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Research

Mechanistic models can reveal infection pathways from prevalence data: the mysterious case of polar bears *Ursus maritimus* and *Trichinella nativa*

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Parasites exhibit a diverse range of life history strategies. Transmission to a host is a key component of each life cycle but the difficulty of observing host–parasite contacts has often led to confusion surrounding transmission pathways. Given limited data on most host–parasite systems, flexible approaches are needed for disentangling the obscure transmission dynamics of these systems. Here, we develop a modelling framework for formally testing long-standing hypotheses regarding how the parasitic nematode *Trichinella nativa* is maintained at high prevalences in polar bear populations. We evaluated transmission from marine prey, from scavenging terrestrial carrion, from cannibalism and from scavenging on dead infected bears as possible pathways, and assessed their respective importance by comparing model-projected prevalences for each mechanism against observed total and age-specific population prevalences in the Southern Beaufort Sea polar bear subpopulation. Cannibalism and the scavenging on conspecifics have previously been assumed to be critical transmission pathways, but despite data scarcity, our model exposes these mechanisms as ineffective across a wide range of plausible parameter values. Instead, our analyses suggest that transmission from the consumption of infected marine prey, and in particular seals, can explain observed prevalence levels by itself, with other transmission pathways likely playing varying small contributing roles. Furthermore, our model suggests that transmission declines with bear age, perhaps due to age-dependent changes in diet or immunity. By formalising multiple transmission mechanisms in a unified, mathematical framework, we distilled several hypotheses to a likely main mode of *T. nativa* transmission to polar bears. The specifics of our model are tailored towards the *T. nativa*-polar bear system, but the approach is easily generalized; it provides a powerful, quantitative means for ecologists to explore competing hypothesis for parasite transmission and other difficult-to-observe animal interactions even in data-poor systems.

Keywords: cannibalism, host–parasite model, hypothesis testing, polar bear, scavenging, *Trichinella*, trophic transmission

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Introduction

Transmission between hosts is critical for parasites to complete their life cycle and maintain viable populations. Transmission pathways, however, vary widely between species and systems, and are often poorly understood (Bush et al. 2001). Parasites may be specialists, relying on a single host species for their survival and reproduction, or generalists, with the potential to exploit multiple definitive hosts. They may have simple life cycles that occur within one host, or more complex ones that involve trophic transmission, intermediate hosts, vectors or combinations thereof (Bush et al. 2001). Identifying transmission pathways and pinpointing key host species is critical for understanding disease dynamics (Ferrari et al. 2011, Antonovics 2017, Webster et al. 2017), but challenging given the number and diversity of parasite species and life history strategies, the difficulties of observing parasite transmissions in natural environments, and the poor understanding of most sylvatic host–parasite systems (Dobson et al. 2008, Buhnerkempe et al. 2015, Antonovics 2017).

Although transmission pathways can sometimes be revealed experimentally or through field observations, ethical barriers, technical limitations and/or a lack of resources frequently prevent such approaches (Antonovics 2017). Alternatively, mathematical models and computer simulations can provide cost-effective and flexible techniques for exploring uncertainties in transmission dynamics, for example, concerning transmission rates (Craft 2015), key hosts within a parasite life cycle, or reservoir hosts within a system (Fenton et al. 2015). Mathematical models can synthesise existing hypotheses and data into a common framework to provide a formalised method for hypothesis testing (McCallum 2001), including in data-poor systems (Molnár et al. 2013). Mathematical models have a long history of helping to unravel disease and host–parasite dynamics, from Kermack and McKendrick's (1927) initial exploration of disease progression to Anderson and May's (1978) and May and Anderson's (1978) seminal work on the dynamics of host–macroparasite systems. Models have exposed the influential roles that parasite species play in host populations and within ecosystems (Dobson and Hudson 1992, Lafferty et al. 2006), and provide a means for exploring remaining unknowns about the transmission dynamics of parasites between hosts (Zhang et al. 2007, Roberts and Heesterbeek 2013).

Here, we illustrate how a mathematical model can reveal likely transmission pathways from minimal demographic and epidemiological data, such as the prevalence of parasites among hosts (i.e. the proportion of the host population infected). Specifically, we consider a case study of polar bears and the parasitic nematode *Trichinella nativa* (Rausch et al. 1956, Fay 1960, Rah et al. 2005, Seymour et al. 2014a). *Trichinella nativa* is trophically transmitted, with larvae being reliant on carnivory to establish in a host (Dau and Barrett 1981, Kapel et al. 2003). Consequently, although *T. nativa* is present in many Arctic species, only obligate carnivores attain high prevalence (Rausch et al. 1956). The difficulty of observing host–parasite contacts, however, has led to inconclusive speculation regarding the main transmission pathways and key host species that drive the *T. nativa* sylvatic cycle (Seymour et al. 2014a).

Given the circumpolar distribution of polar bears and the high *T. nativa* prevalence (23–78%) and parasite burdens observed in all tested polar bear populations – including bears from Alaska, Norway, Greenland, Russia and Canada – (Forbes 2000, Rah et al. 2005, Seymour et al. 2014a), a popular hypothesis suggests that polar bears are key hosts in the Arctic, maintaining the parasite in their own population and providing the source of infection for other species (Rausch et al. 1956, Larsen and Kjos-Hanssen 1983, Taylor et al. 1985, Forbes 2000, Pozio 2001, Rah et al. 2005, Åsbakk et al. 2010). Consequently, both cannibalism and conspecific scavenging (i.e. the scavenging of polar bear remains by other polar bears) have endured as likely transmission pathways for maintaining *T. nativa* in polar bear populations (Rausch et al. 1956, Larsen and Kjos-Hanssen 1983, Taylor et al. 1985, Born and Henriksen 1990, Forbes 2000, Åsbakk et al. 2010). In addition, given the high prevalence of *T. nativa* in all terrestrial Arctic carnivores, it has also been hypothesised that polar bears may originally acquire their *T. nativa* infections when occasionally spending time on land and scavenging on terrestrial carrion (Connell 1949, Rausch et al. 1956, Madsen 1961, Seymour et al. 2014a). Alternatively, polar bears may simply become infected through their regular marine diet, but this hypothesis has drawn scepticism because of the low *T. nativa* prevalences found in these prey items. Seals constitute the staple diet of polar bears across the Arctic, but their *T. nativa* prevalence (0–1.9% in bearded and ringed seal surveys; Seymour et al. 2014a) is generally an order of magnitude lower than that of polar bears. Walrus may also be occasionally hunted or scavenged in some areas but are an unlikely suspect for maintaining high *T. nativa* prevalences in polar bears throughout the Arctic, given that they are not regularly consumed in every polar bear subpopulation (Thiemann et al. 2008a); that they are generally only hunted by large males (Thiemann et al. 2008a); and that their *T. nativa* prevalence only reaches high levels in areas where the walrus also consume seals (Fay 1960, Seymour et al. 2014b). Other potential carrion items, such as bowhead whales *Balaena mysticetus*, have not been reported to carry *T. nativa* at all (Rausch et al. 1956).

