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# Raccoon contact networks predict seasonal susceptibility to rabies outbreaks and limitations of vaccination

**Jennifer J. H. Reynolds[1]*, Ben T. Hirsch[2,3], Stanley D. Gehrt[4] and Meggan E. Craft[1]**

[1]*Department of Veterinary Population Medicine, University of Minnesota, St Paul, MN, USA;* [2]*Department of Wildlife Ecology and Conservation, University of Florida, Gainesville, FL, USA;* [3]*Smithsonian Tropical Research Institute, Apartado Postal 0843-03092, Panamá, República de Panamá; and* [4]*School of Environment and Natural Resources, The Ohio State University, Columbus, OH, USA*

## Summary

**1.** Infectious disease transmission often depends on the contact structure of host populations. Although it is often challenging to capture the contact structure in wild animals, new technology has enabled biologists to obtain detailed temporal information on wildlife social contacts. In this study, we investigated the effects of raccoon contact patterns on rabies spread using network modelling.

**2.** Raccoons (*Procyon lotor*) play an important role in the maintenance of rabies in the United States. It is crucial to understand how contact patterns influence the spread of rabies in raccoon populations in order to design effective control measures and to prevent transmission to human populations and other animals.

**3.** We constructed a dynamic system of contact networks based on empirical data from proximity logging collars on a wild suburban raccoon population and then simulated rabies spread across these networks. Our contact networks incorporated the number and duration of raccoon interactions. We included differences in contacts according to sex and season, and both short-term acquaintances and long-term associations. Raccoons may display different behaviours when infectious, including aggression (furious behaviour) and impaired mobility (dumb behaviour); the network model was used to assess the impact of potential behavioural changes in rabid raccoons. We also tested the effectiveness of different vaccination coverage levels.

**4.** Our results demonstrate that when rabies enters a suburban raccoon population, the likelihood of a disease outbreak affecting the majority of the population is high. Both the magnitude of rabies outbreaks and the speed of rabies spread depend strongly on the time of year that rabies is introduced into the population. When there is a combination of dumb and furious behaviours in the rabid raccoon population, there are similar outbreak sizes and speed of spread to when there are no behavioural changes due to rabies infection.

**5.** By incorporating detailed data describing the variation in raccoon contact rates into a network modelling approach, we were able to show that suburban raccoon populations are highly susceptible to rabies outbreaks, that the risk of large outbreaks varies seasonally and that current vaccination target levels may be inadequate to prevent the spread of rabies within these populations. Our findings provide new insights into rabies dynamics in raccoon populations and have important implications for disease control.

**Key-words:** contact network, epidemiological modelling, host–pathogen interactions, infectious disease management, network modelling, proximity logging collar, rabies, raccoon, transmission, wildlife disease

## Introduction

Understanding the mechanisms underpinning the spread of infectious diseases in populations is critical for disease control (Anderson & May 1991; Grenfell & Dobson 1995; Lloyd-Smith *et al.* 2005; Keeling & Rohani 2008). The contact structure of a population can significantly affect infectious disease transmission, and therefore, knowledge of host contact patterns can be crucial for predicting and controlling disease outbreaks (Keeling 1999; Newman

*Correspondence author. E-mail: j.j.h.reynolds@dunelm.org.uk

2002; Keeling & Eames 2005; Craft 2015). While many traditional epidemiological models assume that the probability of contact is equal for every pair of individuals in the population (Anderson & May 1991), contact patterns for humans and animals are often heterogeneous.

Network theory provides a set of tools for capturing and analysing heterogeneity in contact structure. Contacts between individuals that may lead to pathogen transmission can be represented using networks, where each node represents an individual, and edges between nodes represent interactions that allow for potential transmission. Unlike disease models that assume a population is well mixed, network models can be used to represent contact heterogeneity. Network modelling is therefore a powerful method to understand and predict the dynamics of infectious disease and has been used extensively in the study of human diseases such as AIDS (Anderson, Gupta & Ng 1990), SARS (Meyers *et al.* 2005) and influenza (Kenah *et al.* 2011).

Network theory has more recently been applied to wildlife populations to describe and analyse social interactions; this is often called social network analysis (Croft, James & Krause 2008; Wey *et al.* 2008). Network analysis can be used to study sociality in wildlife populations and thus offer insight into animal behaviour (Krause, Croft & James 2007; Wey *et al.* 2008). Networks have also been used to explore the role of contact patterns in the transmission of pathogens for a range of wildlife species (Drewe 2010; Rushmore *et al.* 2013; VanderWaal *et al.* 2013). Contact networks have been empirically constructed using behavioural data on social contacts (e.g. Hamede *et al.* 2009) and also using data on possible exposure to a common environmental source (e.g. Godfrey *et al.* 2009), both of which have the potential to create transmission opportunities. In addition, networks based on actual transmission events have also been constructed for some wildlife species such as the giraffe (VanderWaal *et al.* 2014) and the wild mouse lemur (Zohdy *et al.* 2012). However, determining the contact structure of wildlife populations can be challenging (Craft & Caillaud 2011). For example, observing all individuals in a population through time is problematic, making it difficult to construct a complete contact network. The use of new wildlife monitoring technologies, such as proximity logging collars (which record when individuals are in close proximity to each other), can help overcome this difficulty, although logistical issues or high costs often limit the number of individuals that can be monitored.

The mathematical simulation of pathogen spread across empirically derived wildlife networks has proven to be a useful method to test hypotheses about pathogen transmission, and the results can have practical applications for infectious disease management (e.g. Cross *et al.* 2004; Craft *et al.* 2009, 2011; Hamede *et al.* 2012; Rushmore *et al.* 2014). Simulating pathogen spread involves modelling the progress of a pathogen across a contact network through time. Because contact networks can change through time and static networks may fail to properly capture the system and lead to incorrect inferences (Blonder *et al.* 2012), it is important to model appropriate temporal changes by using dynamic networks, when appropriate (Chen *et al.* 2014). For example, Hamede *et al.* (2012) change their Tasmanian devil contact networks seasonally, to reflect seasonal changes in devil contacts. This changing of the structure of networks is sometimes known as 'rewiring' (Watts & Strogatz 1998; Blonder *et al.* 2012).

In this study, we construct a wildlife contact network using proximity collar data from a wild raccoon (*Procyon lotor*) population. Raccoons are a common species across North America; their population densities often increase in conjunction with the availability of anthropogenic food sources found in urban and suburban areas (Hoffmann & Gottschang 1977; Prange, Gehrt & Wiggers 2003). Though previously thought to be solitary (Ewer 1973; Barash 1974), there is now evidence that raccoons have social networks that are highly connected (Hirsch *et al.* 2013a) and involve sex-specific social behaviours (Prange, Gehrt & Hauver 2011; Hirsch *et al.* 2013b). The length and number of contacts depend on the sex of the raccoon (Prange, Gehrt & Hauver 2011; Hirsch *et al.* 2013b). The degree to which raccoons associate with conspecifics varies, with some pairs of raccoons only forming short-term acquaintances and other pairs forming long-term social associations (Prange, Gehrt & Hauver 2011). In addition, raccoon contact patterns differ during the breeding and non-breeding seasons (Prange, Gehrt & Hauver 2011; Hirsch *et al.* 2013a,b).

