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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 important 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 no 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 individual-based longitudinal parasite intensity data is all but impossible in most wildlife systems due to the difficulty of consistently re-trapping individuals and the fact that accurately measuring parasite intensity often relies on 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 Pacala and Dobson, 1988; Wilson et al., 2002). 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, in the form of an unparameterized dynamic model, can be combined with this type of 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.

Because 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], linking unparameterized macroparasite models with empirically observed age-intensity/prevalence profiles is challenging. Often these models do not have easily defined likelihood functions, such that standard likelihood-based fitting techniques can not be used. One alternative approach for fitting individual-based models to age-intensity/prevalence data is Approximate Bayesian Computing (ABC) (Beaumont, 2010). 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 observed data (Conlan et al., 2012; Kosmala et al., 2015), yet its ability for identifying the structure of parasite transmission has not yet been demonstrated in macroparasite systems using commonly collected age-intensity/prevalence data.

We fill this knowledge gap by developing an individual-based model for the macroparasite raccoon roundworm *Baylisascaris procyonis*. Raccoon roundworm is recognized as a threat to both human and wildlife health (Page et al., 2011; Weinstein et al., 2017) and 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/prevelance data and used the resulting model to explore the efficacy of various management strategies for 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 demonstrate 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 for inferring transmission dynamics from age-intensity/prevalence data using 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 potential management strategies for raccoon roundworm.

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 km^2 world with ten equally sized two km^2 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 (gehrt, 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 km^2 world according to zone specific carrying capacities. The average starting zone densities from lowest to highest were 1.58, 12.32, 27.56, 40.68, 52.84, 61.84, 69.74, 74.52, 77.18, and 81.74 raccoons per two km^2. 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. 1)

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 use a logistic parasite-induced mortality

function $1 - \frac{e^{\beta+\alpha*log(load+1)}}{1+e^{\beta+\alpha*log(load+1)}}$, where α gives the per parasite effect on the log odds raccoon survival probability over a month and β relates to the threshold at which parasite-induced mortality begins to occur (Wilber et al., 2016). We estimated these parameters using... 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 (Fig. 1).

The model assumes that average raccoon litter size in a given year decreases with increasing raccoon density (Fig. 1). 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).

Raccoons 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. We modeled the probability of a juvenile raccoon dispersing to any particular zone (including the zone in which they were born) being proportional to the ratio between the carrying capacity of that zone and the current density of the zone. This means that juvenile raccoons were more likely to disperse to zones that had less raccoons relative to the zones carrying capacity [TODO: CHECK THE IMPLEMENATION OF THIS]. Once a juvenile disperses to a new zone, we assumed that it remained in this zone for the rest of its life.

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. Raccoons only encounter eggs in the zone that they occupy. 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 the determines how encounter probability scales with the size of the environmental egg pool, and the rodent encounter probability are all unknown for this system and needed to be estimated using age-intensity/prevalence data estimated and the ABC method.

2.4.4 Worm death

Worms die according to an age-dependent survival function that follows the logistic curve $\frac{1}{\exp(-(a+b\text{age})}$ (Fig. 1) [DEFINE THIS CONSISTENTLY]. 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 Inferring susceptibility and transmission parameters with ABC-SMC

2.5.1 Simulating age-intensity/prevalence profiles

As shown in Table 1, the raccoon IBM had four parameters relating to susceptibility and transmission that were unknown: 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, and the probability of a raccoon consuming a rodent in a month.

Using our model, we explored whether commonly collected age-intensity/prevalence profiles contained enough information to estimate these unknown parameters. In particular, we considered a set of known susceptibility and transmission parameters that produced age-intensity/prevalence profiles generally consistent with what we saw in raccoon-*B. procyonis* systems (Fig. X). With these known parameters, we then simulated the raccoon IBM until it was stationary and then sampled 189 raccoons according to the age-structure given in Weinstein (2016). Using these simulated and sampled raccoons, we built an age-intensity/prevalence profile that we considered our "*in silico* observed" data (as opposed to the "empirically observed" data given by Weinstein (2016)).

