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Running title: Managing a macroparasite: leveraging data and models to mitigate human risk from wildlife infections

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1 Introduction

Rinderpest is eliminated (Roeder, 2011), rabies is reduced (Freuling et al., 2013), and strategies are currently being implemented to manage infectious disease in a number of other wildlife systems [CWD, Koala STDs, etc.]. In many cases, these management strategies have been guided by mathematical models (e.g. Restif et al., 2012; McCallum, 2017). Mathematical models are an important component when managing wildlife disease because they can be used to both inform to design of field-based management strategies and assess the efficacy of a strategy after implementation (Restif et al., 2012). While a number of models have been successfully applied to managing viral, bacterial and protozoan agents in wildlife populations [give examples, many found in McCallum], models directed towards managing wildlife macroparasites are still lacking (McCallum, 2017, , but see X X X for macroparasite models in livestock).

The scarcity of management-based models for macroparasites in wildlife is likely for two reasons. First, macroparasites, such as helminths that do not directly reproduce within a host (Anderson and May, 1979), often have complex life cycles and load-dependent effects on host fecundity and mortality. While these characteristics by no means preclude modeling of macroparasites in wildlife, they do require models that track the distribution of parasite loads across hosts in addition to the total host population and mean parasite load (Anderson and May, 1978; Cornell, 2010). These types of models can quickly become challenging to implement, parameterize, and analyze when they are built to ask system-specific questions regarding parasite management in wildlife systems (McCallum, 2017). Second, in contrast the dramatic effects that microparasite epidemics can have on wildlife populations (e.g. Webb et al., 2006; Hewson et al., 2014) [others], the role of macroparasites as a general regulating factor of wildlife populations is unclear (Tompkins et al., 2002, 2011). This has potentially led to the perception that detailed macroparasite models in managed wildlife systems are less critical. However, even macroparasites that have equivocal effects on the dynamics of wildlife populations can still be an important concern for human health (Page, Graeff, Kazacos). Therefore, macroparasite models are needed that si-

multaneously incorporate the macroparasite dynamics in a wildlife reservoir as well as the the risk this reservoir poses to human populations.

One of the major challenges when using models to manage macroparasite infections in wildlife populations is the scarcity of data. Collecting longitudinal parasite intensity data is all but impossible in wildlife systems due to the difficulty of consistently re-trapping individuals and the fact that accurately measuring parasite intensity often relies sacrificing a host [citation]. Because of these challenges, the type of data that is often available in wildlife macroparasite systems is cross-sectional age-intensity/prevalence data (i.e. age-intensity/prevalence profiles). An important question in parasite ecology is what can we infer about the host-macroparasite system given an observed age-intensity/prevalence profiles (Pacala and Dobson, 1988; Wilson et al., 2002; Duerr et al., 2003)? With limited prior knowledge about a particular system, the answer is “not much” given that multiple mechanisms can lead to very similar age-intensity/prevalence profiles (Wilson et al., 2002; Duerr et al., 2003). However, given some prior knowledge about a host-macroparasite system, this type of data could still be useful for identifying the nature of important components of host-macroparasite systems, such as the structure of transmission. If this prior knowledge (e.g. in the form of an unparameterized dynamic model) can be leveraged with this type commonly collected parasitological data, it could aid in the development of empirically parameterized wildlife-macroparasite models that could be used to address questions regarding the management of human exposure risk.

One of the challenges in linking unparameterized macroparasite models with empirically observed age-intensity/prevalence profiles is that most system-specific macroparasite models rely on individual-based models to capture the complexities of parasite load heterogeneity, age-structure, and concomitant immunity (e.g. Cornell, 2005; Drawert et al., 2017; McCallum, 2017) [OTHERS]. Often these models do not have easily defined likelihood functions with respect to observed age-intensity/prevalence profiles, making it challenging to use likelihood-based fitting techniques to estimate model parameters from this type of data. An alternative approach is to use Approximate Bayesian Computing (ABC) (Beaumont, 2010) to link these individual-based

models to commonly collected age-intensity/prevalence data. In its simplest form, ABC substitutes the likelihood function with a user-defined distance function and then tries to identify a set of model parameters that minimizes the distance between an observed pattern (e.g. the observed age-intensity/prevalence profile) and the same pattern generated by the model for a given set of parameters (e.g. a simulated age-intensity/prevalence profile) (Toni et al., 2009; Beaumont, 2010). The ABC method has been successfully applied in host-microparasite systems to estimate transmission dynamics from data of X and X (Conlan et al., 2012; Kosmala et al., 2015). However, its efficacy has not yet been demonstrated in macroparasite systems [NOT SURE IF THIS IS TRUE], particularly using commonly collected age-intensity/prevalence data [PRETTY SURE THIS IS TRUE].

