International Journal for Parasitology 46 (2016) 59–66

Contents lists available at ScienceDirect

International Journal for Parasitology

journal homepage: www.elsevier.com/locate/ijpara

Detecting and quantifying parasite-induced host mortality from intensity data: method comparisons and limitations

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ARTICLE INFO

Article history:
Received 19 June 2015
Received in revised form 15 August 2015
Accepted 24 August 2015
Available online 16 October 2015

Keywords:
Parasite aggregation
Negative binomial distribution
Crofton Method
Host survival function
Lethal dose

ABSTRACT

Parasites can significantly impact animal populations by changing host behaviour, reproduction and survival. Detecting and quantifying these impacts is critical for understanding disease dynamics and managing wild animal populations. However, for wild hosts infected with macroparasites, it is notoriously difficult to quantify the fatal parasite load and number of animals that have died due to disease. When ethical or logistical constraints prohibit experimental determination of these values, examination of parasite intensity and distribution data may offer an alternative solution. In this study we introduce a novel method for using intensity data to detect and quantify parasite-induced mortality in wildlife populations. We use simulations to show that this method is more reliable than previously proposed methods while providing quantitative estimates of parasite-induced mortality from empirical data that are consistent with previously published qualitative estimates. However this method, and all techniques that estimate parasite-induced mortality from intensity data alone, have several important assumptions that must be scrutinised before applying those to real-world data. Given that these assumptions are met, our method is a new exploratory tool that can help inform more rigorous studies of parasite-induced host mortality.

1. Introduction

Infectious agents can impact animal populations by changing population dynamics and stability (Dobson and Hudson, 1992; Tompkins et al., 2002), altering predator–prey interactions (Joly and Messier, 2004), and even causing species' decline and extinction (De Castro and Bolker, 2005; McCallum, 2012). Accurately estimating the impact of these infectious agents in wildlife is critical to understanding what regulates host and parasite populations, making predictions about disease transmission and managing disease outbreaks (Langwig et al., 2015). The impact of pathogens such as rabies (Coyne et al., 1989), bovine tuberculosis (Cox et al., 2005) and rinderpest (Tillé et al., 1991), are typically modelled based on the presence or absence of disease, such that host survival is not generally considered to be a function of the number of infectious agents present within the host. In contrast, models of macroparasites generally assume that pathology increases with parasite burden and host survival probability must be treated as a function of infection intensity (Anderson and May, 1978). Helminths exhibiting this intensity-dependent pathology have significant impacts on human health (Brooker et al., 2004), domestic livestock economics (Roeber et al., 2013) and wildlife survival (Kirk, 2003; Logiudice, 2003). While it is generally assumed that some fraction of wild host populations succumb to parasitic infection, it is notoriously difficult to actually quantify parasite-induced host mortality (PIHM) in wild animal populations because it is difficult to observe the dead or dying hosts most impacted by parasitism (McCallum, 2000).

Ideally, PIHM is quantified by experimentally infecting and tracking individual hosts in the wild population; however, for logistical and ethical reasons this method is rarely feasible (McCallum, 2000). Snapshot data of parasite intensities across multiple hosts is much easier to collect and has often been used to identify the presence of PIHM (Crofton, 1971; Lester, 1977, 1984; Lanciani and Boyett, 1989; Royce and Rossignol, 1990; Ferguson et al., 2011), and to quantify the relationship between infection intensity and host mortality (Adjei et al., 1986).

Crofton (1971) first proposed that PIHM could be identified from parasite intensity data by comparing the observed parasite distribution in sampled hosts with the distribution predicted in the absence of parasite-induced mortality. This method assumes that, prior to host mortality, infection intensity in the host population follows a negative binomial distribution and the tail of the distribution is truncated as intensity-dependent pathology removes

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http://dx.doi.org/10.1016/j.ijpara.2015.08.009
0020-7519/

the most heavily infected hosts. Assuming mortality occurs only in heavily infected hosts, evidence of this parasite-induced mortality should then be detectable by iteratively fitting a negative binomial distribution to hosts with lower and lower parasite intensities, and comparing these truncated predicted distributions with the corresponding truncated observed parasite data (Fig. 1, see Supplementary Data S1.1 for additional detail).

While the Crofton Method detects the presence of PIHM, it makes no attempt to quantify the relationship between infection intensity and host survival probability; information that is necessary for estimating parasite impacts on host populations (Anderson and May, 1978; Tompkins et al., 2002). Adjei et al. (1986) suggested that this relationship could be calculated by first using the Crofton Method to estimate the pre-mortality parasite distribution and then using this distribution to calculate the probability of host survival with increasing parasite intensity. To do this, Adjei et al. (1986) modelled host survival as a logistic function and then used a generalised linear model (GLM) to estimate the parameters of the host survival function (see Supplementary S1.2 for a technical description of the Adjei Method). Although this method can predict the host survival function, it has several technical drawbacks. When mean infection intensity is high or sample sizes are small, the observed intensity data must be subjectively binned into intensity ranges in order to fit the GLM framework. Furthermore, for the Adjei Method to work, any observed intensity values greater than predicted values must be modified and set equal to the predicted values (see Supplementary Data S1.2 for details); a questionable act of data manipulation. These manipulations may introduce bias, reduce the precision and limit the power of this method to detect and quantify parasite-induced host mortality.