Raccoons are host to a wide range of transmissible pathogens, including canine distemper virus (Roscoe 1993) and zoonotic diseases such as rabies and leptospirosis (Junge *et al.* 2007). Here, we focus on rabies, a directly transmitted viral infection of the central nervous system (Krebs, Wilson & Childs 1995). Rabies is a significant cause of concern to public health (Rupprecht, Hanlon & Hemachudha 2002; Blanton *et al.* 2011; Dyer *et al.* 2013). Raccoons are the most frequently reported rabid wildlife species in the United States, with almost 2000 cases of rabid raccoons reported during 2012 (Dyer *et al.* 2013). The specific rabies virus affecting raccoons is termed the 'raccoon rabies virus (RRV) variant'; this is the pathogen we model in this manuscript. Raccoons play a dominant role in the maintenance of rabies in the United States, acting as an important reservoir (Krebs, Wilson & Childs 1995; Fu 1997). Rabies is currently endemic in raccoons across the eastern United States and is spreading westward (Dyer *et al.* 2013). The exceptionally high raccoon population densities in urban and suburban areas (Gehrt 2003) increase the threat of human rabies exposure (Kappus *et al.* 1970; Jenkins & Winkler 1987). Oral raccoon vaccine (ORV) baiting has proved to be a successful tool to control the geographic spread of rabies in some areas (Robbins *et al.* 1998; Rosatte *et al.* 2009), yet rabies remains endemic in raccoon populations within ORV baiting zones (Slate *et al.* 2009).

Once a raccoon becomes infected with rabies, the virus spreads through the nervous system, brain and salivary glands. The incubation period ranges between *c*. 3 and 19 weeks (Tinline, Rosatte & MacInnes 2002); during this period, raccoons show no clinical signs. After this, a raccoon is infectious for a period of *c*. 1 week (Hanlon, Niezgoda & Rupprecht 2007). A range of different behaviours have been observed in infectious (rabid) raccoons, including 'furious' behaviour (characterized by boldness and aggression towards other animals) and 'dumb' behaviour (characterized by ataxia/impaired mobility or paralysis) (Hubbard 1985; Jenkins & Winkler 1987; Winkler & Jenkins 1991; Rosatte *et al*. 2006). The disease is almost always fatal to raccoons. The main route of rabies transmission between raccoons is biting (Rupprecht, Hanlon & Hemachudha 2002), although mutual grooming, and the associated transfer of saliva, is a likely alternative route (Dyer *et al*. 2013). Because direct contact between raccoons is required in order for transmission to take place, the duration of direct contacts is likely to be an important factor in transmission.

In this study, we use mathematical techniques to simulate RRV spread across empirically derived raccoon contact networks. We produce a dynamic system of networks representing raccoon contacts through time based on detailed data on raccoon social proximity. The network model is designed to capture the number and duration of raccoon interactions. We rewire all networks seasonally and incorporate differences in contacts according to raccoon sex. Our model is novel in its inclusion of both short-term acquaintances and long-term associations, which we term 'inconsistent' and 'consistent contacts', respectively; this is an extra level of temporal detail, included to better represent facets of raccoon social structure. We use our model to investigate rabies dynamics when the virus is introduced at different times of the year. We then (i) assess the implications of behavioural changes in rabid raccoons and (ii) test the effectiveness of vaccination as a management strategy to prevent the spread of rabies.

## Materials and methods

### CONSTRUCTING NETWORKS

Contact networks were constructed based on data from proximity logging collars (SirTrack Ltd., Havelock North, New Zealand; Prange, Gehrt & Hauver 2011) placed on wild raccoons within a 20-ha area of Ned Brown Forest Preserve in suburban Cook County, Illinois (Prange, Gehrt & Wiggers 2003), from July 2004 to July 2005. The collars recorded the amount of time adult raccoons spent in close proximity (within 1–1·5 m) and the identity of the raccoons. Any contacts <1 s in duration were excluded (Prange, Gehrt & Hauver 2011; Drewe *et al*. 2012). Raccoons were aged according to tooth wear (Grau, Sanderson & Rogers 1970), and all raccoons >12 months of age were fitted with radio-collars. A total of 42 adult raccoons (20 males and 22 females) were collared, and these represented close to 100% of all adult raccoons in the core 20-ha area (Prange, Gehrt & Hauver 2011).

The contact data were formatted into 52 adjacency matrices, each comprising the time each raccoon pair (or dyad) spent in close proximity during each week of the year. The matrices for the breeding and non-breeding seasons were analysed separately. The breeding season corresponded to the 16 matrices (or weeks) from December 11th to April 1st; all other matrices corresponded to the non-breeding season. Certain raccoon dyads were recorded in close proximity consistently throughout a season, while contacts between other raccoon dyads were less consistent. Because consistent and inconsistent contacts can have different effects on disease spread (e.g. Pinkerton & Abramson 1993), we examined the distribution of the number of weeks each raccoon pair made contact over a season and defined 'consistent contacts' to be those raccoon dyads that fell within the upper quartile of the distribution, while all other contacts were classified as 'inconsistent contacts'.

Separate weekly networks of consistent and inconsistent contacts were produced, for each season. Networks consisted of nodes (individual raccoons) and undirected edges, which represented the time each pair of raccoons spent in close proximity per week. We represent the number of nodes by $n$, and each node was assigned a sex (male or female). The following summary measures were calculated for each network:

1. *Mean degree*: The mean number of edges per node, or the mean number of unique individuals contacted per week.
2. *Density*: The number of edges in the network divided by the total number of all possible edges. The total number of all possible edges is given by $n(n–1)/2$.
3. *Clustering coefficient*: A measure of how clustered the network is, defined here as the number of triangles (any set $\{(i, j), (j, k), (k, i)\}$ of three edges) divided by the number of open triples (a pair of edges $\{(i, j), (j, k)\}$). If this ratio is conserved, then the definition for the standard clustering coefficient given by Opsahl & Panzarasa (2009) is also conserved. This term is sometimes called transitivity.
4. *Edge sex ratio*: The ratio of the number of edges between female raccoons, to the number between males and females, to the number between males (female–female: male–female: male–male contacts).

Due to mortality and proximity collar failure over the course of the fieldwork, proximity data from certain individuals were not available throughout the entire study period (Prange, Gehrt & Hauver 2011; Hirsch *et al*. 2013a,b). Thus, for each season, we based our analyses on the 15 individuals that were consistently present across the weekly matrices. The 15 raccoons were representative of the raccoon population; contacts of individuals with functioning collars did not differ significantly from contacts of individuals with malfunctioning collars (Prange, Gehrt & Hauver 2011; Hirsch *et al*. 2013a; B. T. Hirsch, unpublished data). To investigate pathogen spread in suburban raccoons with a biologically meaningful population size, the initial networks of 15 raccoons were used to create larger networks of 90 raccoons. 'Scaling up' was carried out in two steps to ensure the larger networks had the correct network properties. First, we created networks of 45 raccoons, approximately the number of individuals in the core 20-ha field site (Prange, Gehrt & Hauver 2011). In this step, network densities and clustering coefficients were kept the same as in the networks with 15 raccoons, but we increased the number of nodes to 45; this yielded a corresponding increase in the mean degree. Secondly, we scaled up from 45 to 90 individuals. In this step, we kept the same mean degrees and clustering

 

coefficients as the 45 raccoon networks. The edge sex ratios were conserved in both steps. Exponential random graph models (ergms) were used to create the 90 raccoon networks with the correct summary measures. Specifically, we used the R (R Core Team 2013) package 'statnet' (Handcock *et al.* 2003), which includes ergm tools, and employed the method of simulated annealing (via the function 'san').

Edges in all networks were weighted by the total time that each raccoon pair spent in close proximity. We fitted statistical distributions to the time raccoon dyads spent in proximity each week, separately for each season, for consistent and inconsistent contacts, and for male–male, male–female and female–female contacts. We tested the fit of a broad range of candidate distributions (normal, Poisson, log-normal, exponential, geometric, logistic, negative binomial) by minimizing the negative log likelihood. In each case, the best statistical fit was the log-normal distribution. There was no evidence of correlation between degree and duration of contact ($P$ value = 0·34). For each contact in the networks, a time was assigned by sampling from the log-normal distribution with the mean and standard deviation corresponding to the season, type of contact and sex of the raccoons (Table S1, Supporting information).