2.5.2 The ABC-SMC method

Given our "*in silico* observed" data, we then tested whether we could recover the known susceptibility and transmission parameters using Approximate Bayesian Computing with Sequential Monte Carlo (ABC-SMC) (Sisson et al., 2007; Toni et al., 2009; Beaumont, 2010; Kosmala et al., 2015). ABC is a likelihood-free parameter estimation method and is often used with simulation-based models in which the likelihood of data 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. Moreover, it allows one understand (?) which parameters in the model are identifiable from the data provided (Toni et al., 2009).

First using the "*in silico* observed" age-intensity/prevalence data, we implemented the ABC-SMC algorithm following Sisson et al. (2007); Toni et al. (2009)

[TODO: Move this to the supplement]

1. We specified uniform priors around our four unknown transmission and susceptibility parameters: $\mu_{\text{encounter}} \sim \text{Uniform}(10, 1000)$, $k'_{\text{encounter}} = \frac{1}{1+k_{\text{encounter}}} \sim \text{Uniform}(0, 1)$, egg encounter $\sim$ $\text{Uniform}(0.001, 5)$, and $p_{\text{rodent encounter}} \sim \text{Uniform}(0, 1)$.

2. We drew 10,000 samples from each prior distribution such that we had 10,000 vectors of parameters (henceforth a 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, consistent with how we sampled the "observed" data.

5. Using this sample, we then constructed the age-intensity/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-intensity data points, the eight observed interquartile ranges for the age-intensity data points, and the eight observed age-prevalence data points into a vector S_{obs} = [age-intensity$_1$, age-intensity$_2$, ..., age-intensity$_8$, iqr$_1$, iqr$_2$, ..., iqr$_8$, age-prev$_1$, age-prev$_2$, ..., age-prev$_8$]. We did the same thing for all 10,000 simulated age-abundance/prevalence profiles such that we had a 10,000 x 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 intensity data, IQR data, and 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. Upon sampling a particle, we perturbed each parameter in a particle by $\sigma_i \text{Uniform}(-1, 1)$ where σ_i is the standard deviation of the ith parameter in the 500 accepted parameters. This perturbation helps the algorithm explore nearby parameter space. If this perturbation pushed any parameter inside a particle outside of the range of its prior distribution, we set the new particle equal to the old particle without perturbation [POTENTIALLY IMPLEMENT DIFFERENTLY].

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 (Toni et al., 2009)

$$w_t^{(i)} = \frac{\pi(\theta_t^{(i)})}{\sum_{j=1}^{N} w_{t-1}^{(j)} K_t(\theta_{t-1}^{(j)}, \theta_t^{(i)})} \tag{1}$$

where θ_i refers to particle i, t refers to the tth iteration greater with $t > 1$, $K_t(\theta_{t-1}^{(j)}, \theta_t^{(i)})$ is probability of the current particle based on the previous particle and the perturbation kernel K_t. In summary, 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).

Applying this algorithm to the "*in silico* observed" data, we were able to obtain posterior distributions for the four [MAYBE FIVE?] transmission and susceptibility parameters in the raccoon-*B. procyonis* model using information contained in the age-intensity/prevalence profiles. We then examined the marginal posterior distributions of each parameter to determine which of these parameters were inferable from the age-intensity/prevalence data and whether the known parameters could be recovered with any precision. Finally, we performed posterior predictive diagnostics and compared the simulated age-intensity/prevalence profiles from the estimated posterior distributions to the "*in silico* observed" data.

2.5.3 Application of ABC-SMC to empirically observed data

After identifying the successes and limitations of the inferring known transmission and susceptibility parameters from simulated age-intensity/prevalence data, we applied the ABC-SMC algorithm discussed above to the empirically observed age-intensity/prevalence data in the raccoon-*B. procyonis* system. [Also tested different assumptions about concomitant immunity?]