In this paper we fill this knowledge gap by developing an individual-based model for the macroparasite raccoon roundworm *Baylisascaris procyonis*, which is recognized as a threat to both human and wildlife health (Page et al., 2011; Weinstein et al., 2017). Like many macroparasites, is difficult to control due to its complex life cycle and resistant environmental infectious stages. Worms mature in the raccoon gut and infected raccoons can release over a million parasite eggs per day [citation]. Eggs survive for over a year (Shafir) and accumulate at communal raccoon defecation sites termed latrines [citation]. Individual raccoons contribute to multiple latrines and these contaminated sites expose raccoons and other species to eggs. Eggs infect juvenile raccoons, leading to high parasite loads in animals as young as four months (Weinstein, 2016) [TODO: Fill in these citations: Kazacos, Boyce]. Raccoon susceptibility to eggs declines with age and adult raccoons acquire worms by eating infected small mammals and birds [citation]. In these birds and mammals, larval worms do not mature. Instead, larval worms migrate from the gut into other tissues including the brain, often causing fatal neurological damage. Raccoons scavenge these incapacitated animals [citation], and this trophic transmission maintains infection in older raccoons that are less susceptible to eggs [citation]. Nearly all infected raccoons release eggs; however higher parasite loads in juveniles suggests that this age class contributes disproportionately to human disease risk [citation].

Using this individual-based model, we perform simulations to understand the conditions under which known transmission parameters can be recovered from age-intensity/prevalence data using the ABC approach. We then fit the individual-based model to observed age-intensity/prevalence data and used the resulting model to explore the efficacy of various management strategies on reducing human risk to *Baylisascaris procyonis* exposure. This work demonstrates that given some prior knowledge of a host-macroparasite system reflected in the form of a dynamic model, age-intensity/prevalence data can be used to infer unknown parameters in the transmission function. Moreover, we use this approach to show that it is largely ineffective to reduce human risk to raccoon roundworm via strategies such as culling, deworming, and birth control. Rather, we found that the only effective management strategy was reducing the environmental pool of parasites via cleaning-up raccoon latrines.

2 Methods

The methods are broken into three section. First, we provide an overview and justification of the individual-based model that we built to explore the dynamics of *B. procyonis* in raccoon populations. Second, we describe how we used this model to test the ability and limitations to infer transmission dynamics using age-intensity/prevalence data and the ABC method. Finally, after fitting this model to an empirically observed age-intensity/prevalence profile in raccoon roundworm, we describe how we implemented different management strategies raccoon roundworm and how they affected the model's predictions regarding human risk to *B. procyonis* exposure.

2.1 Description of the individual-based model

To evaluate raccoon roundworm management strategies, we built an individual-based model for a macroparasite with long-live environmental infectious stages and complex life cycle. We describe this model using the Overview, Design concepts, Details (ODD) protocol (Grimm et al., 2006, 2010), providing first an Overview of the model, then describing the Design Concepts,

followed by the Details of submodels, parameterization and initialization. Additional details on submodels and annotated code are available in the Supplementary Material.

2.2 Overview

2.2.1 Purpose

We developed an individual-based macroparasite model for raccoon and raccoon roundworm to compare how host, parasite, and infective stage targeted management affect host demography, parasite demography and human exposure.

2.2.2 Entities, state variables, and scales

In this model, raccoons are the main model entity and exist as an age structured, closed, all-female population. Five attributes describe each raccoon: an identification number, an age, an alive or dead status, a location and a parasite load. We track the female parasite load (i.e. the infrapopulation) within each raccoon. Raccoons can acquire new worms at each time step and, for every raccoon, we explicitly track the age of each worm cohort. Worms accumulate and die over time, and we calculate parasite load by summing across parasite cohorts within each raccoon. Infected raccoons contaminate the environment with long-lived parasite eggs, which we model as an environmental egg pool. As even a single worm can produce more than a million eggs per month [citation], we assume that infected raccoons rapidly saturate local latrines with eggs. Thus, we do not explicitly track each egg, and instead use infection prevalence in the local raccoon population as a proxy for local egg production. Eggs decay over time () and we calculate the current environmental egg pool as a weighted sum of past egg production. Eggs also infect rodents that co-occur with raccoons. We use the environmental egg pool to estimate rodent infection at each time step. [However, because *B. procyonis* is not reproductive in rodents, infected rodents do not directly contribute to the environmental egg pool.]