## SIMULATING PATHOGEN SPREAD

Our weekly consistent and inconsistent 90 raccoon networks for the breeding and non-breeding seasons were used to form a dynamic representation of the contacts made between raccoons over a year. The networks of consistent contacts were kept the same throughout a season. In contrast, the networks of inconsistent contacts were rewired each week; we calculated the average measures of the weekly inconsistent contact networks for each season, and each week, a new network was generated that conformed to these average measures. The consistent and inconsistent networks were amalgamated at each weekly time step to give the total contacts (i.e. the total number of raccoons that each individual made contact with each week, and the duration of time each pair was in contact over the week). The time interval of a week was motivated by the infectious period of rabies. Any coarser time division would overestimate the contacts made during the infectious period and thus potentially overestimate rabies transmission.

Our system of networks was used to simulate rabies spread through the raccoon population. We stochastically modelled rabies transmission, starting from a randomly assigned index case, that is the first infectious raccoon. An *SEIR* framework was used, where $S$ represents the number of susceptible individuals, $E$ the number exposed (infected but not yet infectious), $I$ the number of infectious and $R$ the number removed. (Here, removed individuals are dead, as rabies is a fatal disease.) A susceptible raccoon can become infected with rabies from contact with an infectious raccoon. We define $\beta(t)$ in our model as the probability that transmission occurs given an edge between a susceptible and infectious raccoon in the weekly network. Thus, $\beta(t)$ varies between 0 (when transmission never occurs) and 1 (when transmission always occurs). As $\beta(t)$ is likely positively correlated with the duration of contact between an infectious and susceptible raccoon, but has not been quantified, we take it to be an increasing function of the total duration of contact between a raccoon pair in a week, which we denote by $t$. (Note that $t$ is the weight of the edge between the raccoon pair in that week's network.) Specifically,

$$\beta(t) = \frac{t}{a + t}$$

for $t > 0$, where parameter $a$ sets the 'steepness' of the curve. Figure 1 illustrates this function and how it varies with parameter $a$. Because biting is believed to be the primary method of transmission, raccoons may need to spend very little time in contact in order to spread rabies. This motivated our choice of a sigmoidal, saturating function for transmission. We experimented across a range of biologically reasonable values of parameter $a$, between 0 and 1; this ensures a steep initial increase in the probability of transmission with time.

Simulations were coded and run with R (R Core Team 2013). At each weekly time step, three events may occur:

1. Susceptible individuals may become exposed if there is an edge to an infectious individual and if transmission occurs. Transmission occurs according to a Bernoulli trial (or binomial trial) where the probability of transmission is $\beta(t)$.
2. An exposed individual may become infectious, according to a Bernoulli trial where the probability of becoming infectious is $\sigma$ (see Table 1). In order to exclude biologically unrealistic incubation periods of $<3$ weeks (Niezgoda, Hanlon & Rupprecht 2002), we included a stipulation that exposed raccoons must not become infectious before they have been in the exposed class for 3 weeks. Note that the stochastic nature of our model imposes a distribution of incubation periods that is right-skewed with a long tail. Hence, there is a possibility of some raccoons having a very long incubation period, which has been reported in the literature (Bigler, Mclean & Trevino 1973; Tinline, Rosatte & MacInnes 2002).
3. Any susceptible or exposed individual may die naturally (i.e. not from rabies), according to the death rate given in Table 1.

A fourth event always occurs at each weekly time step:

4. Infectious individuals die (as the infectious period $1/\gamma$ is 1 week; see Table 1).

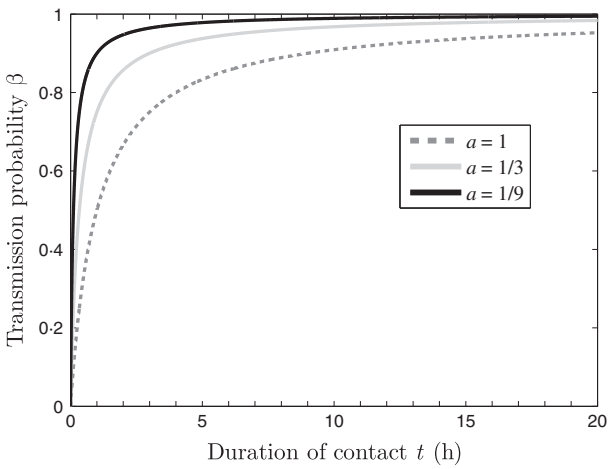


**Fig. 1.** The transmission probability $\beta$ is dependent on the total duration of contact between a raccoon pair in a week ($t$). We define parameter $a$ as a 'steepness parameter'; as $a$ increases, the curve becomes less steep. Three possible functions are plotted, each with a different value of $a$ as shown. These values are chosen so that probability $\beta$ at $t = 1$ h is 0·5 (yielding $a = 1$), 0·75 ($a = 1/3$) and 0·9 ($a = 1/9$).

**Table 1.** Epidemiological and demographic parameters used in the raccoon rabies model. When possible, demographic model parameters were from a long-term study of wild raccoons in Ned Brown Forest Preserve

| Parameter | Value | Year(s) | Citation(s) |
|---|---|---|---|
| Raccoon rabies | | | |
| Incubation period ($1/\sigma$) | Average: 5·5 weeks<br>Minimum: 3 weeks | | Tinline, Rosatte & MacInnes (2002) |
| Infectious period ($1/\gamma$) | 1 week | | Hanlon, Niezgoda & Rupprecht (2007) |
| 'Steepness parameter' of the transmission probability ($a$) | Varied between 0 and 1 | | |
| Raccoon demographics | | | |
| Average death rate for adult raccoons, in the absence of rabies | 25% year$^{-1}$ | 1995–2001 | Prange, Gehrt & Wiggers (2003), Gehrt & Prange (2007), S. D. Gehrt, unpublished data |
| Gestation length | 9 weeks | | Sanderson & Nalbandov (1973) |
| % of adult females to have a litter per year | 92% | 1995–2002 | Prange, Gehrt & Wiggers (2003), S. D. Gehrt, unpublished data |
| Average litter size | 3·6 | 1995–2002 | S. D. Gehrt, unpublished data taken from autopsied road killed females |
| Date of juvenile 'independence' | 10 months | 1990–1992 | Gehrt & Fritzell (1998b) (Texas) |
| Juvenile survivorship (0–10 months) | 26% | 1995–2002 | S. D. Gehrt, unpublished data on age ratios of raccoon road kills |
| Peak mating | 14 February–7 March | 2005 | Hauver *et al.* (2010) |

In addition, the entry of new raccoons was included in the model, according to the demographic parameters given in Table 1. Raccoons younger than 10 months are dependent on their mothers, and because they were assumed to have the same contacts as their mother, they were not explicitly included in the model. New 10-month-old raccoons entered the simulated population between December 11th and April 1st, according to the birth and survivorship values given in Table 1. We assumed that *c*. 50% of births are a result of the 3-week peak mating period. Because of the frequent contacts and likely fluid transfer between mother and offspring, 10-month-old raccoons were assigned the same disease status (susceptible, exposed, infectious or removed) as their mother.

Initial conditions were $(S, E, I, R) = (89, 0, 1, 0)$; all animals were susceptible except one infectious individual, which was randomly selected. We ran simulations for 2 years, by which time all outbreaks had ceased. To investigate the effects of seasonal contact patterns, we introduced rabies into the raccoon population at three different times of the year: at the start of the breeding season, at the start of the non-breeding season and halfway through the non-breeding season. These three time points are roughly equally spaced. Simulations were repeated 500 times for each value of $a$ and for each of the three times of disease introduction. We calculated the final outbreak size for each simulation (defined as the total number of raccoons that died from rabies) and the time taken for rabies to spread (defined as the time taken for half of the population to become infected).