2.6 Management strategies for *B. procyonis*

We used our fully parameterized model to explore 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: the total raccoon population, the total worm population, the level of human risk, adn 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 biological basis of the management strategies and there implementation in the model are described below

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... in the Sup Material). Finally, we assumed that the number of raccoons were caught each month (`num_trapped`) followed a Binomial distribution with parameters N = the number of traps deployed that month and p = per trap probability of catching a raccoon as described above. Because we assumed that all individual raccoons were equally likely to be trapped, we then randomly selected `num_trapped` 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 it was no longer able to reproduce for the remainder of its life. However, this raccoon could still be trapped again in proceeding months.

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, on average 60% of these baits were degraded or consumed by animals other than raccoons [citations]. 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. This strategy was implemented by reducing or eliminating the environmental egg pool each month. By reducing or eliminating the egg pool, this strategy directly affected the risk of human infection as humans obtain infections through eggs in the environment. Therefore, we would *a priori* expect this strategy to be effective. However, our expectations are less clear regarding the efficacy of the aforementioned strategies compared to latrine cleanup. Similar to the other strategies, latrine cleanup could be specifically implemented in particular zones of human overlap as each zone has its own independent egg pool.

3 Results

3.1 Inferring transmission and susceptibility parameters from age-intensity/prevalence data

3.1.1 Simulated data

In summary, there were four parameters that we identified as

3.1.2 Empirical data

3.2 Managing human risk to *B. procyonis* exposure

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.

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Submodel/parameter description	Function/Value	Method or Source
Parasite-induced host mortality: Load-dependent probability of worm-induced raccoon death over a month.	$1 - \frac{1}{1+e^{-(\beta+\alpha \log(\text{load}+1))}}$ $\alpha = -4.2$, $\beta = 22$	Parameters selected to match empirical empirical data suggesting that raccoons can suffer parasite-induced mortality between 120 - 1000 worms, but still have some non-zero survival probability above 200 worms (Kazacos, 2001)
Kit death: The probability of a raccoon kit dying its first month	0.31	Based on Montgomery (1969), kit survival probability is 0.52 over 7 weeks. Assuming equal weekly survival probability, the monthly death probability is $1 - (0.52^{1/7})^4 \approx 0.31$
Random death probability: Monthly, age-independent probability of death	0.01	[TODO: Why?]
Senescence: The monthly probability of dying due to senescence related events increases	$\min(1, (1 - 1.74 \times 10^{-5}) \times \text{age}^2)$	Parameterized such that raccoon monthly death probability is one at and above 20 years old [citation]
Age of egg resistance: The age in months at which raccoons begin to develop concomitant immunity to *B. procyonis* eggs	4 months	[CITATION]
Concomitant immunity function: At 4 months old, raccoons begin developing concomitant immunity such that the probability of an egg developing to an adult decreases.	$e^{-\text{conc. immunity} \times (\text{age in months} - 4)}$ conc. immunity = Unknown	Only applied to raccoons that are 4 months old or greater. Estimated via ABC-SMC
Rodent encounter probability: The probability of raccoon encountering and consuming a rodent in a month	Unknown	Estimated using ABC from age-intensity/prevalence profile (Weinstein, 2016)
Mouse worm mean: Mean worm load in *Peromyscus* mice	3.49 worms per mouse	Estimated from Weinstein, unpublished [or published?]
Larval worm infectivity: Probability of a larval worm establishing	0.25	[Where does this come from?]
First reproduction age: The age at which a raccoon can reproduce	12 months	[citation?]
Maximum litter size: The maximum number of female kits in a raccoon litter	2 kits	[Where does this come from?]
Encounter mean: Given an encounter with eggs in the environment, the mean number of *B. procyonis* eggs that a raccoon encounters	Unknown	Estimated from age-intensity/prevalence profile
Encounter k: Given an encounter with eggs in the environment, this parameter determines the variability in the number of eggs encountered by a raccoon	Unknown	Estimated from age-intensity/prevalence profiles
Egg contact function: The probability of a raccoon encountering a clump of eggs in the environment depends on the number of eggs in the environment	$1 - e^{-\text{contact} \times \log(\text{eggs})}$ contact parameter unknown	Estimated from age-intensity/prevalence profile