We track hosts, parasites, and environmental egg pools in a spatially implicit 20 km² world

with 10 equally sized 2 km² zones. Human population density increases consecutively from zones one through ten (Fig X, x) and zones with higher human density support larger raccoon populations (citation, Fig. X).

2.2.3 Process overview and scheduling

We update the model on a monthly time step. The first event that occurs in a time step is raccoon survival. Surviving raccoons then reproduce, lose parasites, gain parasites, disperse, age, and contribute to the environmental egg pool. Following the typical raccoon life cycle, raccoons reproduce once per year, with kits entering the population in the following time step. All other events occur monthly in all ten zones.

2.3 Design Concepts

2.3.1 Emergence and Interactions

Although some infectious agents can regulate their hosts (), raccoon roundworm does not regulate raccoons [citations]. Unmanaged raccoon populations are limited by resource availability (), which we model by setting a raccoon carrying capacity in each zone. As raccoon population density increases within a zone, reproduction declines following a Ricker function (Gurney and Nisbet, 1998). Simulated raccoons reproduce once per year (in the spring), and this pulsed reproduction produces realistic annual population cycles and age distributions for an unmanaged raccoon population (gehr, others).

We add macroparasites to this simulated raccoon population following classic macroparasite modeling theory (Anderson and May, 1978, 1991; Cornell, 2010). Individual host-parasite interactions generate an aggregated parasite population with realistic age-intensity and age-prevalence profiles. The parasite population cycles due to changing host demography, and these emergent patterns match the widely observed fall rise in mean raccoon parasite loads (eg page 2016, weinstein2016, many others).

2.3.2 Stochasticity

We determine the following modeling events using a random number generator in R version 3.2.4: raccoon survival, raccoon reproduction, raccoon litter size, raccoon dispersal, parasite loss, and parasite gain. The probability of a particular realization of any of these events depends on the biology of the raccoon-*B. procyonis* described in *Submodels*.

2.3.3 Initialization

We began each simulation with 500 two-year old raccoons stochastically distributed throughout the 20 km² world according to zone specific carrying capacities (average starting zone density: 1.58, 12.32, 27.56, 40.68, 52.84, 61.84, 69.74, 74.52, 77.18, and 81.74 raccoons per zone). Initially, each raccoon hosted 10 worms and no eggs contaminated any zones. The mean rodent worm load was initially 3.49 based on empirical observations (Weinstein, unpublished) and the variance of rodent worms was set to 87.08 based on the canonical scaling relationship between log-mean parasite load and log-variance in parasite load (Shaw and Dobson, 1995).

2.4 Submodels

2.4.1 Raccoon death

We assume that all raccoons experience a constant per month death probability and then augment this age-independent death rate to account for high nestling mortality (Montgomery 1969) and senescence. Nestling raccoons (<1 month) have constant added risk of death and we model senescence as an increasing age-specific hazard rate, parameterized to ensure that raccoons cannot live past 20 years old [citation]. These three mortality processes lead to a “bathtub”-shaped hazard function for raccoon mortality (Fig. X)

Raccoons can also die from *B. procyonis* infection if worms cause an intestinal obstruction (Stone, 1983; Carlson and Nielsen, 1984). Fatal obstructions can occur at loads as low as 141 worms and have been recorded in animals with 636 and 1,321 worms (Stone, 1983; Carlson and

Nielsen, 1984), however raccoons can survive with over 200 worms (Kazacos 2001). To model this potential parasite-induced host mortality, we used a logistic parasite-induced mortality function $1 - \frac{e^{\beta + \alpha \log(\text{load} + 1)}}{1 + e^{\beta + \alpha \log(\text{load} + 1)}}$, where α gives the per parasite effect on the log odds raccoon survival probability and β roughly corresponds to the threshold at which parasite-induced mortality begins to occur (Wilber et al., 2016). We estimated these parameters using the aforementioned data [which data]. Our parameterization corresponds with the empirical observation that parasite-induced mortality is not a large factor affecting raccoon dynamics.