In addition to simulating across a range of $a$, we wanted to choose the most likely value of $a$ for raccoon rabies. Solving for the basic reproductive rate, $R_0$, for raccoon rabies for a simple deterministic model assuming frequency dependence and homogeneous mixing yields $R_0 = 0{\cdot}97\ \beta'$, where $\beta'$ is the transmission rate. For our stochastic network model, the $R_0$ value will be less than this (Newman 2002). A genetic analysis of a raccoon rabies outbreak estimated the reproductive rate to range from 1·02 to 1·16 (Biek *et al.* 2007). To ensure maximum agreement with this estimate, as a baseline for subsequent simulations, we take $\beta$ to be its largest value and thus parameter $a$ to be the smallest value from the three main values tested (1/9).

### SENSITIVITY ANALYSIS: BEHAVIOURAL CHANGES IN RABID RACCOONS

When an animal becomes infectious, normal social behaviour and interactions with other animals may change. In studies of rabid raccoons in regions of the United States and Canada, a range of different behaviours have been observed (Hubbard 1985; Jenkins & Winkler 1987; Winkler & Jenkins 1991; Rosatte *et al.* 2006). The behaviours broadly fall into two classes: (i) 'dumb' behaviour, characterized by ataxia/impaired mobility or paralysis, and (ii) 'furious' behaviour, characterized by aggressive behaviour towards conspecifics and other species. In addition, some rabid raccoons display no abnormal behaviours (Rosatte *et al.* 2006). In line with these observations, we used our model to explore the effects of behavioural change on disease dynamics. Because the contact data used to build our networks were collected on non-rabid raccoons, we considered two hypothetical types of changes.

First, we changed the probability of rabies transmission due to behavioural changes from rabies infection. More aggressive behaviour in a furious rabid raccoon, for example biting and fighting, may lead to an increased probability of transmission between a rabid and a susceptible raccoon (corresponding in the model to a decrease in the parameter $a$). Similarly, if a rabid raccoon displays dumb behaviour, the probability of transmission may decrease (corresponding to an increase in the parameter $a$). To capture these effects, we ran 500 simulations where all infectious raccoons were furious, using a value of $a = 1/18$, and 500 simulations where all infectious raccoons were dumb, using $a = 2/9$. In addition, we ran 500 simulations for a more realistic scenario of a combination of behaviours; specifically, a third of infectious raccoons exhibited furious behaviour, a third dumb behaviour and a third were normal (using $a = 1/9$), which corresponds to that reported in the literature (Winkler & Jenkins 1991; Rosatte *et al.* 2006). We report the results for when rabies was introduced at the start of the breeding season; the same trends occur when the virus is introduced at the other times of the year.

Secondly, rabies could cause behavioural changes that could change the number of contacts made by rabid raccoons (see Appendix S1 for details on the methods).

### TESTING CONTROL STRATEGIES

The aim of current vaccination programmes is to achieve rabies immunity for 60–70% of raccoons in an area (Rosatte *et al.* 2009), with the purpose of preventing rabies spread. We used our model to test the effectiveness of vaccination when 65% of raccoons are immune from ingesting oral bait vaccine. For each of 500 simulations, we selected at random which raccoons became immune and assumed vaccination occurs prior to the introduction of rabies into our raccoon population. The value of parameter $a$ was fixed as 1/9. Additionally, we experimented with changing the vaccination level from 65% to a range of other levels (45%, 55%, 75% and 85%). To test the likelihood of perfect vaccination success, we monitored the proportion of simulations for which rabies fails to spread beyond the index infectious case. We also recorded the proportion of simulations where rabies spreads to more than 10% of the raccoon population. This latter proportion is used as a measure of the likelihood that rabies is not restricted to small outbreaks, which would increase the risk to public health.

## Results

### RACCOON CONTACT NETWORKS

Contacts in the non-breeding season are generally shorter in duration than in the breeding season, and male–male contacts usually last the longest (Table S1). In the breeding season, raccoons typically have more inconsistent contacts than consistent ones (as reflected by the mean degree), whereas the reverse is true in the non-breeding season (Table S2); this pattern is likely due to mating behaviour. The consistent network in the non-breeding season is the most clustered and the inconsistent network in the non-breeding season the least clustered (Table S2). The raccoon networks reflected the highly seasonal contact structure of the raccoon population and the sex-specific differences in the duration and number of contacts made.

### RACCOON RABIES SPREAD

Final outbreak sizes for raccoon rabies are bimodally distributed; rabies either fails to spread, or if it does spread, typically most of the population becomes infected (Fig. 2). Final outbreak sizes tend to be greater when rabies is introduced at the start of the breeding season (mid-December) or halfway through the non-breeding season (mid-August), compared to at the start of the non-breeding season (early April). We can use the proportion of simulations where the outbreak size is 80 raccoons or greater (which we define as ω) as one measure for comparison purposes. The ω values indicate that there is a greater likelihood of very large outbreaks when rabies is introduced halfway through the non-breeding season (mid-August), for all three $a$ values (Fig. 2). The magnitudes of outbreaks tend to increase as parameter $a$ decreases (as the transmission probability increases).

Season also affects the speed of rabies spread. Rabies spreads faster when introduced at the start of the breeding season than when introduced at the start of, or halfway through, the non-breeding season (Fig. 3). This finding is consistent throughout the range of values of parameter $a$; however, the rate of spread in the non-breeding season is more sensitive to changes in $a$ than in the breeding season.

### SENSITIVITY ANALYSIS: BEHAVIOURAL CHANGES IN RABID RACCOONS

When all rabid raccoons exhibit dumb behaviour, the final outbreak size tends to decrease, and the probability of rabies failing to spread slightly increases (Fig. S1a compared to S1d). When infectious raccoons are all furious, the outbreak size tends to increase (Fig. S1b,d). When there is a combination of behaviours in the population, there is little qualitative change in the distribution of outbreak sizes compared to when there are no behavioural changes (Fig. S1c,d).

The speed of rabies spread was also affected by changes in the probability of rabies transmission due to behavioural change, although not dramatically. The mean time taken for half of the population of raccoons to become infected is 9·6 weeks when all infectious raccoons are dumb, and 8·0 weeks when they all are furious. For the combination of behaviours, the mean time is 8·8 weeks. When there are no behavioural changes, the mean time is 8·6 weeks.

When we changed the number of contacts made by rabid raccoons, our results had the same trends but were more dramatic for the experiments where all infectious raccoons became either dumb or furious (see Appendix S1 and Fig. S2 for results).

### TESTING CONTROL STRATEGIES

When 65% of the raccoons are vaccinated, there is a reduction in rabies incidence (Fig. 4). When compared to a non-vaccinated population, the chance of rabies failing to spread beyond the index case increases from 3–7% to 18–21%, and the chance of rabies spreading to more than 10% of the population decreases from 93–97% to 62–77%. Vaccination is less effective at reducing rabies spread when rabies is introduced at the start of the breeding season. In this case, the chance of rabies failing to spread beyond the index case is 18% (compared to 18% and 21% for introduction at the start of and halfway through the non-breeding season, respectively), and the chance of rabies spreading to >10% of the population is 77% (compared to 62% and 64%). Also, there is less of a change between the vaccinated and non-vaccinated populations when rabies is introduced at the start of the breeding season.