Here, we compare the respective importance of the four major transmission pathways that have been hypothesized in the literature for explaining the high *T. nativa* prevalence in polar bears: 1) transmission from marine prey (ringed seals, bearded seals, walrus); 2) transmission from scavenging

terrestrial carrion; 3) cannibalism; 4) conspecific scavenging. For this, we formalize each proposed transmission pathway in a mathematical model of parasite transmission and compare the model-projected prevalences for each mechanism against the observed total and age-specific prevalences in the Southern Beaufort Sea (SB) polar bear subpopulation (Durner et al. 2016).

Methods

Study system and data

Adult *Trichinella nativa* worms breed in mammalian intestines, where they release larvae for several weeks. Larvae penetrate striated muscle to encyst and form 'nurse cells' where they develop to the infective stage and remain dormant until the infected muscle tissue is consumed by another host (Dau and Barrett 1981). A protective, calcified layer allows encapsulated nurse cells to survive for decades, even in dead flesh (Dau and Barrett 1981, Kapel et al. 2003). Upon ingestion, stomach acid activates the nurse cells, releasing infective larvae that penetrate the mucosa of the small intestine and develop to their reproductive, adult stage (Dau and Barrett 1981). Because the host immune response cannot expel larvae once they are encysted, both individual parasite burdens and the proportion of bears that are infected tend to increase with age (Taylor et al. 1985, Born and Henriksen 1990, Rah et al. 2005, Åsbakk et al. 2010). Given that transmission of *T. nativa* occurs when hosts eat infected muscle tissue, it is the rates at which polar bears feed on different host species that ultimately determines *T. nativa* transmission rates.

We considered the *T. nativa* host–parasite dynamics in the SB polar bear subpopulation, where data on host demographics and on the prevalence of the parasite within polar bears (Rausch et al. 1956, Dau and Barrett 1981, Rah et al. 2005) and within their food sources (Rausch et al. 1956) were available from the same time period. The range of the SB subpopulation encompasses the southern portion of the Beaufort Sea (and adjacent land areas), up to about 70°N, from western Alaska to near Paulatuk, Northwest Territories, Canada (Regehr et al. 2006). This range is part of the so-called 'Divergent Sea Ice Ecoregion', where extensive sea ice forms annually along the shores but then retreats northwards each summer (Amstrup et al. 1986). During our study period (1950s–1990s) the SB subpopulation was initially overharvested, but then increased to a population size near 1800 individuals following regulation implementation after the 1973 Agreement on the Conservation of Polar Bears (Amstrup et al. 1986, Regehr et al. 2006).

Polar bear birth and survival rates were available from mark–recapture and telemetry studies for the SB subpopulation (Amstrup and Durner 1995, Regehr et al. 2007a, 2010). Transmission rates for *T. nativa* infections were estimated from polar bear consumption data of terrestrial foods, marine prey and conspecifics, derived from literature sources and personal communications (Table 1). Model predictions of population demographics, as well as of the total population and age class specific *T. nativa* prevalences, produced by the different transmission pathways were tested by comparison against empirical values. The SB stage distribution was available from capture summary statistics (Regehr et al. 2006) and a growth rate just above one has been suggested for most of our study period, i.e. once regulations were implemented (Amstrup et al. 1986, Regehr et al. 2006). Estimates of the total population prevalence of *T. nativa* in SB polar bears were available from two surveys conducted over 30 years apart (55% in 1949–1952, n = 104 (Rausch et al. 1956, Fay 1960), 55% in 1982–1999, n = 253 (Rah et al. 2005)), suggesting that *T. nativa* was near an endemic, steady state. Rah et al. (2005) also suggested that the prevalence of infection increases in SB bears with age, which has also been observed in other subpopulations (Born and Henriksen 1990, Åsbakk et al. 2010) (Table 1).

Model development

Our model tracks the number of uninfected (i.e. susceptible, S) and infected (I) bears in a population over time (Fig. 1, 2), stratified by age (i, in years), and accounting for infections arising both from interspecific (marine prey and terrestrial scavenging) and intraspecific (cannibalism and conspecific scavenging) transmission sources (Fig. 1, 2). Parasite transmission (individuals moving from S_i to I_i) and bear mortality are both modelled as continuous processes that can happen at any time during a year. The remaining demographic processes (births and aging) are implemented by stopping and re-initializing the model once a year, when all bears are moved up to the next age class ($i \rightarrow i+1$), new cubs are added as a birth pulse, and bears that have reached the modelled maximum age of 25 years (Kingsley 1979) are removed. We evaluated the degree to which each transmission pathway can create the observed *T. nativa* prevalence by running model components representing different transmissions mechanisms (pathways 1–4 in Fig. 1) in isolation from one another and in combination.

We used the following differential equations to represent the within-year infection dynamics in a polar bear population exposed to *T. nativa* through inter- and intraspecific transmission mechanisms (Fig. 2):

$$\underbrace{\frac{dS_i}{dt}}_{\text{Change in number of susceptible bears}} = \underbrace{-T_{1,2}(S_i)}_{\text{Transmission of infection via marine prey and terrestrial scavenging}} - \underbrace{T_3(S_i, I_1, \ldots, I_a)}_{\text{Transmission of infection via cannibalism}} - \underbrace{T_4(S_i, I_D)}_{\text{Transmission of infection via conspecific scavenging}} - \underbrace{C(S_i, S_1, \ldots, S_a, I_1, \ldots, I_a)}_{\text{Mortality from cannibalism}} - \underbrace{M(S_i)}_{\text{Regular mortality}} \quad \text{Eq. 1.1}$$

$$\underbrace{\frac{dI_i}{dt}}_{\text{Change in number of infected bears}} = \underbrace{T_{1,2}(S_i)}_{\text{Transmission of infection via marine prey and terrestrial scavenging}} + \underbrace{T_3(S_i, I_1, \ldots, I_a)}_{\text{Transmission of infection via cannibalism}} + \underbrace{T_4(S_i, I_D)}_{\text{Transmission of infection via conspecific scavenging}} - \underbrace{C(I_i, S_1, \ldots, S_a, I_1, \ldots, I_a)}_{\text{Mortality from cannibalism}} - \underbrace{M(I_i)}_{\text{Regular mortality}} \quad \text{Eq. 1.2}$$

$$\underbrace{\frac{dS_D}{dt}}_{\text{Change in number of uninfected dead}} = \underbrace{\sum_{i=1}^{a} M(S_i)}_{\text{Regular mortality across all susceptible bears}} + \underbrace{\frac{(g-1)}{g}\sum_{i=1}^{a} C(S_i, S_1, \ldots, S_a, I_1, \ldots, I_a)}_{\text{Mortality from cannibalism across all susceptible bears}} - \underbrace{R_1(S_D)}_{\text{Removal of dead via decay/non-polar bear scavenging}} - \underbrace{R_2(S_D, S_1, \ldots, S_a, I_1, \ldots, I_a)}_{\text{Removal of dead via conspecific scavenging}} \quad \text{Eq. 1.3}$$

$$\underbrace{\frac{dI_D}{dt}}_{\text{Change in number of infected dead}} = \underbrace{\sum_{i=1}^{a} M(I_i)}_{\text{Regular mortality across all infected bears}} + \underbrace{\frac{(g-1)}{g}\sum_{i=1}^{a} C(I_i, S_1, \ldots, S_a, I_1, \ldots, I_a)}_{\text{Mortality from cannibalism across all infected bears}} - \underbrace{R_1(I_D)}_{\text{Removal of dead via decay/non-polar bear scavenging}} - \underbrace{R_2(I_D, S_1, \ldots, S_a, I_1, \ldots, I_a)}_{\text{Removal of dead via conspecific scavenging}} \quad \text{Eq. 1.4}$$