2.4.2 Raccoon reproduction

Raccoons typically reproduce once a year and can have up to 2 female young per litter (Gehrt 2001, Fritzell 1985, Cowan 1973, Clark et al 1989). Although there is some evidence that populations in the far south of the range may breed year round (Troyer et al 2014) and late litters do occur (Gehrt and Fritzell 1996, 1978a?), here we assume that all reproduction occurs in a single spring pulse.

The model assumes that average raccoon litter size in a given year decreases with increasing raccoon density. This density-dependence corresponds with reproduction in dense populations being limited by both the availability of nest sites and increased infanticide risk in dense populations (Gehrt and Fritzell 1999; Hauver 2010; Wolff 1997). Specifically, we model this density-dependence following a Ricker function (Table X).

Raccoon kits are born into the same human overlap zone as their mothers and stay with their mothers for about a year (citation). After a year, juvenile raccoons disperse to a new zone (which could be same)...

2.4.3 Worm transmission

Raccoons acquire *B. procyonis* through two transmission routes: encounter with *B. procyonis* eggs in the environment and by consuming other animals already infected with *B. procyonis* adults (e.g. rodents) (citations). We assume that raccoons encounter eggs with some probability that is

proportional to the total number of eggs in the environment (on a zone-dependent basis). Conditional on encountering eggs, the total number of eggs encountered follows a negative binomial distribution with a fixed aggregation parameter and fixed number of mean eggs encountered. Finally, the probability that any one of these eggs actually infects the raccoon is a decreasing function of how infected the raccoon (i.e. increased load decreases susceptibility) and is zero if the raccoon is older than 4 months (citations). Finally, the worms are acquired from rodents if the raccoon is older than 4 months. Rodents are encountered and consumed with some probability and the number of worms in the rodent follows a negative binomial distribution with the mean that has a maximum value of 3.49 worms per mouse and decreases with decreasing environmental egg pool. As stated above, variance scales with the mean according to Taylor's Power Law (Shaw and Dobson, 1995). The aggregation parameter and mean parameter when encountering eggs in the environment, the parameter that determines how encounter probability scales with the size of the environmental egg pool, and the rodent encounter probability are all estimated using the ABC method described below.

2.4.4 Worm death

Worms die according to an age-dependent survival function that follows the logistic curve $\frac{1}{\exp(-(a+bage))}$. This means that in any time step, worms of age *age* have a probability of dying according to the above function (Fig X). This function was parameterized with data from Olsen 1958 [Sara, more detail about this study here]. Note that we do not include any negative density-dependence in parasite death rate as we instead included this density-dependence in the probability of a worm successfully establishing (see *Worm transmission*).

2.5 Fitting susceptibility and transmission parameters with ABC-SMC

As described in Table X, most of the parameters in this model were estimated based on previous *B. procyonis* studies in other systems and previous work by S.W. in the current system. However, there were five transmission and susceptibility parameters in the model that were largely

unknown in the raccoon-*B. procyonis* system. These were the mean number of eggs encountered by a raccoon in the environment over a monthly time step, the variability in the number of eggs encountered over a monthly time step, an egg encounter parameter relating the eggs in the environment to the probability of a raccoon encountering an egg, the probability of a raccoon consuming a rodent in a month, and a parameter determining how concomitant immunity increases with raccoon age (Table X).

To estimate these parameters, we used Approximate Bayesian Computing with Sequential Monte Carlo (ABC-SMC) (Sisson et al., 2007; Beaumont, 2010; Kosmala et al., 2015). ABC is typically referred to as a likelihood free parameter estimation method and is often used with simulation-based models in which the likelihood of a data point given the model is intractable to compute. Instead, ABC uses a user-defined distance function to determine how “well” the simulation model parameterized with a set of parameters drawn from some prior distribution can reproduce the observed data. This approach allows one to estimate an approximate posterior distribution for unknown parameters in a model based on how well they can reproduce observed data.

