transfer of individual animals from susceptible to infectious categories according to a conventional Gillespie process (Gillespie, 1976):

\[
S_i(t) - S_i(t - 1) = I_i(t) - I_i(t + 1) = \beta S_i(t)I_i(t)
\]

\[
I_i(t) - I_i(t - 1) = R_i(t) - R_i(t + 1) = \mu I_i(t) + \gamma I_i(t)
\]

\[
S_i(t), I_i(t) \text{ and } R_i(t) \text{ are the numbers of susceptible, infectious and recovered and/or removed individuals in the } i \text{th pack at time } t.
\]

Based on mortality and seropositivity data from known or suspected CDV (Alexander and Appel, 1994; Alexander et al., 2010; van de Bildt et al., 2002) and rabies (Hofmeyr et al., 2000, 2004; Kat et al., 1995; Scheepers and Venzke, 1995) outbreaks in wild dogs, \( \beta \) was chosen so that on average outbreaks led to infection in 90% of pack members. The rate of recovery from the pathogen with life-long immunity is \( \gamma \), and the rate of mortality from the pathogen is \( \mu \). The sum of \( \gamma \) and \( \mu \) is set to 0.2, corresponding to a 5 days infectious period. When all wild dogs exposed to rabies were assumed to die, \( \gamma \) was set to 0 and \( \mu \) was set to 0.2; when 5% of dogs were assumed to recover with immunity, \( \gamma \) was set to 0.01 and \( \mu \) to 0.19. When simulating CDV dynamics \( \gamma \) was set to 0.04 or 0.1, and \( \mu \) was set to 0.16 or 0.1, corresponding to 20% or 50% of dogs recovering with immunity.

Vaccination strategies were designed to maintain a fixed number of vaccinally protected wild dogs in each of a predetermined number of packs. Vaccination took place on day 120 or 300, corresponding to dates when pups are 2-months old and could consume oral vaccine at the den, or 8-months old and just large enough to safely dart and vaccinate parenterally. Vaccine-induced immunity was assumed to persist for 2 years.

Pathogen introduction and mortality were run as continuous stochastic processes. Breeding, vaccination, succession to dominance, and pack fusion and fission were fixed-time annual events (see Figs. B.1. and B.2. in Appendix B of the Supplementary electronic material for flow diagrams of the model).

We investigated each of the following scenarios, varying the number of animals vaccinated per pack in each case:

1. Vaccination of varying numbers of packs (1–10) in a population of a fixed maximum size, initially comprising 10-packs.
2. Vaccination of all age groups when pups are either 2- or 8-months old, versus vaccinating just adults and yearlings (i.e., excluding pups), versus vaccinating just pups.
3. Prioritizing vaccination of the dominant pair.
4. Vaccination against pathogens from which 0–50% of exposed/infected individuals recover and develop life-long immunity.
5. Vaccination of a population exhibiting a component Allee effect.
6. Vaccination distributed among packs experiencing different levels of pathogen exposure, targeting only low-risk, only high-risk, or a mixture of high- and low-risk packs.

We measured the effectiveness of vaccination strategies as the numbers of packs remaining after 20 years. This measure was more informative than extinction probability because, for some parameter values, simulations predicted substantial population decline but low probabilities of extinction within the 20-years simulation. As there were stochastic processes in the model, we ran 1000 simulations for each scenario and reported the average result. Unless otherwise stated, default values were: initial population size of 10 low-risk packs, vaccination of all ages including pups aged 8-months, no prioritization of the dominant pair, no acquired immunity, and no Allee effect. Packs were initiated with 16 individuals as follows: one dominant adult male, one dominant adult female, five non-dominant males, two non-dominant females, three yearling males and four yearling females.

### 3. Results

For all simulations, as \( d_c \) – the rate of pathogen introduction – decreased, final population size increased. At the lowest \( d_c \) examined, few vaccinated animals were needed for a population to maintain its original size (Fig. 1a). Sensitivity analyses indicated that the major findings from the model analyses were qualitatively robust to plausible variation in age-specific mortality and maximum pack size, although minor quantitative differences existed; full results are detailed in Appendix A (Supplementary electronic material: Figs. A1–A12).

#### 3.1. Number of packs vaccinated

We evaluated the effect of varying the number of packs vaccinated in a 10-pack population. For all values of \( d_c \), final population size was greater as more packs were vaccinated (Fig. 1a–c); however, at the low \( d_c \), there was little difference in the number of packs remaining with 0 versus 10-packs vaccinated (Fig. 1a). In addition, for all \( d_c \), the benefit of vaccinating additional packs decreased as the number of packs vaccinated approached 100%. At both moderate and high \( d_c \), all but the highest levels of coverage failed to prevent the population from declining during the 20-years simulation (Fig. 1b and 1c). Simulations also indicated that vaccinating few individuals in many packs was more effective than vaccinating many individuals in few packs and there was little benefit to vaccinating more than 4 dogs/pack (Fig. 1b). For example, at moderate...
Dr., vaccinating two wild dogs in eight packs resulted in a final population of \( \frac{C_2}{8} \) packs after 20 years, whereas vaccinating either four or eight wild dogs in two packs resulted in a final population of \( \frac{C_2}{6} \) packs after 20 years.