where $S_i(t)$ and $I_i(t)$ are the numbers of susceptible and infected individuals of age i at day-of-the-year t (Jan 1: $t=0$; Dec 31: $t=365$), and $S_D(t)$ and $I_D(t)$ are the numbers of uninfected and infected dead bears on the landscape (Fig. 2). We assume an age specific per capita regular mortality rate, $M(S_i)=\mu_i S_i$ and $M(I_i)=\mu_i I_i$, that includes natural deaths and kills by humans, and that no bear can live past a maximum age a. Additional mortalities occur through cannibalism, $C(S_i, S_1, \ldots, S_a, I_1, \ldots, I_a) = \sum_{j=1}^{a} \omega_{ji}(S_j + I_j) S_i \frac{1}{N}$ and $C(I_i, S_1, \ldots, S_a, I_1, \ldots, I_a) = \sum_{j=1}^{a} \omega_{ji}(S_j + I_j) I_i \frac{1}{N}$, when a bear of age j cannibalises a bear of age i (at per capita cannibalism contact rate ω_{ji}). $N(t) = \sum_{i=1}^{25}(S_i(t) + I_i(t))$ is total population size at time t. Except for the relatively small impact of cannibalism on population growth via the additive mortality, we did not consider any density dependence nor a carrying capacity in our model, because we only needed to approximate the observed age structure and historical birth and death rates in order to be able to distinguish between pathways of *T. nativa* transmission.

The interspecific transmission rate, $T_{1,2}(S_i)=(\sigma_i+\nu_i)\gamma_i S_i$, for a bear of age i is the sum of the rates at which bears consume infected marine prey, σ_i, and infected terrestrial carrion, ν_i, multiplied with the probability that infection occurs following consumption, γ_i. Intraspecific transmission occurs when a bear kills and feeds on another bear (cannibalism; $T_3(S_i, I_1, \ldots, I_a) = \sum_{j=1}^{a} \omega_{ij}\gamma_i S_i I_j \left(\frac{1}{N}\right)$), or when a bear finds and feeds on a deceased bear (conspecific scavenging; $T_4(S_i, I_D) = c_i \gamma_i S_i I_D$). We assume cannibalism is a frequency-dependent process because cannibalism appears equally rare across populations, regardless of population density (Stirling 1974, Taylor et al. 1985). Conspecific scavenging is assumed to be density-dependent at a per capita contact rate, c_i, because polar bears are opportunistic scavengers, meaning that an increased availability of bear carcasses would likely increase consumption rates (Larsen and Kjos-Hanssen 1983, Lunn and Stirling 1985). Bears are moved to the 'Dead' class (S_D or I_D) when they die of regular causes or cannibalism and are removed from the landscape through conspecific scavenging $R_2(S_D, S_1, \ldots, S_a, I_1, \ldots, I_a) = \sum_{i=1}^{a} \frac{c_i(S_i + I_i)S_D}{g}$ and $R_2(I_D, S_1, \ldots, S_a, I_1, \ldots, I_a) = \sum_{i=1}^{a} \frac{c_i(S_i + I_i)I_D}{g}$, or through decay/scavenging by other species, $R_1(S_D)=\theta S_D$ and $R_1(I_D)=\theta I_D$. For cannibalism and conspecific scavenging, we accounted for the number of 'muscle meals' that a single bear carcass can provide on average, g, meaning that 1) we only consider those portions of a carcass that may be infected, 2) that one meal is removed from a cannibalised bear before its carcass is moved to the 'Dead' class and 3) that carcasses are removed from their 'Dead' class one meal at a time via conspecific scavenging.

Table 1. Demographic and epidemiological model parameters for the *T. nativa* transmission dynamics in the Southern Beaufort Sea polar bear population. All parameter values were determined from published literature, except where stated otherwise. Stage classes are cubs 'C' [0–1 years], yearlings 'Y' [1–2 years], subadults 'S' [2–5 years], adults 'A' [5–24 years], senescent 'O' [24–25 years]. Lower and upper bounds used for each parameter in the sensitivity analyses are shown in brackets. All rates are per capita. For calculated parameters, sources containing information incorporated into those calculations are preceded by the phrase 'This study using [#]'.

Parameter	Definition	Units	Value	Source
α	Birth rate	Cubs (solitary adult female)$^{-1}$×year^{-1}	0.2176	[1] [2] [3] [4]
μ_i	Regular mortality rate	Regular mortality×day^{-1}	O: 4.48×10^{-4} A: 8.23×10^{-5} S: 1.99×10^{-4} Y: 4.16×10^{-4} C: 1.18×10^{-3}	[2] [5] [6]
σ_i	Consumption rates of infected marine prey	Infected marine prey meal×bear^{-1}×day^{-1}	O,A,Y,C: 6.10×10^{-4} S: 2.77×10^{-4}	This study using [7] [8] [9] [10]
ν_i	Consumption rates of infected terrestrial carrion	Infected carrion meal×bear^{-1}×day^{-1}	O,A,S,Y,C: 1.61×10^{-5}	This study using [7] [8] [9] [11]
ω_{ij}	Cannibalism contact rates, where rows are stage-specific rates of cannibalising and columns are stage-specific rates of being cannibalised	Cannibalism meal× bear^{-1}×day^{-1}×10^{-5}	C: 0, 0, 0, 0, 0 Y: 0, 0, 0, 0, 0 S: 5.89, 3.53, 1.77, 0, 0.589 A: 4.71, 2.95, 2.95, 0, 1.18 O: 0, 0, 0, 0, 0 (C, Y, S, A, O)	This study using [7] [8] [11] [12]
c_i	Conspecific scavenging rates	Conspecific scavenging meal×bear^{-1}×day^{-1}	O,A,Y,C: 1.07×10^{-2} S: 0.84×10^{-2}	This study using [6] [7] [8] [11]
γ	Probability of infection given contact	–	O,A,S,Y,C: 0.70 (0.1; 1)	[13] [14]
θ	Rate of polar bear carcass decay	Carcass removed×day^{-1}	20^{-1} (10^{-1}; 120^{-1})	This study using [15]
g	Average number of muscle meals provided by one carcass	–	3 (1; 6)	This study using [15]
Observed prevalence data				
PL	Average prevalence level of *T. nativa* in SB polar bear subpopulation	–	55% in 1949–1952, n=104 55% in 1982–1999, n=253	[9] [16] [17]
PL_{stage}	Prevalence level of *T. nativa* in different stage classes of polar bears	–	Cubs and yearlings: 19.4%, n=93 Subadults: 40.9%, n=93 Reproductively mature adults: 57.4%, n=101 Prime reproductive adults: 73.0%, n=89 Old adults: 83.9%, n=56 Adults: 70.1%, n=246	[17]

[1] Regehr et al. 2006; [2] Regehr et al. 2009; [3] Hunter et al. 2007; [4] Van de Velde et al. 1985, Atkinsen and Ramsay 1995, Derocher 1999; [5] Amstrup and Durner 1995; [6] Regehr et al. 2007b; [7] Stirling and Øritsland 1995; [8] Kingsley 1979; [9] Rausch et al. 1956; [10] Dau and Barrett 1981; [11] Gormezano and Rockwell 2013; [12] Stirling 1974; [13] Nelson et al. 1966; [14] Kapel et al. 2003; [15] pers. comm. S. Amstrup and I. Stirling, 5/10/2018; [16] Fay 1960; [17] Rah et al. 2005.