After 30 years, and despite clear limitations (McCallum, 2000), these methods (particularly the Crofton Method) are still discussed among parasitologists and are the primary techniques for examining population-level impacts of parasitism using parasite intensity data. In these methods, PIHM can only be identified by visually examining plots of the pre-mortality parameters predicted by the Crofton Method and determining whether they show a "kink" over a range of truncation values (Fig. 1B; Lester, 1984; Ferguson et al., 2011). These qualitative criteria make it difficult to compare PIHM between studies and a more rigorous and quantitative method is needed to both detect and quantify host mortality. The survival function given by the Adjei Method may be used to do this; however, it requires manipulation of the original data and its accuracy remains untested.

In this study, we propose a novel method for detecting and quantifying PIHM that ameliorates many of the aforementioned deficiencies of the previous methods. Our method does not require data alteration, is highly generalizable and uses standard statistical techniques to quantitatively determine whether PIHM is occurring in a system. We use simulations to compare our method with the Adjei Method to test the ability of both to (i) detect occurrence of PIHM and (ii) estimate the host survival function. We then apply both methods to real datasets previously used in PIHM analyses and compare the results. Finally, we discuss the limitations of inferring PIHM from intensity data and how these methods fit in modern quantitative parasitology.

2. Materials and methods

2.1. A novel, likelihood-based method for estimating PIHM

Our method (henceforth the Likelihood Method) begins with the same assumptions as the Adjei Method: namely that infection has occurred and hosts with fatal parasite loads have died prior to the population sampling. As discussed by Adjei et al. (1986), this is not necessarily unrealistic as some parasite infections occur primarily in younger hosts with parasite-induced mortality occurring soon after infection (e.g. Schotthoefer et al., 2003; Johnson and McKenzie, 2008).

The Likelihood Method then assumes that prior to mortality the parasite distribution can be described by the distribution $g(x;\ \phi)$, which specifies the probability of a host having x parasites before mortality occurs. ϕ is a vector of parameters that describes the shape of this distribution. The probability of a host surviving with x parasites from infection until sampling is given by the host survival function $h(\text{survival};\ x,\ \theta)$ where θ specifies any additional parameters needed to define the host survival function.

With these two assumptions, we can define a distribution that gives the probability of having a parasite load of x parasites, conditional on host survival, $P(x|\text{survival})$. Using standard rules of conditional probability this distribution can be written as

$$P(x|\text{survival}) = \frac{P(\text{survival}|x) * P(x)}{P(\text{survival})} \quad (1)$$

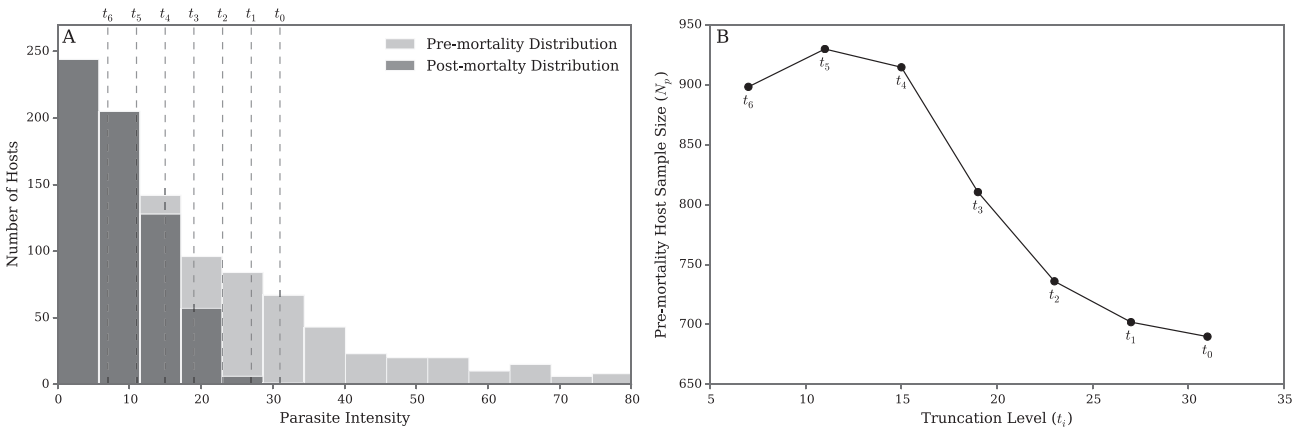

Fig. 1. A schematic representation of the iterative approach of the Crofton Method. (A) The light grey shows the pre-mortality distribution that the Crofton Method is trying to estimate from the dark grey post-mortality distribution. The Crofton Method proceeds by truncating the post-mortality data at different levels (t_i, e.g. $i = 0,\ldots,5$) and finding the pre-mortality host population size (N_p), pre-mortality mean parasite intensity (μ_p), and pre-mortality parasite aggregation (k_p) that best fit the truncated data. (B) The parameter N_p is then plotted against the truncation level t_i to determine if a "kink" occurs in the parameter values (Lester, 1984). This "kink" indicates that parasite-induced host mortality is occurring in the system. In the given example, parasite-induced host mortality is occurring in the system as visualised by the distinct "kink" at t_4.