

Which mechanisms drive seasonal rabies outbreaks in raccoons? A test using dynamic social network models

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Summary

1. The timing of raccoon rabies outbreaks in the eastern USA is non-random and often exhibits a seasonal peak. While fluctuations in disease transmission can be driven by seasonal changes in animal population dynamics, behaviour and physiology, it is still unclear which causal factors lead to seasonal outbreaks of raccoon rabies.

2. We used dynamic network modelling to test which of three seasonally changing factors are most likely responsible for raccoon rabies outbreaks: (i) birth pulses, (ii) changes in social network structure and (iii) changes in social contact duration.

3. In contrast to previous predictions, we found that a change in social contact duration was the single most important driver of rabies seasonality. More specifically, co-denning for thermoregulation during the winter increases the amount of time individuals spend in close contact, which in turn should lead to peaks in rabies transmission during the winter.

4. Increased time spent in close proximity during cold winter months has implications for seasonal disease patterns in raccoon populations across a latitudinal gradient, as well as potentially being important for pathogens transmitted by close contact in other wildlife hosts.

5. *Synthesis and applications.* By incorporating detailed empirical data describing variation in raccoon contacts into a network modelling framework, it is possible to determine the likely causal mechanisms driving seasonal disease patterns. This can be crucial information for wildlife and public health officials implementing wildlife disease control programmes.

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

Introduction

Seasonal changes in population dynamics, behaviour and animal physiology can significantly influence the spread of wildlife diseases (reviewed in: Altizer *et al.* 2006). In many cases, these seasonal processes affect the timing and/or severity of disease outbreaks (Gremillion-Smith & Woolf 1988; George *et al.* 2011). For wildlife diseases such as rabies, understanding and predicting the underlying mechanisms for seasonal disease outbreaks can be critically important for implementing and optimizing disease control strategies (Rees *et al.* 2008; Rosatte *et al.* 2009).

However, for most host and pathogen combinations, the link between seasonal changes in animal behaviour and disease transmission is not well understood. Multiple seasonal factors such as birth pulses, changes in social contact patterns and physiological susceptibility to disease can all impact the timing of disease outbreaks simultaneously, making it difficult to determine causal links between these factors and seasonal disease patterns. Altizer *et al.* (2006) state ‘*more explicit modelling approaches are needed to explore how multiple seasonally varying parameters interact with one another and with other perturbations*’. We follow this suggestion and develop a modelling approach to tease apart the causal factors leading to seasonal rabies outbreaks in raccoons *Procyon lotor*.

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Rabies is a multihost pathogen that negatively affects humans, livestock and wildlife of conservation concern (Blanton *et al.* 2011). It is one of the most important zoonotic wildlife diseases in the world; an estimated one person dies from rabies every 15 min, while another 300 or more are exposed (Rupprecht, Hanlon & Hemachudha 2002). Of particular concern in North America is an epizootic variant of raccoon rabies that spread across most of the mid-Atlantic and north-eastern regions of the USA, as well as south-eastern Canada, during the 1980–1990s (Childs *et al.* 2000). This raccoon rabies variant differs from other variants found in North America, which are most commonly associated with skunk, fox and bat hosts. Government officials in the USA have mounted a major effort to slow and reverse the spread of raccoon rabies using oral rabies vaccination (ORV) (Slate *et al.* 2005, 2009). The current programme of rabies surveillance, vaccine baiting and treatment in the USA costs somewhere between \$300 million to \$1 billion USD per year (Uhaa *et al.* 1992; Rupprecht *et al.* 1995; Sterner *et al.* 2009), and given the large cost of these programmes, any information that leads to more effective and efficient rabies prevention policies could lead to significant monetary savings and a reduction in human rabies cases.

In the USA, some studies have reported that the number of raccoon rabies cases peaks during March–May, with possible secondary peaks occurring during September–October (Florida: Bigler, McLean & Trevino 1973; Virginia: Jenkins & Winkler 1987; Jenkins, Perry & Winkler 1988; and Torrence, Jenkins & Glickman 1992). The causal mechanisms that drive these seasonal patterns are currently in dispute. Early research concluded that an increase in the number of social contacts during the winter mating season leads to peaks in rabies cases a few months later (Bigler, McLean & Trevino 1973). Recent research has led to the conclusion that raccoons are far more social than originally assumed (Gehrt & Fritzell 1998a; Chamberlain & Leopold 2002; Gehrt & Fox 2004; Pitt, Lariviere & Messier 2008; Prange, Gehrt & Hauver 2011; Robert, Garant & Pelletier 2012); males are more social than females (Gehrt & Fritzell 1998a; Chamberlain & Leopold 2002; Gehrt & Fox 2004; Pitt, Lariviere & Messier 2008); adult male–female and adult male–male contacts are more frequent and of longer duration during the winter breeding season (Gehrt & Fritzell 1998a; Prange, Gehrt & Hauver 2011; Hirsch *et al.* 2013a,b); and social contacts range from brief encounters to long-term stable associations between pairs of individuals (Prange, Gehrt & Hauver 2011; Reynolds *et al.* 2015a). In regard to rabies outbreaks, Bigler, McLean & Trevino (1973) posited that an increase in aggressive interactions between male raccoons during the mating season, as well as seasonal increases in home range size, could increase the likelihood of rabies exposure during the winter. Once exposed, raccoons become infectious several weeks later (average of 5–5 weeks: Tinline, Rosatte & MacInnes 2002; range of 10–107 days: Winkler & Jenkins 1991) and