 

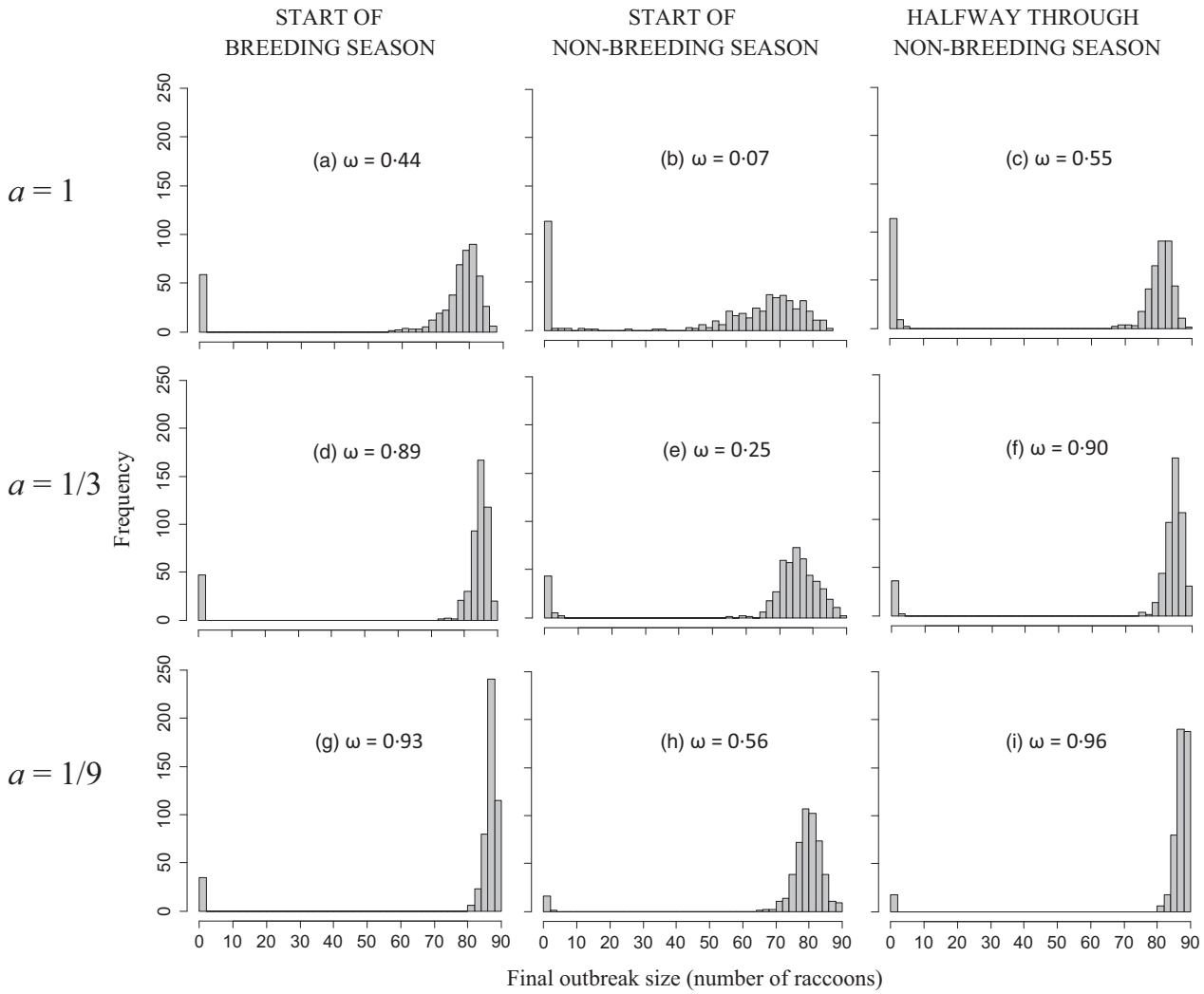


**Fig. 2.** Frequency distributions of final outbreak size for three values of parameter *a*, for rabies introduction at the start of the breeding season (panels a, d, g) and non-breeding season (b, e, h), and halfway through the non-breeding season (c, f, i). Five hundred simulations are performed for each panel, and the raccoon population comprises 90 individuals. As *a* increases, the transmission probability (β) decreases. The ω values are the proportion of simulations where the final outbreak consists of 80 or more raccoons; these are given as one way of comparing panels.

As vaccination coverage increases, there is a nonlinear increase in the likelihood that rabies does not spread from the index case, and a nonlinear decrease in the likelihood that rabies spreads to >10% of the raccoon population (Fig. 5). The slopes of these likelihoods get steeper as the vaccination level increases, so for higher vaccination coverage, a smaller change in coverage causes a larger change in the vaccination effectiveness. For the range of vaccination coverage simulated, all perform at least slightly better than no vaccination (where rabies does not spread in 7% of simulations and spreads to >10% of the population in 93% of simulations).

## Discussion

In this study, we formulate a dynamic network model using empirically derived raccoon contact data to simulate pathogen spread through a raccoon population. As reported in previous studies, raccoon social interactions change over time, especially with respect to sex-specific contacts (Gehrt & Fritzell 1998a; Hirsch *et al*. 2013b). Using RRV variant as a case study, we demonstrate that outbreak sizes and the speed of pathogen spread depend on the time of pathogen introduction. Outbreaks tend to be larger when rabies is introduced halfway through the non-breeding season, while rabies spreads faster when rabies is introduced at the start of the breeding season. These findings have important ecological and health implications for raccoons, humans and other animals.

Stochasticity can have dramatic implications for whether an infectious disease spreads through a population or not (Lloyd-Smith *et al*. 2005). In our model, both the stochastic nature of rabies infection and the underlying social structure of the raccoons mean that rabies final

 

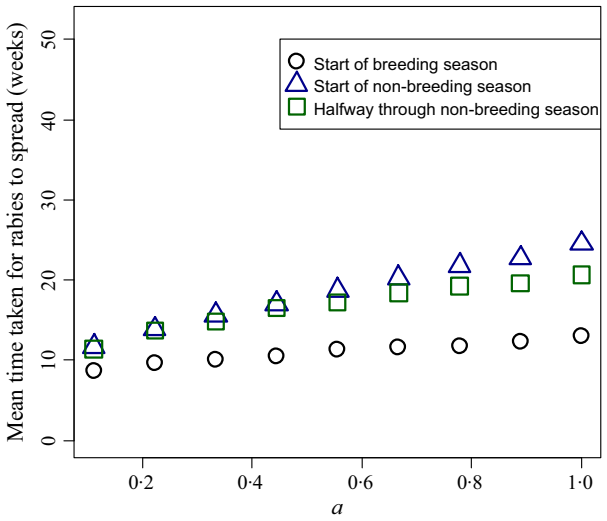


**Fig. 3.** Mean time taken for rabies to spread, for a range of values of parameter *a*, when an infectious raccoon is introduced into the population at three different times of a year. We define the time taken for rabies to spread as the time for half of the raccoon population to become infected with rabies. (We consider only those simulations for which at least half the population becomes infected.)