The fully expanded differential equations (Eq. 1–4) are provided for convenience in Supplementary material Appendix 1 Eq. A1.

Model parameterization

In accordance with how polar bear data are generally reported in the literature, we assumed all age-dependent model parameters to be constant within five life history stages: cubs (C) [0–1 years old], yearlings (Y) [1–2 years], subadults (S) [2–5 years], adults (A) [5–24 years], senescents (O) [24–25 years]. We estimated daily instantaneous mortality rates (μ_i) from annual survival probabilities reported in Amstrup and Durner (1995) by assuming a constant hazard rate throughout the year, i.e. $\mu_i = -\ln\left(\text{annual survival}_i\right) \times \left(365 \text{ days}\right)^{-1}$ (Supplementary material Appendix 1 A1.A.i). Because survival data are only reported in the literature for females and

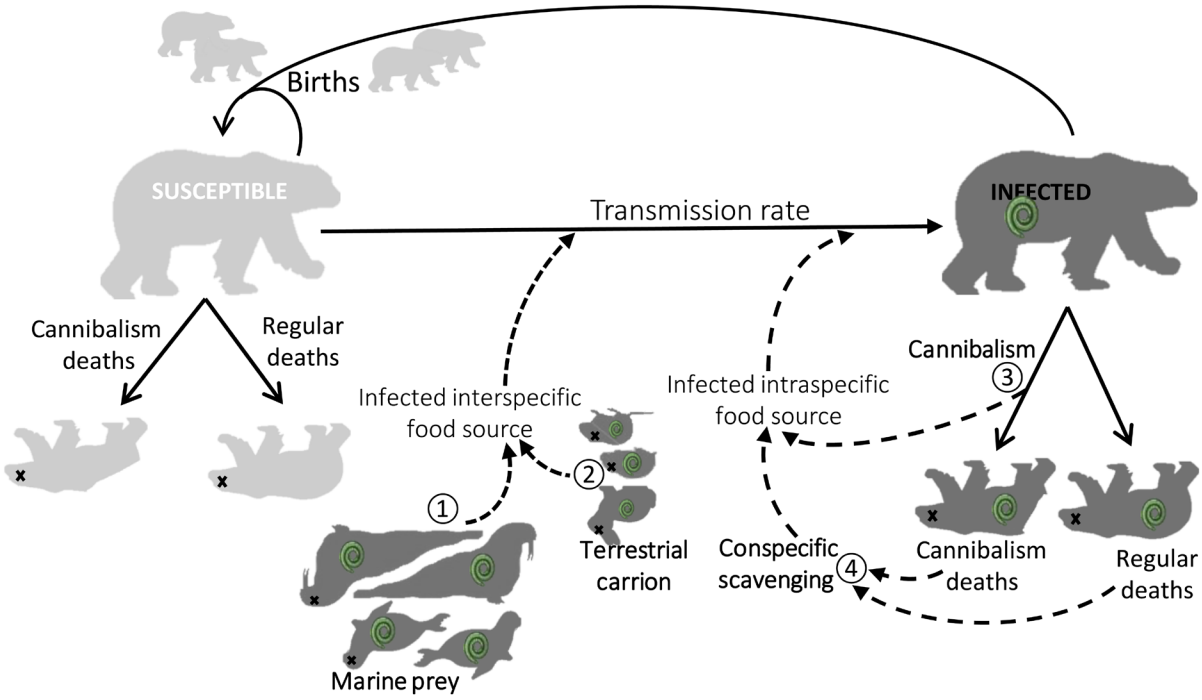

Figure 1. Schematic illustrating possible transmission pathways of *Trichinella nativa* to and among polar bears. Susceptible bears could become infected through any of the following four mechanisms: 1) consumption of infected marine prey (ringed/bearded seals and walrus); 2) consumption of infected terrestrial carrion; 3) killing and consuming an infected polar bear (cannibalism); and/or 4) scavenging a dead infected polar bear (conspecific scavenging). Of these, we refer to marine prey/terrestrial carrion as interspecific sources, and to cannibalism/conspecific scavenging as intraspecific sources. Reproduction and mortality rates, both from cannibalism and from regular sources, are assumed to be the same for both infected and uninfected bears.

dependent young given their mother's survival, we assumed equal adult mortality rates, regardless of sex or reproductive status, for our analyses, and that all dependent young are

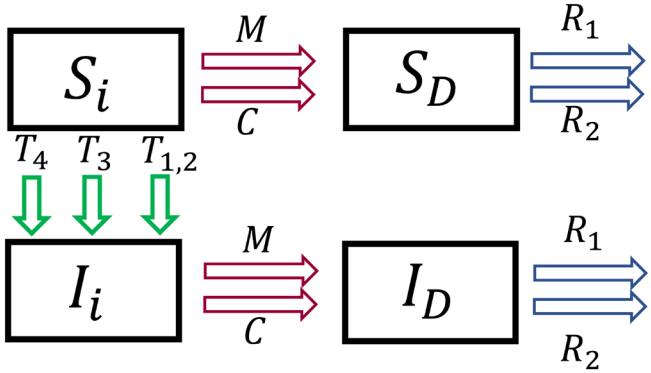

Figure 2. Simplified model flow chart illustrating pathways of *Trichinella nativa* transmission to and among polar bears. Susceptible bears (S_i) of age class *i* move to the infected bear class (I_i) by consuming infected marine prey or terrestrial carrion ($T_{1,2}$), killing and consuming an infected polar bear (T_3; cannibalism) or scavenging a dead infected polar bear (T_4; conspecific scavenging). Both susceptible (S_i) and infected bears (I_i) can move to their respective dead class (S_D, I_D) via regular mortalities (M) or cannibalism (C). Dead bears (SD, ID) are removed from the landscape through decay/ scavenging by other species (Ri) and through scavenging by polar bears (i.e. conspecific scavenging) (R2). Please note subscripts D, D, i, 2.

accompanied by their mother. Unobserved subadult mortality rates were estimated by scaling adult mortality rates according to the ratio of subadult-to-adult survival that was observed in later studies (i.e. during the 2000s; Regehr et al. 2009 their Table 3). The number of births each year was set as the number of solitary adult females, calculated from the sex (Regehr et al. 2006) and female reproductive status ratios observed in the SB (Hunter et al. 2007), multiplied by the probability of solitary females breeding (Regehr et al. 2009) and a mean litter size of two cubs (Velde et al. 1985, Atkinsen and Ramsay 1995, Derocher 1999) (Supplementary material Appendix 1 A1.A.ii). Immigration and emigration rates to the population are generally considered negligible (Amstrup et al. 1986) and were not included in our model.

Data informing the epidemiological components of our model are scarce, preventing accurate estimates for several parameters. Therefore, we estimated best-guess baseline values along with generous lower and upper bounds for each parameter (Table 1; Supplementary material Appendix 1 A1.B–D and A2) and subsequently explored the parameter space within these bounds to evaluate each transmission pathway's potential for maintaining *T. nativa* infections in polar bears.