remain infectious for about a week (Winkler & Jenkins 1991; Rees *et al.* 2008); therefore, individuals exposed to rabies during the winter could drive rabies outbreaks in late winter or early spring.

Evidence to support the theory that raccoon rabies seasonality is driven by changes in the number of social contacts during the winter is not conclusive. While it has been assumed that levels of aggression between male raccoons increase during the mating season, there is a lack of documentation of these behavioural patterns in the literature. There is also little evidence that male raccoons expand their home ranges during the winter. In one suburban population (Ned Brown Forest Preserve, Illinois), a reduction in available food during the winter is believed to cause adult males to decrease the time spent active, with the ultimate result that male home ranges shrink during winter months (Prange, Gehrt & Wiggers 2004).

Even though the conditions leading to these predictions are not well supported, other seasonal changes may lead to increased rabies cases during the same time frame. For example, the duration of social contacts between pairs of raccoons is often greater during winter months. This increase in contact time is likely due to co-denning behaviour for thermoregulatory purposes during cold winter months (Hirsch *et al.* 2013a). If increased physical proximity leads to a greater probability of pathogen transmission (Salathé *et al.* 2010), disease transmission may increase during the winter season. The **contact duration** hypothesis is consistent with previous studies suggesting that seasonal outbreaks of rabies in skunks are due to winter co-denning behaviour (Gremillion-Smith & Woolf 1988).

While raccoons do not associate with significantly more conspecifics during the winter mating season, **social network** structure does change. The raccoon mating system is polygynandrous, with both sexes mating with multiple individuals in late winter (Gehrt & Fritzell 1999; Roy Nielsen & Nielsen 2007; Hauver *et al.* 2010). Because of this behaviour, opposite sex dyads are much more likely to contact each other during the mating season (Prange, Gehrt & Hauver 2011), which leads to a seasonal shift in social network structure (Hirsch *et al.* 2013a,b). It is feasible that the seasonal rewiring of social contacts increases network connectivity over the course of the year, and thus leads to an increased probability of rabies outbreaks in comparison with a static contact network (Bansal *et al.* 2010; Hirsch *et al.* 2013b). If rabies transmission increases due to dynamic social network rewiring, these seasonal behavioural shifts could be the primary cause of seasonal rabies peaks in the spring. However, because the mating season in our study population (December–March; Hauver *et al.* 2010) largely coincides with cold winter months, both mating and co-denning could lead to similar timing of rabies outbreaks. Because the mating season leads to shifts in social contact structure, an increase in contact duration, and co-denning, we can tease apart the importance of these two causal mechanisms by using empirical data on raccoon social contact patterns and contact durations.

An alternate hypothesis is that tightly seasonal **birth pulses** lead to large bursts of newly susceptible raccoons. This in turn could lead to seasonal peaks in disease outbreaks, which has been seen in a variety of infectious agents and hosts (e.g. measles in humans in sub-Saharan African (Dorélien, Ballesteros & Grenfell 2013); nematodes in Soay sheep (Gulland & Fox 1992)). Duke-Sylvester, Bolzoni & Real (2011) used the birth pulse hypothesis in models of landscape level rabies synchrony across a latitudinal gradient. They predicted that pronounced seasonal birth pulses in northern raccoon populations would lead to an increase in landscape level asynchrony of rabies outbreaks, while southern populations with lower levels of birth seasonality should have higher outbreak synchrony. The authors, however, acknowledge that the reporting of rabies cases in the USA has not yet been conducted on a sufficiently detailed spatial scale to test their model.

In this paper, we test whether three different, but non-mutually exclusive, seasonal factors cause seasonal rabies outbreaks in a suburban raccoon community:

1. Birth pulses.
2. Social network shifts.
3. Contact duration changes.

We tested the effects of these three factors by simulating a set of experiments on a dynamic social network model (Reynolds *et al.* 2015a). This contact network model differs from traditional well-mixed disease models (Anderson & May 1991) in that associations are not random, and the probability of disease transmission is based on the observed social contact structure of a suburban raccoon population currently free of rabies. To test what factor, or set of factors, are most likely leading to seasonal rabies peaks observed in raccoon populations, we ran six different model scenarios to test the three major hypotheses (1 – birth pulses, 2 – social network shifts and 3 – contact duration changes):

Model 0 – normal model based on observed data and includes a seasonal birth pulse (Reynolds *et al.* 2015a).