To use ABC to parameterize the unknown parameters in the raccoon-*B. procyonis* IBM, we used observed age-abundance and prevalence profiles for raccoons and *B. procyonis* in our system of interest (Weinstein, 2016). For many mammal macroparasite systems, this type of intensity and prevalence data is the best available parasitological data as longitudinal, individual-based measures of worm intensity are nearly impossible due to both the difficulty of re-trapping individual hosts and the destructive nature of measuring parasite loads. Therefore, developing methods to estimate transmission and susceptibility parameters from this type of data is important for a wide-range of host-macroparasite systems.

Using this age-intensity/prevalence data for the raccoon-*B. procyonis* system, we then implemented the ABC-SMC algorithm as follows (Sisson et al., 2007; Toni et al., 2009)

1. We specified uniform priors around our five unknown transmission and susceptibility parameters: [PRIORS HERE]

2. We drew 10,000 samples from each prior distribution such that we had 10,000 vectors of parameters (in the ABC literature each parameter vector is called a particle).
3. For each of the 10,000 particles, we simulated our model for 100 months to remove the effects of the initial conditions.
4. For a given simulation, we then sampled 189 raccoons over the last 24 months of the simulation which is consistent with the number of raccoons used to build the age- abundance/prevalence profiles given in Weinstein (2016). Specifically, we distributed these 189 sampled raccoons according the eight age classes given in Weinstein (2016).
5. Using this sample, we then constructed the age-abundance/prevalence profile from the simulated data and compared it to the observed age- abundance/prevalence.
6. To compare the simulated and observed data, we first combined the eight observed age-abundance data points, the eight observed interquartile ranges for the age-abundance data points, and the eight observed age-prevalence data points into a vector $S_{obs} = [\text{age-intensity}_1, \text{age-intensity}_2, \dots, \text{age-intensity}_8, \text{iqr}_1, \text{iqr}_2, \dots, \text{iqr}_8, \text{age-prev}_1, \text{age-prev}_2, \dots, \text{age-prev}_8]$. We did the same thing for all 10,000 simulated age-abundance/prevalence profiles such that we had a $10,000 \times 24$ matrix S_{sim} where the columns matched the 24 dimensions in S_{obs} and the rows corresponded to one of the randomly drawn particles.
7. To put the abundance measures and the prevalence data on the same scale, we standardized each column in S_{sim} by subtracting the mean and dividing by the standard deviation of each column. We then performed this identical transformation on S_{obs} *based on the column-wise means and standard deviations from S_{sim}* . This transformation of S_{obs} put the values of S_{obs} in terms of deviations relative to the mean of the simulated data.
8. We then calculated the Euclidean distance between each row in S_{sim} and S_{obs} (i.e. the L2 norm), which resulted in 10,000 distance measures. We then accepted the 500 particles that resulted in the 500 smallest distances.

9. We then equally weighted each of these 500 accepted particles and resampled them with replacement 10,000 times. The upon sampling a particle, we perturbed each parameter in a particle using $\sigma_i \times \text{Uniform}(-1,1)$ where sigma is the standard deviation of the ith parameter in the 500 accepted parameters. This perturbation helps the algorithm explore nearby parameter space.
10. We then repeated 3 - 9 with the 10,000 perturbed parameters 5 times with one important change to step 9 after the first round of sampling. After identifying 500 accepted particles we used the following function to weight each particle: X. This is the importance weighting of a particle that accounts for the fact that particles are no longer being sampled from the prior distribution (Beaumont, 2010) (DOUBLE CHECK WORDING).
11. After the fifth iteration [check], the particles converged on a posterior distribution [How did we determine this...deviation in posterior standard deviation and mean].

This algorithm provided posterior distributions for the five unknown transmission and susceptibility parameters in the raccoon-*B. procyonis* model using commonly obtained age-abundance/prevalence profiles.

Before applying this algorithm to the empirical raccoon-*B. procyonis* data, we tested the ability of this algorithm to recover known transmission parameters from age-intensity/prevalence data. To do this, we fixed the four parameters of interest at specific, known values and simulated the raccoon IBM for 100 years (Supplementary Material). We then sampled 189 raccoons present over the last two years of the model with the same age-structure as given in Weinstein (2016) and considered this our “observed *in silico*” data. We then ran the ABC-SMC algorithm using this “observed *in silico*” data and found that we could recover the true mean number of eggs encountered, the variability in eggs encountered, and the probability of encountering a rodent with some specificity, but could not reliably recover the egg contact parameter [WC] (see Supplementary Material). [Post-hoc simulation analyses illustrated that this parameter has little influence on the resulting age-intensity/age-prevalence profiles, which is consistent with the ABC-SMC

algorithm not being able to identify it (Supplementary Material).] With this caveat in mind, we applied the ABC-SMC algorithm to the observed age-intensity/prevalence data from Weinstein (2016).