For the transmission from marine prey, we first considered a baseline scenario where polar bears exclusively consume ringed and bearded seals, which are their primary prey in the SB region (Iverson et al. 2006, Thiemann et al. 2008b,

McKinney et al. 2017). We estimated consumption rates of infected seals (σ_i) from the average number of weaned seals consumed by an individual polar bear of a given age *i* (Stirling and Øritsland 1995) multiplied by the estimated *T. nativa* prevalence in SB seal populations (Rausch et al. 1956). We assumed that dependent cubs and yearlings encounter the same number of seals as their mothers and consume small portions of every catch, thus resulting in the same consumption rate of infected seals as in adults. Subadult seal consumption rates were estimated by scaling down observed adult consumption rates (Stirling and Øritsland 1995) allometrically with body mass (Supplementary material Appendix 1 A1.B).

Subsequently, we used sensitivity and scenario analyses to explore how deviations from our baseline assumptions about the diet may affect transmission rates and the *T. nativa* prevalence in polar bears. Specifically, we considered two scenarios: 1) reduced infections in seals, assuming that only subadult and adult seals may harbour *T. nativa*, but not freshly weaned pups that may not yet have had time to become infected; and 2) additional hunting or scavenging of non-seal marine prey, where we show the effect of adding one non-seal meal to each bear's baseline seal diet every year. We used walrus in these analyses to show the possible bounds of such effects, as walrus tend to carry the highest *T. nativa* prevalence [0.9%] (Fay 1960, Seymour et al. 2014a) among potential prey items (Supplementary material Appendix 1 A2). Beluga are sometimes reported in the diet of SB polar bears, but only one mildly infected whale has ever been found to carry *T. nativa* in hundreds of samples (Forbes 2000).

Consumption rates of infected terrestrial carrion (ν_i) and contact rates for cannibalism (ω_{ij}) and conspecific scavenging (c_i) are difficult to estimate due to a lack of quantitative data on these interactions in the SB subpopulation. Because these infection pathways likely vary in intensity throughout the year, probably reaching their maxima during summer months when sea ice is at its minimum and bears are fasting on land or on pack ice (Cherry et al. 2009, Rode et al. 2015), we turned to the summer fasting period of the well-studied Western Hudson Bay (WH) population for parameter estimation guidance (Stirling et al. 1999). Specifically, we used diet composition data from faecal samples that were collected during the summer months in 2006–2008 (Gormezano and Rockwell 2013) to estimate rates of terrestrial scavenging and intraspecific interactions during a time of maximal food stress (Lunn and Stirling 1985, Parks et al. 2006). These estimates provide a reasonable upper bound for corresponding parameters in the SB subpopulation (Supplementary material Appendix 1 A1.C–D), given that the ice-free period is shorter in SB than in WH (Stern and Laidre 2016) and that many SB bears remain on the sea ice their entire lives (Atwood et al. 2016). To translate Gormezano and Rockwell's (2013) diet composition data into rates of terrestrial scavenging, we assumed that each bear consumed on average 10 kg of food over the course of each summer during their study period, independent of age (i.e. $\nu_i = \nu$). Given that WH bears generally feed very little while on shore (Lunn and Stirling 1985, Rode et al. 2015), these are again generous assumptions that likely further overestimate the contribution of these infection pathways to observed *T. nativa* prevalences in polar bears. Rates of infection via terrestrial scavenging were obtained by partitioning our assumed food intake into different prey species according to Gormezano and Rockwell's (2013) diet composition data, correcting for potential differences in prey diversity among WH and SB bears (Supplementary material Appendix A1.C), and accounting for observed *T. nativa* prevalences in each species (Supplementary material Appendix 1 A1.C).

Rates of cannibalism and conspecific scavenging were estimated based on the bears' annual total food intake and the diet composition data, which show that on average 2.4% of an individual's onshore diet came from consuming (parts of) other polar bears (Gormezano and Rockwell 2013). For our baseline, we assumed that 2% of the annual food intake comes from conspecific scavenging, and only a small portion (0.1%) comes from cannibalism (Supplementary material Appendix 1 A1.D.i), because higher cannibalism rates would mean that each bear kills and cannibalizes at least one other bear each year, thus driving the population to extinction (Supplementary material Appendix 1 Fig. A5). We structured both types of conspecific intake by stage, scaling conspecific scavenging (Supplementary material Appendix 1 A1.D.iii) and cannibalism rates (Supplementary material Appendix 1 A1.D.ii) according to the total food intake rates of each stage and further partitioning out cannibalism rates according to observations on which stages are likely perpetrators and which are likely victims. Adult bears, for example, are the most likely group to engage in cannibalism, while cubs and yearlings are the most likely to be cannibalized (Supplementary material Appendix 1 A1.D.ii).

The final infection parameters estimated were the decay rate (θ), the number of muscle meals provided by a polar bear carcass (g) and the probability of host infection given contact with a *T. nativa* infected food source (γ_i). The scarcity of food available on the Arctic landscape suggests high scavenging rates by other species, implying that carcasses would not remain available for very long on the landscape [$\theta = (20\ \text{day})^{-1}$]. Furthermore, shifting of ice and open water during the freeze-up and break-up seasons suggest that carcasses will not remain past either season, so we set an upper boundary of [$\theta = (120\ \text{day})^{-1}$]. The solitary lifestyle and large home range size (Amstrup et al. 2000) of SB polar bears suggest that meal sharing carcasses will be rare, especially given that most mortalities are young, small bears (Amstrup and Durner 1995, Regehr et al. 2007a). Moreover, healthy adult polar bears preferentially consume fat over protein, meaning that only subadults and nutritionally stressed adults would regularly consume the muscle from a bear carcass that is critical for *T. nativa* transmission. We therefore assume that a carcass will provide only three meals ($g = 3$) on average but also explore an upper bound of $g = 6$ (Table 1). Experimental studies on the probability of infection given contact with *T. nativa* (γ_i) show varied

resistance between mammalian species (Nelson et al. 1966). Experimental infections in Arctic seals suggest high infectivity, such that even a small number of ingested larvae could result in a substantial number of larval cysts (Kapel et al. 2003). We assumed a relatively high baseline value for the probability of infection given contact ($\gamma = 0.7$), but also explored a generous range from $\gamma = 0.1$ to $\gamma = 1$. We assumed throughout that γ is age-independent (i.e. $\gamma_i = \gamma$).

Model analysis

We used the model to evaluate the plausibility of the four hypothesized transmission pathways by comparing observed stage-specific and population-averaged prevalence levels (Table 1) against model projections for each proposed transmission mechanism, both in isolation from one another and in various combinations. For our baseline simulations, we initialised the model at stable age distribution with a low population size of 300 polar bears and ran it for 100 years. This timeframe ensured both that prevalence levels had stabilized and that the maximum number of bears never exceeded 4500, thus preventing inflated transmission from (density-dependent) conspecific scavenging in an unrealistically large population. Subsequently, we also explored a scenario that mimics the pre-1973 overharvest (and thus, an increased carcass availability for conspecific scavenging on the landscape) by initializing our model with approximately 2000 bears, and running the first 20 years of our simulation with increased bear mortality rates (Supplementary material Appendix 1 Fig. A6).