Model 1 – evenly spaced births (i.e. no seasonal birth pulse).

Model 2a – social contact patterns reflecting those of the mating season throughout the entire year.

Model 2b – social contact patterns reflecting those of the non-mating season throughout the entire year.

Model 3a – mating season contact durations (longer durations) throughout the entire year.

Model 3b – non-mating season contact durations (shorter durations) throughout the entire year.

We predicted that if any of these seasonal factors were the predominant causal agents leading to seasonal peaks in rabies outbreaks, model simulations testing for the importance of those factors would differ from the normal rabies model. Because the three seasonal factors are not mutually exclusive, we also compared rabies outbreak patterns between models. The effect of each factor on rabies transmission was quantified by comparing (i) the rate of

rabies spread, (ii) the average outbreak size and (iii) the proportion of simulations that did not result in a rabies outbreak. We also predicted that when the primary drivers of seasonal outbreaks are removed from the models, simulations would result in rabies patterns that are significantly less seasonal than the normal model.

Materials and methods

CONSTRUCTING CONTACT NETWORKS

We constructed contact networks based on data obtained using proximity logging collars placed on wild raccoons (SirTrack Ltd., Havelock North, New Zealand; Prange, Gehrt & Hauver 2011). These collars recorded the amount of time adult raccoons spent in close proximity (within 1–1.5 m) and the identity of the raccoon pair. Fieldwork was conducted within a 20-ha area of Ned Brown Forest Preserve in suburban Cook County, Illinois (Prange, Gehrt & Wiggers 2003). 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). This core area is located in a subsection of the park that is completely bounded by roads or a lake. We consider this larger, bounded area as our study area. Although there is likely limited movement between raccoons in our study area and the surrounding urban environment, we treated this raccoon community as an isolated raccoon population. The total number of independent raccoons (10 months or older) located in this bounded raccoon subpopulation was roughly 90 individuals during the study period. We used data collected from July 2004 to July 2005.

Contact data from the proximity collars were formatted into 52 weekly adjacency matrices, comprising the total time each pair of raccoons spent in close proximity during that week. We chose to use weekly summaries of all pairwise contacts because (i) this reflects the infectious period of a rabid individual (Winkler & Jenkins 1991; Craft 2015), (ii) we are uncertain how infectiousness changes during the course of a week, and (iii) this avoided issues commonly associated with proximity loggers whereby sustained associations are recorded as multiple shorter contacts (Drewe *et al.* 2012). The mating season corresponded to 16 matrices from 11 December to 1 April and all other matrices corresponded to the non-mating season; matrices for the mating and non-mating seasons were analysed separately. Certain raccoon pairs were recorded in close proximity consistently throughout a season, while contacts between other pairs were less consistent. The adjacency matrices were used to formulate weekly contact networks for each season, and for consistent and inconsistent contacts (for full details, see Reynolds *et al.* 2015a).

Nodes in the networks represent independent contact units (single adult raccoons or mothers with dependent young) and network *edges* were weighted by the amount of time each raccoon pair spent in close proximity in that week (i.e. total contact duration). We found different contact rates between dyads, which depended on the season, the sex of the raccoons involved and whether the contact patterns were inconsistent or consistent. To create each edge, we sampled from the statistical distribution that best fit the observed distribution of contact durations for each specific contact type (Reynolds *et al.* 2015a). Due to raccoon mortality and proximity collar failure over the course of the field study, proximity data from certain individuals were not available

throughout the entire study period (Prange, Gehrt & Hauver 2011; Hirsch *et al.* 2013a,b). Therefore, we restricted our analyses to the 15 raccoons that were consistently present in all weekly matrices. Network statistics calculated from these 15 individuals (including: mean degree, density, clustering coefficient and edge sex ratios) were used to scale up the weekly networks to a larger population of 90 raccoons (Reynolds *et al.* 2015a), which represents the approximate number of raccoons in our study area. The movement, behaviour and demographic patterns of these 15 individuals were indistinguishable (as detected through radiotelemetry) from other radiocollared individuals monitored during 20 + years of study in this area (Prange, Gehrt & Wiggers 2003; Prange, Gehrt & Hauver 2011).