Infectivity: The per egg probability that, after contacting a raccoon, the egg hatches and develops into an adult worm	0.02	Value from Croll et al. (1982) and Woodruff 1974
Resistance function: Function determining how current parasite load affects the probability of a raccoon becoming infected in a time step	$0.02 \times e^{-\text{resistance} \times \text{load}}$ resistance = 0.03 UNITS?	Croll et al. (1982)
Egg decay: The decay of eggs in the environment. Specifically, the contribution of past eggs to the current egg pool declines exponentially with time according to the parameter egg decay.	$e^{-\text{decay} \times \text{time}}$ decay = 0.3 time^{-1}	Parameterized such that 2% of eggs in the environment survive after 1 year [citation]
Worm survival function: The age-dependent probability that worm infecting a raccoon survives for a month	$\frac{1}{1+\exp(-(a_{\text{thresh}}+b_{\text{thresh}} \times \text{worm age}))}$ $a_{\text{thresh}} = 4.710$, $b_{\text{thresh}} = 0.945$	Parameters estimated based on data from [Olsen 1958] (See Supplementary Material)

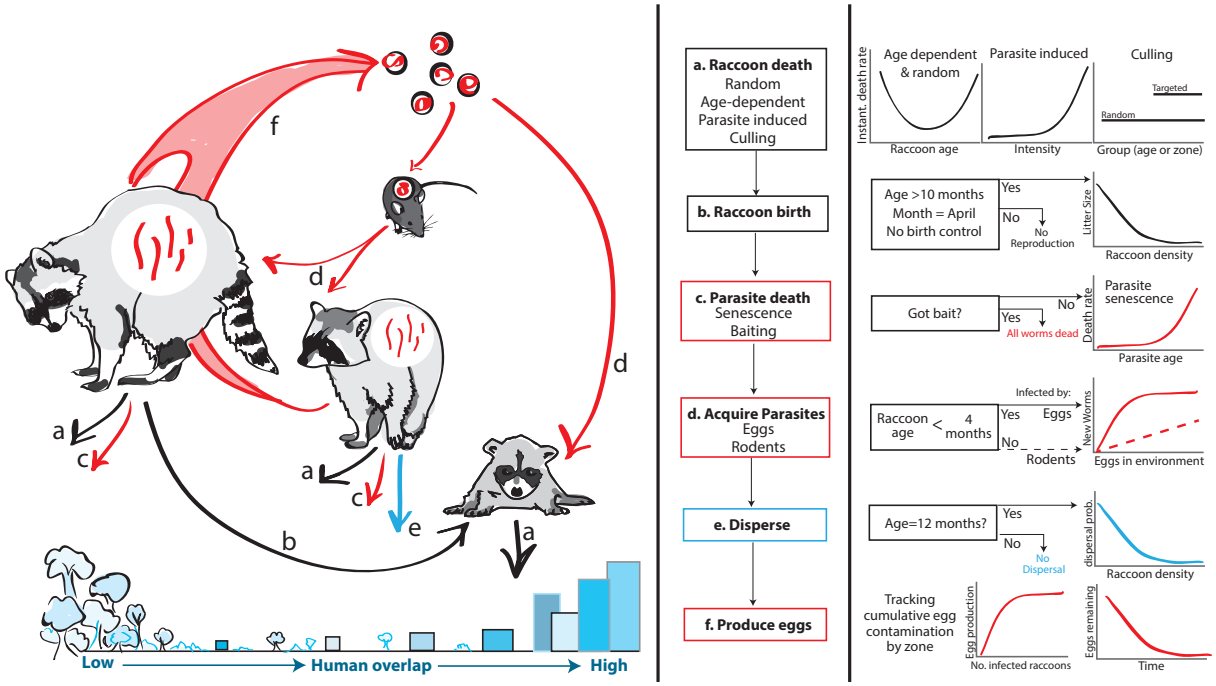

Figure 1: Diagram of raccoon model