3.2. Age of vaccination

We evaluated the benefit of vaccinating all age groups when pups were 2- or 8-months old, of vaccinating only pups, and of excluding pups entirely. Simulations showed that at low, medium and high \( d_r \) (Fig. 2a), fewer packs remained when only pups were vaccinated but that strategies targeting all ages, or just adults and yearlings, differed little in outcome. At very high \( d_r \), targeting all age classes when pups were 2-months old, resulted in more packs remaining than the other strategies (Fig. 2b).

3.3. Prioritizing the dominant pair

Prioritizing the dominant pair for vaccination did not substantially increase the number of packs persisting at low or moderate \( d_r \) (Fig. 3a). However, up to twice as many packs persisted at high \( d_r \) (Fig. 3b).
3.4. Recovery and immunity

Acquired immunity to the pathogen markedly improved simulated populations’ ability to maintain their size in the face of pathogen introduction, and reduced the level of vaccination coverage required to maintain population size. At equivalent vaccination levels, if even as few as 5% of infected wild dogs survived and developed immunity, substantially more packs persisted than if the pathogen caused 100% mortality (Fig. 4a and 4b). At high $d_r$ and no vaccination coverage, if $P \geq 20\%$ of infected individuals developed immunity, the final population size approached that of a pathogen-free population, and equaled it when $P \geq 50\%$ developed immunity (Fig. 4b).

3.5. Allee effect

Incorporating an Allee effect decreased the number of packs remaining (Fig. 5a and b) and reduced the average size of remaining packs (Fig. 5c and d) for a given level of vaccination. The magnitude of this effect was much smaller at low values of $d_r$ and Allee slope. At low $d_r$, there was little difference in numbers of packs remaining between simulations assuming no Allee effect and those assuming an Allee effect with a slope of 0.5. The difference in packs remaining between populations with and without Allee effects decreased, and average pack size increased, at high levels of per-pack vaccination coverage.

3.6. Risk of pathogen exposure

Since wild dog populations may contain some “high-risk packs” with a greater risk of pathogen exposure than “low-risk packs”, we
compared vaccination strategies which targeted only high-risk packs, only low-risk packs, or both high- and low-risk packs with equal probability. We found that as $d_r$ increased, the strategy that imparted the greatest relative benefit changed. At low and moderate $d_r$ (Fig. 6a) more packs remained when only high-risk packs were vaccinated, but the difference between each of the three strategies was slight. At high $d_r$, up to three times more packs remained when either a mixture of both high- and low-risk packs, or just low-risk packs were vaccinated, versus vaccinating only high-risk packs (Fig. 6b: vaccination coverage three per pack).

3.7. Diminishing returns

The number of packs persisting over the 20-years simulation increased as more dogs were vaccinated per pack but the benefits diminished with increasing coverage. We identified the number of wild dogs required to be vaccinated per pack that achieved a final population size = 0.2 packs less than the size achieved when the maximum individuals/pack (15) were vaccinated. This number increased as $d_r$ increased (Fig. 1a–c). In addition, at moderate $d_r$, this number increased (i) as more packs were included in the vaccination effort (Fig. 1b); (ii) as the percent of individuals in which the pathogen induces immunity decreased (Fig. 4a); and (iii) in the presence of an Allee effect (Fig. 5a). Prioritizing the dominant pair did not change this number (Fig. 3a). Excluding simulations in which populations experienced a successional probability of <1, in which a pathogen could induce >20% protective immunity, and which evaluated an Allee effect with a slope >0.5, this number at moderate $d_r$ varied between 3 and 8 (Figs. 1b, 4a and 5a).

4. Discussion

Our simulations suggest that, in principle, vaccination could conserve African wild dog populations’ size and persistence in the face of observed rates of pathogen exposure ($d_r$) and that certain vaccination strategies would be more effective than others. For example, the effectiveness of the strategy would depend on the number of vaccinated dogs per pack and vaccinated packs...
per population, as well as on the age and social status of the individuals vaccinated.

Our simulations showed that little or no vaccination was needed to maintain population size at low \( d_r \). Where \( d_r \) is high, reducing infection levels in the reservoir is one potential way to decrease transmission to wild dogs, thereby reducing or removing the need for wild dog vaccination. Cleaveland et al. (2003) described a domestic dog vaccination campaign in rural Tanzania that reduced the incidence of domestic dog rabies by 97%. If this reduction prompted an equivalent decline in \( d_r \) to wild dogs, it would reduce the observed \( d_r \) of 0.073/pack/year to 0.002/pack/year, a rate so low that wild dog vaccination would likely be unnecessary. Hence domestic dog vaccination against rabies could benefit wild dogs, in addition to reducing rabies incidence in humans. However, to be effective, such domestic dog vaccination would need to be maintained yearly over potentially large spatial scales.