RACCOON DEMOGRAPHICS

When possible, all raccoon demographic parameters were taken from a long-term study (1995–2013) of wild raccoons in Ned Brown Forest Preserve (Table S1). Raccoons typically give birth in the spring, approximately 9 weeks after mating. There is a major peak in mating during weeks 10–12 of the mating season (late February to early March; Hauver *et al.* 2010); we assumed that approximately 50% of births are a result of this 3-week peak mating period. In our study population, 92% of adult female raccoons produce a litter over the course of a year (Prange, Gehrt & Wiggers 2003; Gehrt unpublished data) with an average litter size of 3–6 raccoons (Gehrt unpublished data). Juveniles are dependent on their mothers until approximately 10 months of age, and during this time, there is regular contact and exchange of saliva between mother and offspring due to grooming (Gehrt & Fritzell 1998a,b). It is unlikely that young juvenile raccoons could spread rabies to independent adult raccoons. In our model, raccoons younger than 10 months old were assumed to have the same contacts as their mother and were hence not explicitly included as nodes. Approximately 26% of juveniles survive to 10 months of age (Gehrt unpublished data). For the purposes of our models, we assumed that new 10-month-old raccoons entered our theoretical network 10 months and 9 weeks after the mating season the previous year (between 11 December and 1 April). The average adult death rate, in the absence of rabies, is 25% per year (Prange, Gehrt & Wiggers 2003; Gehrt and Prange 2007; Gehrt unpublished data).

BUILDING THE SIX MODELS

Six simulation models were used to test the three major hypotheses: **Model 0**, Normal model – Networks and demographics as described above.

Model 1, No seasonal birth pulse – All contact patterns and durations were the same as in Model 0, but births were distributed evenly across the year.

Model 2a, Mating season (winter) social contact patterns – In this model, social contacts observed during the mating season were used for the entire year. All other variables, including seasonal births and contact durations, were identical to Model 0.

Model 2b, Non-mating season social contact patterns – In this model, social contacts observed during the non-mating season period were used for the entire year. All other variables, including seasonal births and contact durations, were identical to Model 0.

Model 3a, Mating season (winter) contact durations – Year-round social contact patterns and seasonal births were the same as in Model 0, but contact durations of the mating season were used throughout the year.

Model 3b, Non-mating season contact durations – Year-round social contact patterns and seasonal births were the same as in Model 0, but contact durations of the non-mating season were used throughout the year.

SIMULATING RABIES SPREAD ACROSS CONTACT NETWORKS

For each of the six models, we simulated rabies spread through the raccoon population using a network modelling approach (Craft *et al.* 2009, 2011; Craft & Caillaud 2011; Craft 2015). All epidemiological parameters were taken from studies of the raccoon rabies variant (Table S1). 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. In this instance, removed individuals are dead, as rabies is almost always a fatal disease. We define parameter σ as the rate at which individuals move from the exposed to infectious class, so that $1/\sigma$ is the average incubation period, which we take to be 5.5 weeks (Tinline, Rosatte & MacInnes 2002). Parameter γ is the rate at which infectious individuals die, meaning that $1/\gamma$ is the infectious period, which we take to be 1 week (Hanlon, Niezgodka & Rupprecht 2007). We define $\beta(t)$ as the probability of transmission given an edge between a susceptible and an infectious raccoon. As this rate is likely positively correlated with the duration of contact between an infectious and susceptible raccoon, 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 this is the weight of the edge between the raccoon pair in that week's network). Specifically:

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

for $t > 0$, where parameter α sets the 'steepness' of the curve. Based on published estimates of the basic reproductive rate (R_0) for raccoon rabies (Biek *et al.* 2007) and our previous analyses, we set the value of α at $1/9$ (Reynolds *et al.* 2015a).

We stochastically modelled rabies spread through the raccoon networks (for further details of the procedure, see Reynolds *et al.* 2015a). We started with all nodes susceptible except one randomly selected infectious node (the index case). At each weekly time step, a network of raccoon contacts was created based on network statistics (mean degree, density, clustering coefficient, edge sex ratios) according to the season, including both consistent and inconsistent contacts (Table S2). We monitored the disease status of the nodes in the network (*S*, *E*, *I* or *R*) at each weekly time step and allowed natural death and the entry of new nodes at the rates given in the raccoon demographics section above. For Model 1, instead of seasonal birth, we assumed an evenly distributed birth rate across the year. Because of the frequent contacts and presumed fluid transfer between mother and offspring, when the new 10-month-old raccoons entered our model as a new node, they were given the same disease status as their mother.

We carried out 3000 runs for each of the six models. New contact networks were created for each run, and for each run, the index case and the week of the year in which rabies was introduced were both randomly selected. For models 2a and 2b, contact networks were created from network statistics from the mating season and non-mating season, respectively, and did not shift seasonally throughout the year. Edge weights (contact durations) were sampled from the same distributions as in Model 0. For models 3a and 3b, contact networks were created from the same network statistics as in Model 0, but edge weights were sampled from the distributions corresponding to the mating season and non-mating season, respectively, across the entire year. For simplification of terminology, from here onwards, nodes will be termed 'raccoons', although in reality some of these nodes are mothers with dependent young.