We recognize that the demographic components of our model do not include some of the complexities surrounding a polar bear's life (e.g. cub deaths when the mother dies; Regehr et al. 2009), but the model nevertheless accurately captures the key dynamics that are necessary for understanding the *T. nativa*-polar bear host–parasite dynamics. The model-predicted stable stage distribution [13% cubs, 8% yearlings, 18% subadults and 61% adults] (Supplementary material Appendix 1 Fig. A1) compared favourably to the observed stage structure of the SB subpopulation during the assessed period [12% cubs, 9% yearlings, 19% subadults, 60% adults] (Regehr et al. 2006), and so did our estimated population growth rates: depending on cannibalism levels, projected population growth rates ranged from 0.994 to 1.018, which is consistent with the relatively stable population size during most of our study period (Amstrup et al. 1986, Regehr et al. 2006). Model outputs were generally robust to changes in initial conditions (Supplementary material Appendix 1 Fig. A2, A3), but with prevalence levels stabilizing slightly more slowly in simulations of intraspecific pathways due to their direct dependence on population density (Eq. 1; Supplementary material Appendix 1 Fig. A3d, f). Sensitivity analyses that varied all parameters between generous lower and upper bounds were used to evaluate the robustness of all conclusions (Table 1; Supplementary material Appendix 1 A1.B–D, A2).

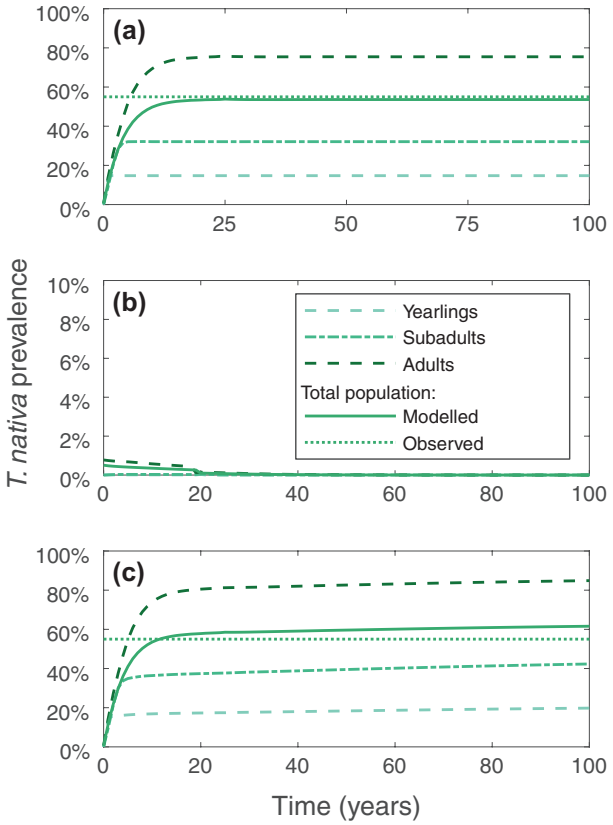

Figure 3. Model simulations of *T. nativa* prevalence in polar bears when only interspecific sources of transmission are considered (a), when only intraspecific sources of transmission are considered (b) and when all sources of transmission are considered in combination (c). The temporal changes in *T. nativa* infection prevalence among yearlings (dashed lightest green), subadults (dot-dashed light green) and adults (dashed darkest green), are depicted immediately following each year's birth pulse. New-born cubs are parasite-free and are therefore not shown. Horizontal dotted green lines show the observed total *T. nativa* prevalence in the SB polar bear subpopulation, solid green lines show the modelled total prevalence. All simulations were initialised with 300 bears at stable stage distribution (as calculated in the absence of cannibalism) and one infected adult. All model parameters are set at their baseline values (Table 1).

Results

Trichinella nativa prevalence dynamics

Model simulations show an increase in *Trichinella nativa* prevalence with age (Fig. 3), which is consistent with field observations (Rah et al. 2005). Transmission from interspecific sources alone (Fig. 1: pathway 1 and 2) results in a total population prevalence of 53.6% in our baseline scenario (Fig. 3a), closely approximating the observed prevalence of 55% (Rausch et al. 1956, Dau and Barrett 1981, Rah et al. 2005). When considering transmission from seal predation in isolation (Fig. 1: pathway 1), the model suggests a total prevalence of 52.9%, while terrestrial scavenging (Fig. 1: pathway 2) in isolation would only produce a 3.3% prevalence