2.6 Management strategies for *B. procyonis*

We used our fully parameterized model to explore of how different management strategies affected the dynamics of worm and raccoon populations and human risk of encounter with *B. procyonis*. We considered four distinct management strategies in addition to combinations of these management strategies: culling raccoons, birth control of raccoons, anti-helmenthic baiting, and latrine cleanup. For each of these management strategies, we considered different levels of management effort (see below for definitions) and explored how different management strategy-by-effort combinations affected four major predictions of the model:

1. The total raccoon population
2. The total worm population
3. The level of human risk [define above]
4. The mean raccoon worm load

For each management strategy, the model was run for 200 months to obtain equilibrium dynamics, at which time the management strategy was enacted monthly for an additional 200 months (FIG. X). Upon completion of the simulation, the four major model predictions listed above were computed based on the mean [max, min, var, too] over the last 24 months of the simulations.

The different management strategies were implemented as follows

Culling raccoons

[SARA: Could you add a paragraph on culling?]

To implement raccoon culling, we assumed that some number of traps were randomly distributed in the raccoon world each month. We assumed that per trap probability of catching a raccoon increased with raccoon density following a type I functional response: $1 - \exp(-\text{raccoon density} \gamma)$. γ was estimated based raccoon trapping data from Prange et al. (2003) and Graser et al. (2012) [probably more description here...]. Finally, we assumed that the number of raccoons were caught each month followed a $\text{num_trapped} \sim \text{Binomial}(N, p)$ where the p = probability of success was the per trap probability and the N = number of trials was the number traps deployed that month. Because we assumed that all individual raccoons were equally likely to be trapped, we then randomly selected $\min(\text{num_trapped}, \text{total population size})$ raccoons from the population to cull in that time step.

We also allowed trapping to occur in specific zones of human overlap and only for particular age raccoons. When trapping in a particular zone(s) of human overlap, trapping probability was determined only by the density of raccoons in that zone(s) and only raccoons in that zone could be trapped and culled. When trapping for a particular target age class, trapping proceeded as described above, but raccoons that were not in the target age class were released.

Birth Control

[SARA: Could you add a few sentences on birth control]

Birth control followed the exact same trapping regime as culling. However, upon trapping a raccoon its reproductive ability was set to zero for the remainder of its life [TODO: define reproductive ability above]

Anti-helminthic baiting

[SARA: Could you add some information on anti-helminthic baiting]

We implemented anti-helminthic baiting by first randomly distributing some number of baits into the raccoon world. Of the initial number of baits distributed into the world [WC], on average 60% of these baits were degraded or consumed by animals other than raccoons. For the remaining baits, we assumed that all raccoons were equally likely to consume a bait and randomly assigned

baits to raccoons in the world. A given raccoon could consume multiple baits in a month. Upon consuming a bait, all the worms in a raccoon were killed, but the raccoon could immediately start acquiring worms again from the environment and rodents. Consuming one bait or multiple baits in a month had identical effects on raccoon worm loads. Similar to culling and birth control, we also allowed for anti-helminthic baiting in specific zones of human overlap.

Latrine cleanup

[SARA: Could you add some background information]

The final management strategy we explored was latrine cleanup. [Define latrine here or above?]. This strategy was implemented reducing or eliminating the environmental egg pool each month. Once again, this strategy could be specifically implemented in specific zones of human overlap.

3 Results

Fitting transmission parameters with age-abundance/prevalence profiles

Figure in this section

- Plots of posterior distributions of parameters transmission parameters
- Plots of observed and predicted age-abundance/prevalence profiles
- Plots of model dynamics without any management after fitting

Management strategies for reducing human risk of B. procyonis infection

Figures in this section

- Heat maps of various strategies
- Heat maps of strategy combinations

4 Discussion

When we talk about birth control- There is currently no approved raccoon contraceptive (), although products developed for dogs and cats could be optimized for raccoons given high cost and not great results, strategy doesn't seem worth pursuing.