STATISTICS

Consistent with other stochastic models of rabies spread in small wildlife populations (Craft, Beyer & Haydon 2013), our model produced a bimodal shape in final rabies outbreak size (Reynolds *et al.* 2015a); rabies either died out after infecting a small number of individuals or a large outbreak occurred. To compare the relative rate at which rabies spread through our raccoon social networks, we calculated the time between rabies introduction into the system (time zero) and the time when 50% or more of the population had contracted rabies. 50% is a cut-off of 45 individuals that falls solidly in the interepidemic trough between small and large outbreaks (Figures S0–3b). To determine the relative size of rabies outbreaks, we calculated the average number of infected raccoons in the simulation runs (maximum $n = 90$ individuals), as well as the number of simulations with no rabies outbreaks (i.e. <50% of the raccoons contracted rabies). To compare the extent of seasonality in our simulation models, we conducted Rayleigh tests using the number of infected individuals per month in each simulation to calculate whether the number of rabies cases during the year differed from an even distribution (Batschelet 1981). We report Z values for the Rayleigh tests, with higher values indicating a more pronounced seasonal peak in rabies cases. Rayleigh test statistics were calculated using the program Oriana 4.02 (Kovach Computing Services). To determine the length and timing of seasonal rabies outbreaks, we defined any week that had a higher than average number of rabies cases in a given model as an outbreak week. Outbreak lengths were calculated by summing the greatest number of consecutive outbreak weeks within a model.

Results

The mean number of infected individuals per week, which ranged from 0.52 to 1.03 in the normal model based on observed data (0), is seemingly low compared to the total population of 90 individuals. However, an average incidence of 1.73 individuals per week would result in all individuals in our population contracting rabies within a year. Our normal Model (0) resulted in a large amount of seasonal variation in the average number of infected individuals per week (Fig. 1). More infectious raccoons than average were found for 17 consecutive weeks, from 1 January to 23 April (Table 1). The average outbreak size was

large (80 out of 90 raccoons), and the majority of simulations resulted in a rabies outbreak (2851 out of 3000 simulations).

Similar seasonal patterns in the timing and duration of rabies outbreaks were observed in models 1, 2a and 2b, which differed from models 3a and 3b (Fig. 1, Table 1). Seasonal rabies outbreaks started during the week of 1 January in models 0, 1, 2a and 2b, and ended the week of 16 April for models 1, 2a and 2b. Rabies outbreaks for the contact duration models 3a and 3b did not exhibit an obvious seasonal peak as shown in the other simulation models (Table 1, Fig. 1). While the evenly spaced birth model (Model 1) was less seasonal than the normal model (resulting in a lower Rayleigh Z score), the timing, duration and peak mean number of infectious raccoons (Model 0 peak value = 1.03, Model 1 = 1.02) of rabies outbreaks were similar in both models (Fig. 1, Table 1).

The amount of time, in weeks, between the initial rabies case and an outbreak was shorter in the mating season contact duration model (Model 3a; 8.8 weeks) and longer in the mating season contacts and non-mating season duration model (Models 2a and 3b; 12.1 & 12.2 weeks) compared to the normal model (Model 0; 10.9 weeks). Additionally, average outbreak size was smaller in the mating contact model and non-mating duration models (Models 2a and 3b), and slightly larger in all other models compared to the normal model (Table 1).

Discussion

Previous authors have posited that birth pulses or changes in intersexual interaction patterns during the mating season cause seasonal outbreaks of raccoon rabies. When we removed both of these factors from our simulation models, seasonal rabies outbreak patterns remained. However, when we kept contact duration patterns constant throughout the year, we found little to no evidence of seasonality. We conclude from our simulation models that seasonal changes in contact duration are likely a major mechanism for seasonal rabies outbreaks. Because these seasonal behavioural changes are likely driven by co-denning for thermoregulatory purposes, we conclude that the main causal factor of seasonal outbreaks is yearly temperature variation, which leads raccoons to sleep in dens with conspecifics for warmth during winter months. This shift from shorter to longer durations of physical contact likely accounts for increased incidence of rabies after the behavioural switch and could have implications for other pathogens spread by close contact. Through the use of network modelling, we were able to tease apart the causal mechanism leading to seasonal rabies outbreaks without the use of extensive laboratory or field experiments. We conclude that our theoretical modelling approach, parameterized by empirical data on temporal variation in contact patterns, may be useful for inferring mechanisms of seasonal outbreaks in other wildlife systems with seasonal