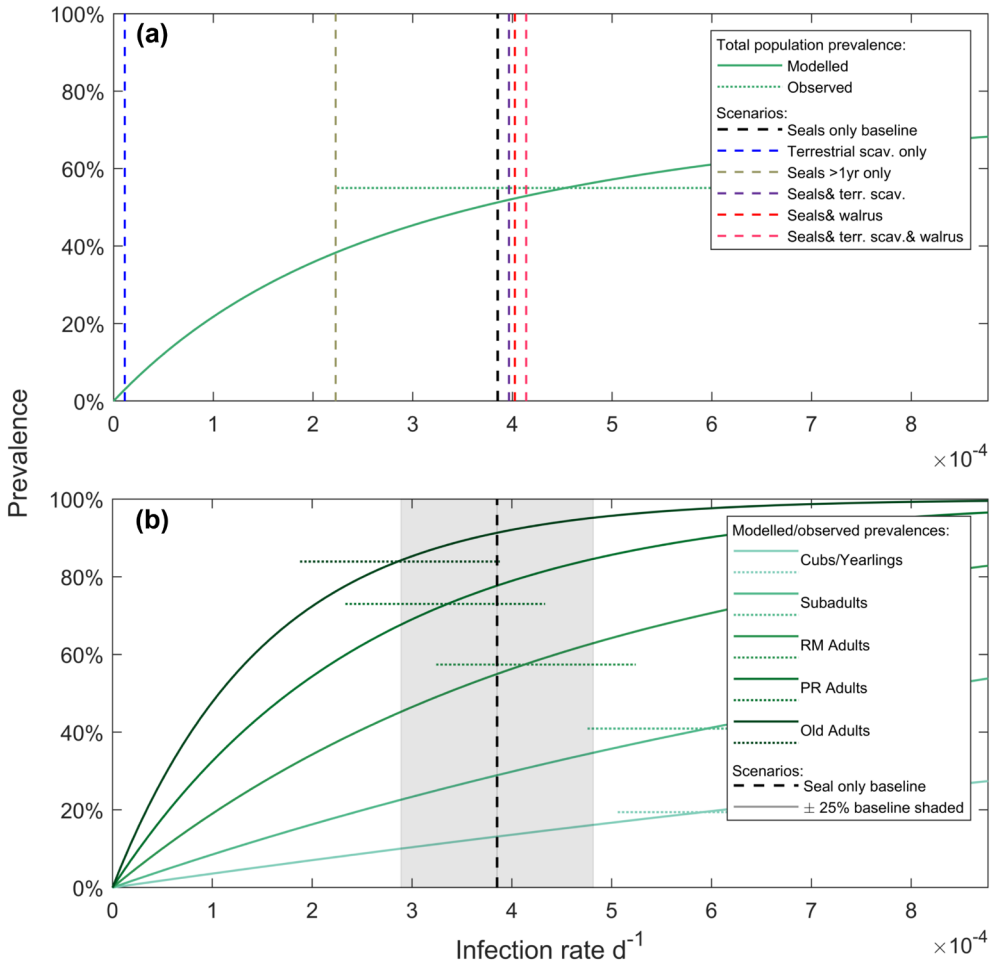

Figure 4. Sensitivity of the model-predicted *T. nativa* prevalence in polar bears to the parameters determining infection rates from interspecific transmission sources. Infection rate is the product of the contact rate with infected marine prey and carrion ($\sigma_i + \nu_i$) and the probability of infection given contact (γ). (a) Total population prevalence (solid green) as a function of interspecific infection rate following model stabilisation. Vertical lines indicate infection rates from, terrestrial scavenging by itself (dashed blue), seal eating only baseline (dashed black), adding one walrus to each bear's seal diet (dashed red), and combinations of these pathways (dashed purple being terrestrial scavenging and seals; dashed pink being seal baseline with an additional walrus meal and terrestrial scavenging) . The vertical dashed grey line assumes that only seals that are at least one year old can harbour *T. nativa* infections. All infection rates are calculated as the weighted average of the stage specific rates assuming a stable stage distribution. Horizontal dotted lines indicate observed total population prevalence (55%) in SB polar bears during 1949–1952 (Rausch et al. 1956, Fay 1960) and 1982–1999 (Rah et al. 2005). (b) Stage specific prevalence as a function of interspecific infection rate following model stabilisation, with cub and yearling prevalence combined into one weighted average value (solid lightest green), subadults (solid light green) and the adult stage separated into reproductively mature (RM) (5–9 years old) (solid dark green), prime reproductive (PR) (10–14 years) (solid darker green) and old (15–25 years) (solid darkest green) adults to allow comparisons with literature values. Horizontal dotted lines indicate the observed prevalence for each stage from a 1982–1999 survey that included multiple polar bear subpopulations but focused on the SB (Rah et al. 2005). Vertical dashed black line shows the baseline infection rate from seal eating only estimated as a weighted average of the stage specific rates, and the shaded area covers infection rates ±25% of this value.

(Fig. 4a; Supplementary material Appendix 1 Fig. A4a). The two transmission pathways are subadditive, meaning that the sum of the prevalences suggested by each pathway independently [56.2%] is greater than the prevalence that is obtained when pathways are run together [53.6%]. Transmission through intraspecific sources (Fig. 1: pathway 3 and 4) alone, by contrast, cannot explain the observed prevalence levels, as model runs stabilise under 0.0005% prevalence (Fig. 3b). Conspecific scavenging alone (Fig. 1: pathway 4) results in a slightly higher prevalence [0.0006%] and the parasite effectively dies out with cannibalism alone (Fig. 1: pathway 3) (Supplementary material Appendix 1 Fig. A4). When considering all four sources of *T. nativa* transmission together, the model suggests a superadditive *T. nativa* prevalence of 61.6%, (Fig. 3c). Similar patterns are observed when comparing age-specific model outputs to the observed age-specific prevalences: interspecific sources create prevalence levels [Cubs and yearlings: 14.8%; Subadults: 32.1%; Adults: 75.5%] (Fig. 3a) close to those observed [Cubs and yearlings: 19.4%; Subadults: 40.9%; Adults: 70.1%] (Rah et al.

2005), whereas intraspecific sources alone cannot maintain significant *T. nativa* prevalence levels [Cubs and yearlings: 0.00005%; Subadults: 0.0001%; Adults: 0.0007%] (Fig. 3b), and all transmission mechanisms together create superadditive prevalence levels [Cubs and yearlings: 19.8%; Subadults: 42.3%; Adults: 84.9%] (Fig. 3c).

Scenario and sensitivity analyses

Sensitivity analyses reveal that *T. nativa* prevalence is highly dependent on transmission from marine prey (Fig. 4a, 5). Our estimated baseline value for the *T. nativa* transmission rate from seal eating accurately captures the observed prevalence at the population level and for reproductively mature (RM) adults, but systematically over- and under-estimates prevalence for older and younger age classes, respectively (Fig. 4b). Increasing or decreasing the baseline transmission rate consequently improves model predictions for younger or older bears, respectively, but worsens predictions for the other end of the age spectrum (Fig. 4b).

Assuming that only seals older than a year harbour *T. nativa*, but not freshly weaned pups, reduces the predicted population prevalence to 39% (Fig. 4a). Adding a walrus meal to the seal diet increases total population prevalence by 1.0%, and by 1.8% when also combined with scavenging on terrestrial species (Fig. 4a).

Model predicted *T. nativa* prevalence is not sensitive to changes in either of the three other transmission mechanisms near their baseline values. Our baseline rate for terrestrial carrion scavenging would need to be increased by several orders of magnitude to predict real-world prevalence levels accurately (Fig. 4a), and intraspecific pathways are unable to create prevalence levels near those observed regardless of the contact rates for cannibalism and conspecific scavenging (Fig. 3b, 5). The initial overharvest of polar bears may have caused a temporary spike in *T. nativa* infections that were acquired through the increased conspecific scavenging opportunities at the time, but the signal of this spike disappeared after one to two bear generations in our simulations, when again seals were required to maintain high *T. nativa* prevalences (Supplementary material Appendix 1 Fig. A6).

Prevalence from conspecific scavenging was not sensitive to changes in the decay rate of corpses, even if decay rate was decreased by 600% relative to our baseline. At the extreme end of the parameter space for probability of infection given contact ($\gamma = 1$) (Fig. 5a) and the number of muscle meals a carcass provides ($g = 6$) (Fig. 5b), prevalence from conspecific scavenging increased to up to 0.40% but still remained far below the observed prevalence in SB [55%].

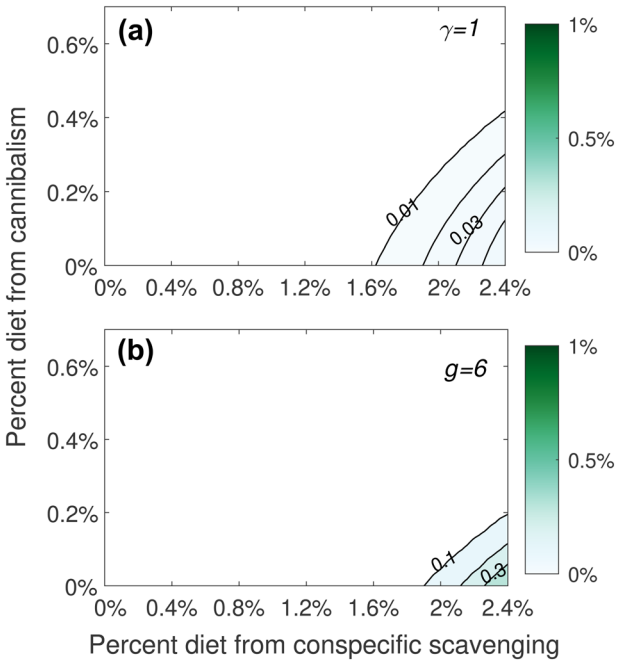

Figure 5. Sensitivity of the model-predicted *T. nativa* total prevalence in polar bears to the parameters determining infection rates from intraspecific transmission sources. The full range of conspecific scavenging and cannibalism rates is explored, from making up zero percent of a bear's diet to their respective biologically feasible upper bounds. Panel (a) shows the prevalence assuming the probability of infection given contact is at its maximum ($\gamma = 1$), and panel (b) shows the prevalence assuming the number of muscle meals that a dead bear provides is at its maximum ($g = 6$). All other model parameters are set at the baseline values from Table 1. Contour lines show the prevalence level of the total population following model stabilisation.