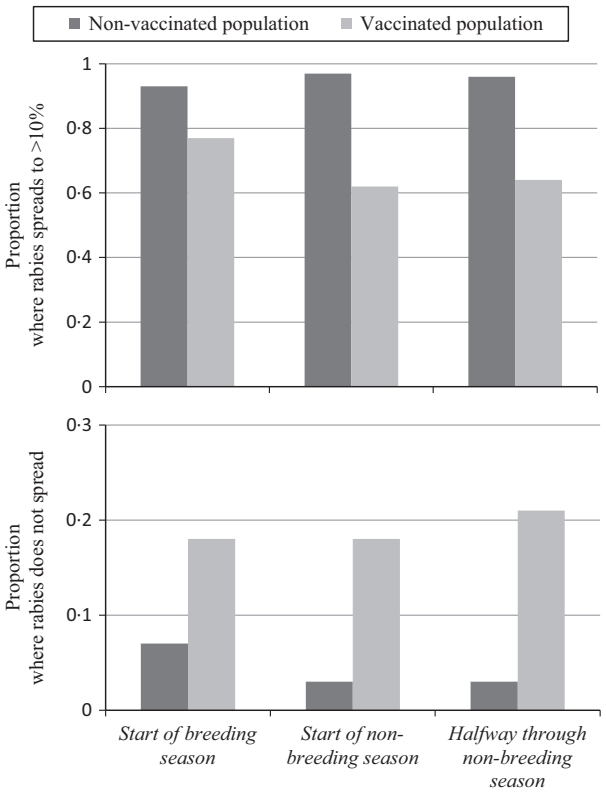


**Fig. 4.** The effects of vaccinating 65% of the raccoon population prior to the introduction of rabies. Corresponding results for the non-vaccinated population are shown for comparison. The upper panel shows the proportion of 500 simulations where rabies spreads to more than 10% of the raccoon population. The lower panel shows the proportion of 500 simulations for which rabies fails to spread beyond the index infectious case, that is no other raccoons become infected.

outbreak sizes display bimodality. This bimodal shape is consistent with other stochastic modelling studies of rabies in other wildlife species, for example Ethiopian wolves (Craft, Beyer & Haydon 2013). Of particular note is our observation that rabies spreads to a large proportion of the population in the majority of our simulations; this ease of spread highlights the importance of preventing rabies from being introduced into new susceptible raccoon populations in the first place. Stochasticity also has an impact on our vaccination results, leading to a wide variation across simulations in the level of vaccination that is effective.

The current method for preventing the spread of raccoon rabies is an extensive ORV program (Slate *et al.* 2005). Our model indicates that when vaccine coverage increases, there is a nonlinear reduction in the spread of rabies in our model; however, feasible vaccination levels do not eliminate the probability of an outbreak. Our simulations indicate that current vaccination target levels of 60–70% immunity may be inadequate to prevent the spread of rabies within our suburban raccoon population. For example, when 65% of raccoons are vaccinated, the probability of a large outbreak remains around 60–80% and depends on the time of rabies introduction. Vaccination levels in our model need to reach ~85% before an effective transmission barrier is reached. It remains uncertain whether the discrepancy in target levels arises from the use of surrogate species (e.g. European fox) to parameterize models for raccoon rabies vaccine programs (Rupprecht *et al.* 2008), or the higher raccoon density in our study population compared to rural areas (where vaccination programmes are often focused). Relevant empirical data come from Rosatte *et al.* (2009), who found that vaccine levels of >71% were needed to control rabies spread in urban and suburban Ontario. In addition, Robbins *et al.* (1998) reported that a vaccination rate of *c.* 63% was sufficient to halt the spread of rabies in suburban Massachusetts.

Our findings have important management implications, as current vaccine baiting strategies may be inadequate to control rabies in moderately high and high-density raccoon populations, typical of urban landscapes. For instance, given our results, it is not surprising that where baiting has been focused in Ohio across the western front of the raccoon rabies epizootic, the Cleveland metropolitan area has been the most problematic for rabies elimination (Slate *et al.* 2009). Additionally, raccoon rabies has not been eradicated in areas of eastern United States despite extensive ORV programmes over several years (Slate *et al.* 2009). While our results on the effects of vaccination may differ compared to areas with lower raccoon population densities, we believe that urban and suburban areas may be particularly important for rabies transmission. Furthermore, wildlife officials may find it harder to

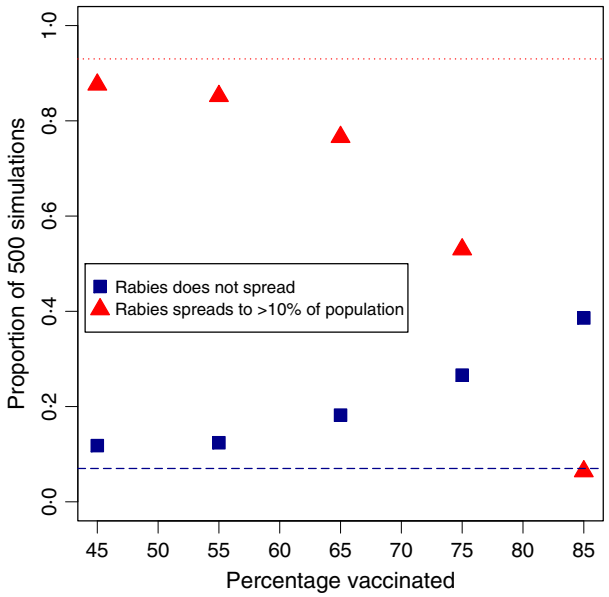


**Fig. 5.** The effects of varying vaccination coverage, when rabies is introduced at the start of the breeding season and vaccination has already been implemented. This figure shows the effects of different vaccination levels on the proportion of simulations where rabies does not spread beyond the index case and the proportion of simulations where rabies spreads to more than 10% of the raccoon population. The blue dashed line indicates the proportion of simulations where rabies does not spread for an unvaccinated population, while the red dotted line indicates the proportion of simulations where rabies spreads to more than 10% of the population for an unvaccinated population.

reach target vaccination levels in these urban and suburban areas. Unlike rural forested areas where baits can be dropped aerially, ORV baits in urban and suburban areas often must be distributed manually. Once the baits are placed, other mammals (such as coyotes, skunks, opossums and possibly domestic dogs and cats) may consume them. Given the close proximity to humans, and the high susceptibility to rabies, urban and suburban raccoon populations are especially important foci for controlling raccoon rabies, contributing to efforts for global rabies eradication (Lankester *et al.* 2014).

Seasonal contact patterns strongly influence rabies spread in our system. The contacts made between raccoons in the breeding season facilitate the more rapid spread of rabies through the population; this is consistent with observations made by Bigler, Mclean & Trevino (1973). Rabies spreads faster when introduced at the start of the breeding season than if introduced halfway through the non-breeding season, and as the rewiring of the consistent networks happens after roughly the same number of weeks in both cases, this rewiring cannot be the sole reason for the faster spread. However, the influx of young raccoons into the population during the breeding season, or the higher number of inconsistent contacts, may be contributing factors to the fast rabies spread in the breeding season. Determining the relative importance of these factors would be an interesting avenue for future investigation.

In our closed model population of 90 raccoons, rabies outbreaks are predicted to last for <2 years. However, immigration of new susceptible raccoons into an area after the population has declined due to rabies infection could allow for endemicity. Rabies could also be maintained in a larger raccoon metapopulation with the infection spreading spatially across 'patches' in different geographic areas (Lucey *et al.* 2002; Smith *et al.* 2005). A potential metapopulation model of the Chicago metropolitan area might include patches of parks and reserves where habitat is suitable for high densities of raccoons, and the within-patch contact structure might be similar to this study. Contacts between patches would be expected to be less frequent than the within-patch contacts. Future empirical research could document the connectivity between different fragmented raccoon populations; this would enable scaling up of our model to a nested metapopulation model (Caillaud, Craft & Meyers 2013) to look at longer term persistence and endemicity.

As expected, furious behaviour of rabid raccoons leads to a faster spread of rabies and increases final outbreak sizes, whereas dumb behaviour has the opposite effects. The extent of these effects is dependent on the mechanism of implementing the behaviour change, for example, changing the transmission probability or changing the number of contacts. As little is known about the way furious and dumb behaviours are manifested in real raccoon populations, an important area for future research would be to determine the degree and mechanisms of behavioural changes. This could be accomplished by using proximity collars on raccoons that might become infected and then quantifying the changes in network structure upon infection [similar to what has been done in domestic dog populations (Knobel *et al.* 2014)]. When there is a realistic combination of dumb, furious and normal behaviours in the population, there is little change in the distribution of final outbreak sizes and speed of spread compared to when there are no behavioural changes. This finding holds for both modes of implementing behavioural change, suggesting that in models of raccoon rabies, when there is an equal proportion of dumb and furious behaviours, ignoring behavioural changes is a reasonable simplification.