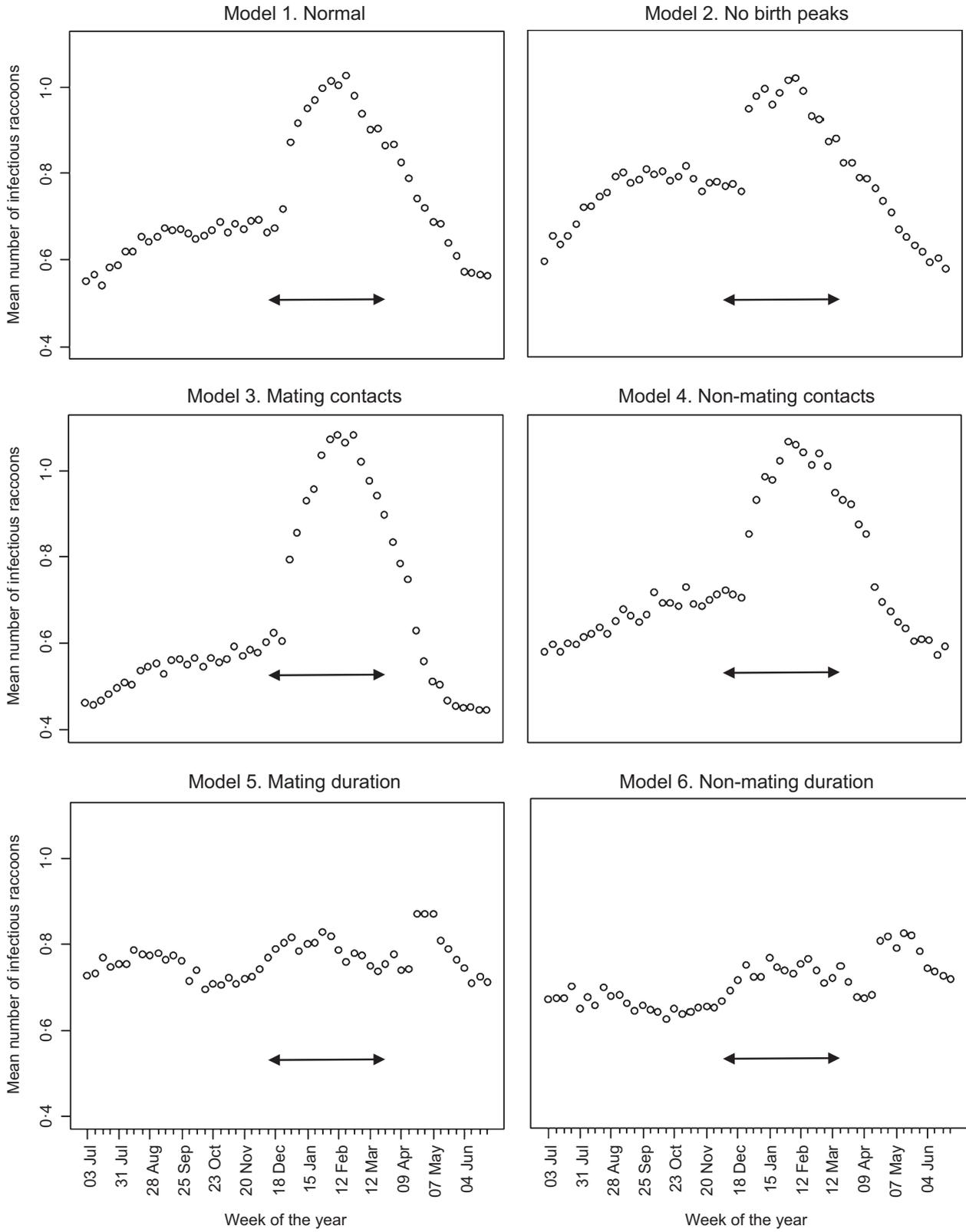


Fig. 1. Mean number of infectious raccoons per week for six different simulation models (3000 simulations per model). Double-sided arrow indicates the mating season.

Table 1. Simulation model results. The ‘number of weeks to outbreak’ is a measure of timing or how quickly rabies spreads in a population; the ‘number of simulations with no outbreak’ is a measure of how often rabies fails to cause an outbreak; and the ‘length of outbreak’ represents outbreak duration. Higher Rayleigh Z values indicate a greater seasonal variation in rabies outbreaks over the course of a year

Model type	# weeks to outbreak	Average outbreak size (max=90)	# simulations with no outbreak (out of 3000)	Length of outbreak (weeks)	Rayleigh Z value
(0) Normal	10.9	80	149	17	1627.32
(1) No birth pulse	10.4	84	144	16	922.73
(2a) Mating contacts	12.1	72	343	16	3510.82
(2b) Non-mating contacts	11.0	83	83	16	1893.13
(3a) Mating duration	8.8	83	76	10	34.13
(3b) Non-mating duration	12.2	77	200	15	164.52

outbreaks of pathogens transmitted by close contact, for example Ebola in great apes (Pinzon *et al.* 2004), bacterial conjunctivitis in house finches (Hosseini, Dhondt & Dobson 2004; Dhondt *et al.* 2012), white-nosed syndrome in bats (Langwig *et al.* 2015) or pulmonary protozoans in voles and shrews (Laakkonen *et al.* 1999).