For example, a domestic dog vaccination campaign in rural Ethiopia failed to prevent a rabies outbreak in Ethiopian wolf populations within the vaccination zone because of high yearly turnover and growth in the domestic dog population, and frequent movement of dogs into both the vaccination zone and wolf habitat (Randall et al., 2006).

Wild dog vaccination was likewise not required if the pathogen induced protective immunity in \( \geq 20\% \) of infected individuals. The protective immunity induced by exposure is equivalent or superior to that achieved through vaccination; thus provided mortality does not exceed 80%, vaccination against pathogens such as CDV and canine parvovirus would probably not be warranted. However, CDV mortality appears to vary widely among wild dogs and in some cases may exceed 80% (Alexander and Appel, 1994; Alexander et al., 1996; van de Bildt et al., 2002). Thus, CDV vaccination may be required in certain situations and may be an important consideration for very small wild dog populations. Anti-rabies antibodies have been found in unvaccinated wild dogs (Gascogne et al., 1993; Prager et al., in preparation). If these antibodies provide acquired immunity (\( \geq 5\% \)), simulations indicate that the vaccine coverage needed to protect wild dog populations from extinction is reduced, often substantially, relative to simulations in which acquired immunity is absent.

At high \( d_r \), vaccination strategies directed at the dominant pair, and including pups as young as 2-months old, provided the greatest benefit. However, such benefits were not detectable at the moderate \( d_r \) derived from empirical data. Nevertheless, as the \( d_r \) experienced by many populations is unknown, where possible prioritization of dominants and inclusion of pups would be prudent.

Population size was conserved to a greater extent as more packs were included in the vaccination effort. Our results suggest that if the number of vaccinations to distribute in the population is fixed, the most effective, though likely more logistically challenging, strategy maximizes the number of packs included in the vaccination effort rather than the number of individuals vaccinated per pack. We also found that above a certain number, the benefit of vaccinating additional animals was minimal. At moderate \( d_r \), vaccinating more than 4–5 wild dogs, per pack (mean wild dog pack size reported for several populations varies between 8 and 12 adults and yearlings (summarized in Woodroffe, 2011)), offered little added benefit, although this number was greater with the Allee effect and at \( d_r \) values greater than that found in our reference populations.

The relative risk of pathogen exposure experienced by the different packs in a population influenced the most effective vaccination strategy. For populations experiencing moderate \( d_r \), vaccinating only high-risk packs provided the greatest benefit; however this benefit was marginal. For populations experiencing high overall \( d_r \) and containing packs experiencing different levels of pathogen exposure, vaccinating both high- and low-risk packs, or just low-risk packs, provided the greatest benefit. Hence, in case \( d_r \) is greater than that estimated from our reference populations, vaccinating both high- and low-risk packs may be advisable. We conjecture that this switch in the relative benefit of the different strategies as \( d_r \) increases is due to a “source/sink” process. At low and moderate \( d_r \), vaccinated high-risk packs act as a “source” of wild dogs to unvaccinated low-risk “sink” packs that experience higher disease-related mortality. However, at very high \( d_r \), even the highest levels of vaccination coverage of high-risk packs fail to prevent deaths of pups too young to vaccinate parenterally; hence, when vaccinated adults die, they cannot be replaced. Therefore vaccinated low-risk packs, for which \( d_r \) are lower and pups are able to survive to vaccination, are needed as the “source” to the high-risk pack “sink”.

\[ \text{Pack size was conserved to a greater extent as more packs were included in the vaccination effort.} \]

![Graphs show effects in populations experiencing moderate (a) and high (b) \( d_r \).](image)
5. Conclusion

Our simulations allow tentative recommendations to improve persistence of African wild dog populations exposed to pathogens such as rabies and CDV. Highly infectious pathogens that may cause 95–100% mortality within-packs, such as rabies, can present an important risk to wild dog population numbers and persistence. For the levels of exposure to such pathogens reported from wild dog study populations, the most effective vaccination strategy would target all age classes, prioritize dominant individuals, and include at least four individuals per pack in as many packs as possible. For pathogens causing lower mortality, such as CDV, acquired immunity may allow wild dog populations to persist and maintain their size without vaccination even in the face of high exposure rates. Finally, if rabies exposure in wild dog populations could be permanently reduced by domestic dog vaccination over the very large spatial scales covered by wild dog home ranges, the need for wild dog vaccination might be reduced or eliminated, thus reducing the degree of intervention needed to prevent devastating wild dog population reductions due to infectious disease.

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Appendix A. Supplementary material

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.biocon.2011.03.025.

References