In addition to providing new insights into the spread and control of raccoon rabies in suburban populations, we have introduced novel methods for wildlife epidemiology and network modelling. First, we designed new techniques to incorporate both long-term associations and short-term acquaintances into a unified framework; we included extra temporal detail by using a combination of networks to model these two different interaction types within each season. Secondly, we devised useful ways to scale up our network, using network metrics to ensure that the larger networks had the same critical properties as the original networks. Our scaling up procedure may be useful for building contact networks in other systems where only a subset of a population is intensively

monitored. Sampling and scaling challenges are common in studies of wildlife (but also for humans and domestic animals) due to financial or logistical constraints or technological problems (e.g. Craft & Caillaud 2011; Nickbakhsh *et al*. 2011; Read *et al*. 2012).

Unlike other network epidemiology studies that focus on rare animals of conservation concern (e.g. Hamede *et al*. 2012; Rushmore *et al*. 2013), our research focuses on a common and high-density wildlife species in a suburban landscape with an important zoonotic disease. Because raccoon rabies is spreading westward, efficient and effective control efforts are of increasing importance. However, our study indicates that current vaccination levels may be inadequate to control the spread of rabies in suburban raccoon populations. Alarmingly, high-density raccoon populations in suburban and urban areas facilitate increased contact with humans and their domestic pets (Kappus *et al*. 1970; Jenkins & Winkler 1987), which highlights the importance of understanding rabies dynamics to prevent transmission to humans and other animals.

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## Data accessibility

Data available from the Dryad Digital repository http://dx.doi.org/10.5061/dryad.gr40r (Reynolds *et al*. 2015).

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## Supporting Information

Additional Supporting Information may be found in the online version of this article.

**Table S1.** Distributions of contact durations, for each type of raccoon contact.

**Table S2.** The summary network measures for each type of network, i.e. those made up of consistent contacts and those made up of inconsistent contacts, for each season, for the simulated population of 90 raccoons.

**Appendix S1.** (Sensitivity analysis): Behavioural changes of rabid raccoons may cause changes in the number of contacts (methods and results).

**Fig. S1.** The effects of behavioural changes, as reflected by a change in the probability of transmission.

**Fig. S2.** The effects of behavioural changes, when these affect the number of contacts made by rabid raccoons.

# Supporting Information

**Table S1**: Mean and standard deviation of the log-normal distributions of contact durations, for each type of contact. Distributions are truncated at 604800 seconds (a week). Durations are the time each raccoon dyad spends in close proximity per week. Expected values and modes are also shown to describe the main characteristics of the probability density functions of each distribution.

| | | **Mean of logarithm ($\mu$) in seconds** | **Standard deviation of logarithm ($\sigma$) in seconds** | ***Expected value (arithmetic mean) in minutes*** | ***Point of global maximum (mode) in seconds*** |
|---|---|---|---|---|---|
| **Breeding season, consistent contacts** | Female-female | 6.41 | 3.78 | *325* | *0.0004* |
| | Female-male | 7.41 | 3.11 | *391* | *0.1041* |
| | Male-male | 6.53 | 1.56 | *38* | *60.1235* |
| **Breeding season, inconsistent contacts** | Female-female | 6.20 | 3.18 | *223* | *0.0200* |
| | Female-male | 6.52 | 2.95 | *227* | *0.1128* |
| | Male-male | 7.43 | 2.94 | *368* | *0.2971* |
| **Non-breeding season, consistent contacts** | Female-female | 4.89 | 1.83 | *12* | *4.6697* |
| | Female-male | 5.11 | 2.18 | *28* | *1.4299* |
| | Male-male | 8.24 | 3.00 | *542* | *0.4677* |
| **Non-breeding season, inconsistent contacts** | Female-female | 3.91 | 1.92 | *5* | *1.2506* |
| | Female-male | 4.43 | 2.00 | *10* | *1.5373* |
| | Male-male | 4.05 | 2.53 | *20* | *0.0953* |

**Table S2**: The summary network measures for each type of network, i.e. those made up of consistent contacts and those made up of inconsistent contacts, for each season, for the simulated population of 90 raccoons.

| | BREEDING SEASON | | NON-BREEDING SEASON | |
|---|---|---|---|---|
| | Consistent network | Inconsistent network (averages) | Consistent network | Inconsistent network (averages) |
| Mean degree | 5.0 | 7.6 | 9.6 | 4.4 |
| Density | 0.057 | 0.085 | 0.108 | 0.049 |
| Clustering coefficient:<br>*Triangles*<br>*Open triples* | 0.15<br><br>*249*<br>*1684* | 0.14<br><br>*561*<br>*4117* | 0.19<br><br>*936*<br>*4928* | 0.10<br><br>*125*<br>*1310* |
| Edge sex ratio (female-female: male-female: male-male) | 1 : 2.52 : 1 | 1 : 3.33 : 1.67 | 1 : 3.01 : 1.76 | 3 : 7 : 1 |

**Appendix S1**: Behavioral changes of rabid raccoons may cause changes in the number of contacts

## Methods

Rabies can restructure its host contact network by mediating changes in host behavior (Bansal et al. 2010). For example, rabid domestic dogs are known to change their movement patterns, often wandering far from home or ceasing to move altogether (Hampson et al. 2009); this behavioral change is expected to change the numbers of contacts with other animals (Knobel et al. 2014). For raccoons, we experimented with changing the degree of an infectious node, with furious raccoons making more contacts and dumb raccoons making fewer contacts. As the extent of these changes is unknown, we assumed that if behavior is furious, an infectious raccoon doubles the number of contacts it makes per week. If dumb, a raccoon is assumed to halve its contacts. We ran 500 simulations where all infectious raccoons were furious and 500 simulations where all infectious raccoons were dumb. We also ran 500 simulations for a more realistic scenario of a combination of behaviors, with a third of raccoons furious, a third dumb, and a third normal. We fixed parameter $a$ as 1/9 and report the results for when rabies was introduced at the start of the breeding season.

## Results

When all infectious raccoons display dumb behavior (by decreasing their contacts), the rabies outbreak size tends to decrease and the probability of rabies failing to spread increases (Fig. S2a,d). When infectious raccoons are furious, rabies tends to spread completely through the

entire population (Fig. S2b). When there is an equal combination of behaviors in the population (dumb, furious, normal), there is little change in the distribution of final outbreak sizes compared to when there are no behavioral changes (Fig. S2c,d).

The change in contacts attributed to behavioral changes also affects the speed of rabies spread. The mean time taken for half of the population of raccoons to become infected is 14.3 weeks when infectious raccoons are dumb, and 5.7 weeks when they are furious. For the combination of behaviors, the mean time is 8.4 weeks. When there are no behavioral changes, the mean time is 8.6 weeks.

**Figure S1**: The effects of behavioral changes, as reflected by a change in the probability of transmission, on final outbreak sizes. For this figure, the outbreak begins at the start of the breeding season. 500 simulations are performed for each behavior type.