While we found evidence that changing the duration of social contacts drives seasonal patterns, our simulation models did not indicate that seasonal shifts in the identity of those contacts caused seasonal rabies peaks. In general, raccoons contact other raccoons more frequently and have more unique social contact partners outside the mating season (Prange, Gehrt & Hauver 2011; Hirsch *et al.* 2013a,b), which could have led to the faster rabies transmission in our non-mating season contact model (Model 2b) compared to the mating season contact model (Model 2a). However, the greater sexual segregation in contact patterns outside the mating season leads to social networks which are less connected overall (Hirsch *et al.* 2013b). Perhaps because the mating season social networks were less connected, total outbreak sizes were smallest in the mating season contact model (Model 2a). In addition, the timing of the peak of rabies outbreaks in models 0–2b (January–March) occurred before the peak mating season (Hauver *et al.* 2010). If shifting contact patterns were the primary driver of rabies outbreaks, we would have predicted the rabies outbreak peaks to occur during the spring.

Contrary to predictions, birth peaks had little effect on the seasonality of rabies outbreaks. While the evenly spaced birth model was slightly less seasonal than the normal model (Table 1: Z values), the size and duration of the seasonal rabies peak were similar in both (Fig. 1). In addition, models 0 and 1 both resulted in an increased number of rabies outbreaks during the winter; thus, birth seasonality does not appear to drive the timing of rabies outbreaks. If the arrival of newly independent individuals (starting 14 December) plays a major role, we would predict that the soonest the seasonal rabies outbreak patterns could occur would be 20 January (accounting for the ~5.5 week rabies incubation time), which is a full 20 days after the seasonal outbreaks in our models. Reproductive

seasonality did affect the number of infected raccoons, as the average outbreak size was slightly larger in the evenly spaced birth pattern model (Model 1) compared to the normal model (Model 0). This result appears logical because the influx of newly susceptible individuals is out of sync with the seasonal rabies outbreaks that are driven by contact duration in the normal model. Unlike the normal model, if births are evenly spaced, new individuals can enter into the population during any time of the year, which leads to more rabies cases outside of the normal peak season. Birth pulses may be more important for disease dynamics in systems with high demographic turnover, where individuals develop immunity, or where new individuals are immunologically naïve (Peel *et al.* 2014).

In contrast to the Duke-Sylvester, Bolzoni & Real (2011) study, we conclude that a tighter birth synchrony at northern latitudes should not change the peak timing of rabies outbreaks, but may affect the final outbreak size (Table 1: Models 0&1). Because contact patterns and contact duration may be different in southern USA raccoon populations, the timing of disease outbreaks in these populations may differ, but not necessarily due to differences in birth synchrony. Although there is a lack of empirical data on raccoon social contact and mating behaviour across North America, the Bigler, McLean & Trevino (1973) study of raccoon rabies in Florida reported spring rabies outbreaks occurring approximately 2 months after peak outbreaks from our model simulations. This difference between rabies outbreak timing in our simulations and previously published studies may be due to the fact that detailed studies have not been carried out at the same latitude as our population, which means that the timing of co-denning should differ between our study and previous studies. Alternately, if rabies spreads more quickly in high-density populations, as found in our study, the time lag between the winter denning season and the seasonal rabies outbreak peak may be shorter, compared to lower density populations such as Bigler, McLean & Trevino (1973) study. A combination of additional detailed studies on the demographic and contact behaviour of raccoon populations, in addition to large-scale rabies monitoring, is needed to distinguish between these hypotheses.

Our result that changes in contact duration, likely via co-denning, is the main driver of seasonal rabies outbreaks has important implications for rabies and other wildlife diseases. We predict that other animal species with large changes in contact duration patterns over the course of a year might exhibit similar patterns in the probability of a large disease outbreak occurring after periods of high contact duration. Indeed, skunks *Mephitis mephitis*, another major rabies host, exhibit similar seasonal co-denning and rabies outbreak patterns in temperate climates (Gremillion-Smith & Woolf 1988; but see also: Guerra *et al.* 2003). If this is a general pattern found in multiple species, our results may help wildlife managers and public health organizations plan and implement appropriate management and vaccination programmes, even in species without empirical data on seasonal disease incidence. Because these results are likely driven by seasonal fluctuations in temperature, we posit that these seasonal patterns could differ at varying latitudes. In particular, we predict that the peak size of seasonal outbreaks may be less prominent in warmer climates where the need for co-denning is reduced. However, there are currently no comparative raccoon contact data supporting the hypothesis that southern raccoons spend less time in close proximity during the winter. It is also important to note that the type of social interaction (sleeping, grooming, aggression, feeding in close proximity) may be more important than the total duration of social contacts in predicting transmission (Drewe 2010).