Culling- potential for a "Hydra effect": It's got a name! Abrams 2009. When does greater mortality increase population size? the long history and diverse mechanisms underlying the hydra effect. Ecology Letters 12:462–474 (named for greek myth, where chopping of one hydra head caused two to grow back. While density dependent birth rates set up possibility, and do lead to increased prev/intensity in animals, overall reduction in rac population still reduces human risk, although not as quickly perhaps as would if no "hydra effect" occurred.

5 Acknowledgments

Acknowledgments, including funding information, should appear in a brief statement at the end of the body of the text. Acknowledgments of specific author contributions to the paper should appear here.

Tables (1 per page)

Figure Legends

Figures (1 per page, labeled "Figure 1", etc)

- Figure sizes should be no more than 6 inches wide and 7 inches high. When possible, submit figures in the size you wish to have them appear in the journal. Most illustrations, except some maps and very wide graphs, should be 1-column width (3 inches) at a resolution of 600 dpi.
- The font size of the x - and y -axis numbers should be slightly smaller than the axis label. A consistent font (Helvetica is preferred) should be used throughout. Use boldface type only if required for journal style. Use sentence case (i.e., only capitalize the first word) for axis

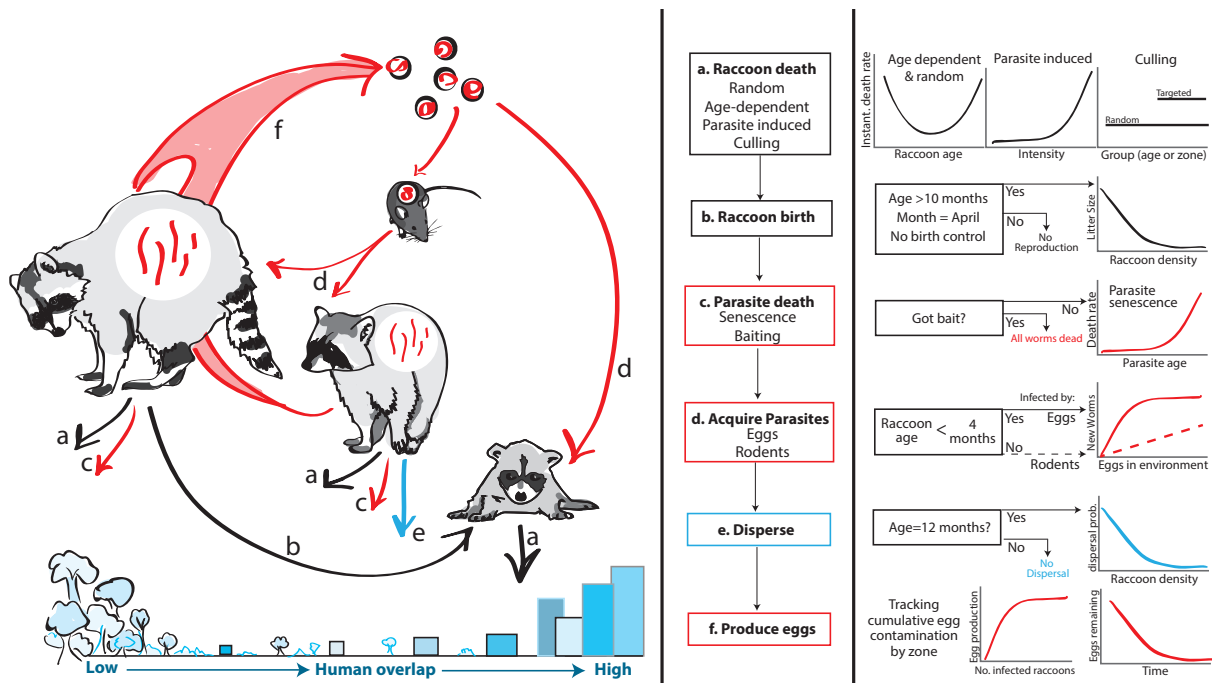


Figure 1: Diagram of raccoon model

titles, labels, and legends.

- For symbols and lines, avoid very small sizes and line thicknesses (1 point width stroke or greater is preferable). All elements of a figure should appear with the same degree of intensity. If different degrees of intensity need to be conveyed, lines should differ by 1 point width for clarity.

Appendices

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