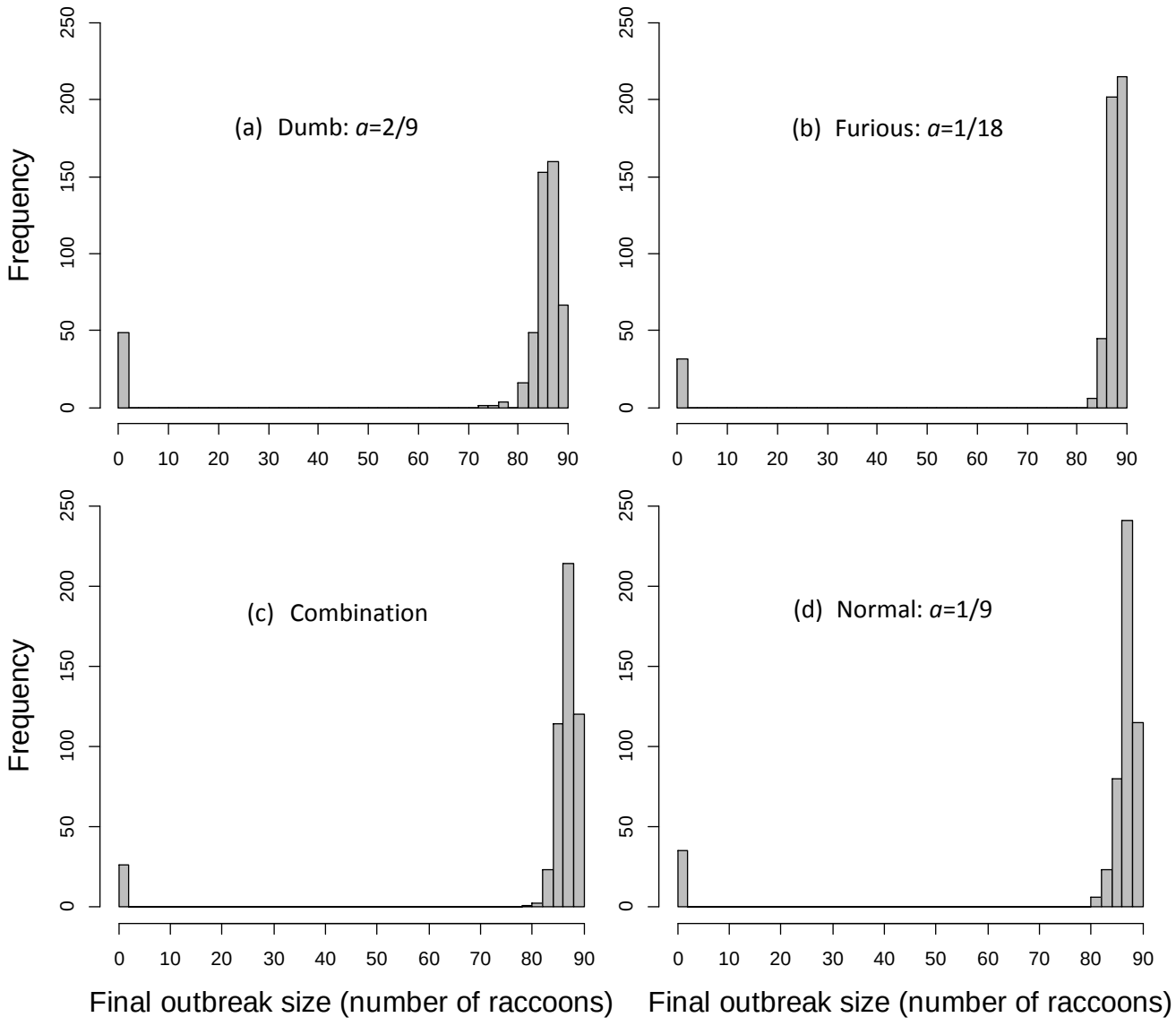
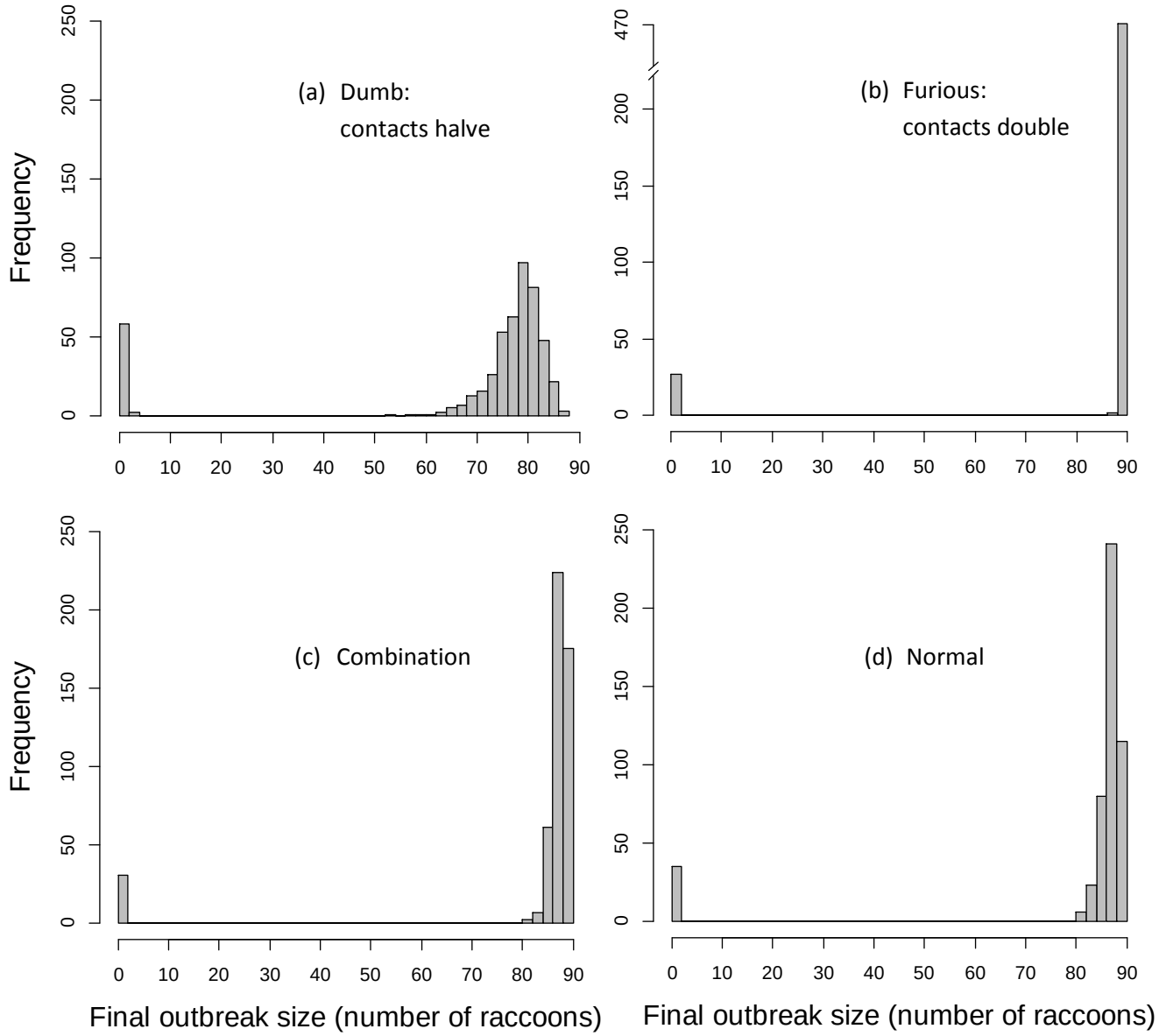


**Figure S2**: The effects of behavioral changes, when these affect the number of contacts made by rabid raccoons, on final outbreak sizes. For this figure, $a$=1/9 and the outbreak begins at the start of the breeding season. 500 simulations are performed for each behavior type. Note the axis break in panel (b).



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