We predict that the timing of rabies outbreaks should vary according to latitude, with northern populations possibly exhibiting outbreaks earlier than southern populations. We also posit that climate change may lead to changes in the seasonality of these wildlife disease outbreaks. For example, rabies outbreaks in the USA may occur later in the year as winters become increasingly mild at northern latitudes. To further test our predictions, future work could determine whether a correlation exists between the timing of rabies outbreaks and the timing and severity of cold winter weather. Here, we suggest that latitudinal differences in the timing of rabies outbreaks could be more closely related to weather patterns than to different patterns of seasonal birth pulses (Duke-Sylvester, Bolzoni & Real 2011). Even if our simulation models had shown that seasonal mating peaks led to seasonal rabies outbreaks, any variation in the mating season may not be particularly important to rabies outbreak patterns because the timing of the mating and birth peaks does not appear to differ substantially across a latitudinal gradient (Gehrt & Fritzell 1998a,b; Chamberlain 1999; Hauver *et al.* 2010; Rosatte *et al.* 2010; Duke-Sylvester, Bolzoni & Real 2011; Troyer *et al.* 2014). However, there is evidence that the duration of these mating and birthing seasons may be longer in southern populations (Troyer *et al.* 2014).

While our simulation model results support the hypothesis that a change in contact duration is a major driver of seasonal rabies outbreaks, additional empirical informa-

tion is needed for confirmation. In particular, detailed, multiyear records of reported rabies cases at similar latitudes to our study population in Illinois should result in seasonal patterns similar to our model. Additionally, it would be ideal to compare data from high- and low-density raccoon populations. Because our data are derived from a relatively high-density suburban raccoon population, rabies may spread more quickly compared to lower density populations (Riley, Hadidian & Manski 1998). While our conclusions are based on modelling rabies transmission within a small, isolated population, more complex patterns could occur at larger spatial scales (Duke-Sylvester, Bolzoni & Real 2011), or when rabies introductions occur only at certain times of the year (Peel *et al.* 2014). In addition, in areas with additional rabies hosts (such as skunks and foxes), complex transmission patterns may emerge that were not captured in our one-species model, and thus, multihost transmission could affect these seasonal patterns as seen in other multihost systems (Lembo *et al.* 2008).

US oral rabies vaccination (ORV) programmes typically distribute ORV baits during the autumn months (August–October) to target newly susceptible juvenile raccoons. During this time, young raccoons are mobile, still closely associated with their mothers and weaned; therefore, maternal antibodies are no longer present to interfere with an immune response to vaccination (Robbins *et al.* 1998; VerCauteren 2015). The exact timing of the baiting depends on latitude, with southern states (NC, TN, GA, VA, WV) dispensing bait 1–2 months later than northern states (ME, NY, OH, PA) (Richard Chipman personal communication). At first glance, one may conclude that the results of our computer simulations indicate that baiting is being conducted too early, and it would be ideal to push back the timing of ORV distribution to coincide with colder winter months when contact durations are longest. However, raccoons also reduce their foraging activity during cold winter months, with some populations exhibiting sharp decreases in body weight during this period (Prange, Gehrt & Wiggers 2004). This variation in raccoon activity has not been incorporated into other models of optimal ORV timing (Clayton *et al.* 2010). We predict that the rate of ORV bait encounters would plummet during these cold winter months; thus, any ORV baiting during this time period is not likely to be an effective strategy. We suggest that to increase the effectiveness of ORV programmes, baiting should be concentrated during late autumn, as close as possible to the change in contact durations, but early enough that the raccoons are still highly active and searching for food. This late autumn ORV timing would also allow a sufficient period of time (10–14 days) in which raccoons can develop antibodies after ingesting ORV baits (Raboral V-RG; <http://www.raboral.com/about-rabies/Pages/raboral-v-rg.aspx>). Indeed, we believe that current ORV programmes are ideally timed, but due to a different set of mechanisms than intended.

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Data accessibility

Data available from the Dryad Digital Repository doi: 10.5061/dryad.gr40r (Reynolds *et al.* 2015b).

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Supporting Information

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

Table S1. Epidemiological and demographic parameters used in the raccoon rabies model.

Table S2. Summary network measures for each type of network.

Figures S0–S3b. Final outbreak sizes for 3000 simulations of each model.