Discussion

Resolving specific pathways of a parasite's transmission is critical for understanding disease dynamics and for examining potential avenues for disease control (Ferrari et al. 2011, Antonovics 2017). Here, we illustrated an approach for disentangling difficult-to-observe parasite transmission pathways from easily collected data on the net result of these transmissions. We determined the most plausible transmission pathway responsible for the high prevalence of *Trichinella nativa* in the SB polar bear subpopulation by synthesizing existing prevalence and demographic data in a mechanistic modelling framework. The low prevalence of *T. nativa* in seals has led to the broad and persistent underestimation of seal-predation as the main transmission pathway to polar bears in favour of less parsimonious mechanisms (Forbes 2000, Seymour et al. 2014a). Indeed, previous 'back-of-the-envelope calculations' suggested conspecific scavenging and cannibalism as the main transmission sources (Larsen and Kjos-Hanssen 1983, Taylor et al. 1985), which, to this day, has overshadowed Fay's original hypothesis that seals drive *T. nativa* dynamics in the Arctic (Fay 1960). Our results indicate that transmission from marine prey, and in particular from seals, can by itself produce the *T. nativa* prevalence patterns that are observed in SB polar bears, whereas terrestrial scavenging, cannibalism and conspecific scavenging cannot, even when parameters are

increased to the extreme end of the biologically possible spectrum. Our modelling approach shows that the long lifespan of polar bears provides ample opportunity to encounter an infected seal, which, coupled with the lack of recovery from *T. nativa*, creates a high prevalence in the population. Due to the limited time spent on land, terrestrial scavenging opportunities are rare and cannot, by themselves, create the consistently high prevalence levels that are observed in the SB subpopulation, and also only marginally increased *T. nativa* prevalences when added to the seal diet (Fig. 4a). The same is true for acquiring infections from walrus, which are generally only hunted by large males (Thiemann et al. 2008b) and also do not regularly show up in fatty acid analyses of diet (Iverson et al. 2006, Thiemann et al. 2008b, McKinney et al. 2017). Because the polar bears' consumption rate of seals is much higher, those infected via terrestrial scavenging or from walrus would have eventually been infected from seals, explaining why these infection pathways are subadditive.

Ultimately, it is the diet composition of polar bears combined with the *T. nativa* prevalences of each prey species that determines the prevalence of *T. nativa* infections in polar bears. Variation exists in both of these factors among polar bear subpopulations (Thiemann et al. 2008b), and this will likely contribute to observed among-subpopulation differences in *T. nativa* prevalence as well. Polar bears in Foxe Basin, for example, have a broader diet than SB bears that also includes Atlantic walrus (Thiemann et al. 2008b). In contrast to their Pacific counterparts, these walruses much more frequently consume seals, and thus also have a much higher *T. nativa* prevalence (up to 40.6%) (Fay 1968, Seymour et al. 2014a) – likely because of the same mechanism of parasite accumulation that was demonstrated here for polar bears. The exact contributions of each prey species to polar bear *T. nativa* prevalence would have to be evaluated separately for each subpopulation, but our broad conclusions highlighting the importance of seals are unlikely to vary majorly. In fact, even in the case outlined above, seals are critical for explaining *T. nativa* infections in polar bears; it is just that some of the worms might be acquired indirectly via walruses that previously ate seals (Fay 1960, Seymour et al. 2014b).

The modelling approach revealed that cannibalism is an ineffective mechanism and may even hinder transmission through conspecific scavenging. Given the density dependence of conspecific scavenging (cf. Eq. A1), any decrease in population size, such as losing a bear to cannibalism, would reduce transmission via conspecific scavenging. Transmission from conspecific scavenging, by contrast, may be increased during periods of high carcass availability, which may occur at high population densities, but also during periods of decline, such as during the overharvest of the 1950s/60s (Supplementary material Appendix 1 Fig. A4, A6), or through recent sea ice loss induced survival declines (Hunter et al. 2010, Bromaghin et al. 2015). Because such infection spikes are short-lived and well below the observed high prevalence, however, the background infection from seal-eating remains required for explaining observed patterns. Indeed, if a population already maintains a high parasite prevalence, the odds that a conspecific meal will cause an infection increases, meaning that intraspecific modes of transmission become more effective when considered in combination with the interspecific pathways, thus creating the simulated superadditive increases in prevalence (cf. also Rudolf and Antonovics 2007). Although intraspecific pathways allow for secondary transmission of *T. nativa* among polar bears, they are poor candidates for the primary transmission pathway.

Our model closely approximates total population prevalence based on transmission from seals, but systematically under- and over-estimates prevalence in younger and older bears, respectively. These results suggest that the force of infection for an individual might decline with age (cf. also Åsbakk et al. 2010), perhaps because of increasing immune resistance (Grencis et al. 2014), and thus a decreasing probability of infection given contact (γ), as individuals age. In addition, diet switching could be an alternative or complementary explanation. As bears age, they become better hunters and/or steal kills from younger bears (Amstrup and Durner 1995), allowing them to preferentially eat fat and avoid potentially infected muscle (Stirling and McEwan 1975). Younger, less adept bears are often forced to exploit leftovers, even from their own kills (Stirling and McEwan 1975), which likely results in greater consumption of *T. nativa* encysted in the muscle tissue. Additionally, younger bears are growing and have therefore higher protein requirements than older bears, so they may even preferentially take muscle meals (Garlick et al. 1975, Kyriazakis et al. 1991). Each scenario would cause the contact rate with infected seal muscle (σ) to decrease with age, thus decreasing the infection rate with age. To disentangle the cause of age-dependent infection rates, we suggest that studies focus on determining the probability of infection given contact over an individual's lifetime. For example, captive polar bear feeding trials could be used to determine if selection for fat or protein changes with age (Best 1985, Dyck and Morin 2011). Additionally, observational studies on the use of seal-kills by wild polar bears across ages could determine realised, age-dependent contact rates with infected muscle tissues. Indeed, if the respective contributions of immunity and diet changes to the observed age-specific infection patterns could be identified, it might be possible to use parasitism levels to identify diet changes (Valtonen et al. 2010) in polar bear populations, for example, due to the ongoing loss of sea ice habitat (Amstrup et al. 2006, Rode et al. 2015, Atwood et al. 2016). Such approaches can complement and improve the accuracy of traditional stable isotope and fatty acid methodologies (Sinisalo et al. 2006) but also offer additional sampling scope, for example by involving hunters and communities in the search for parasites (Brook et al. 2009).

Currently, climate-change-induced sea ice loss has been decreasing the time when SB bears can hunt for seals, and the lengthening fasts have led to declines in body condition, reproduction, survival and abundance (Rode et al. 2014, Bromaghin et al. 2015, Atwood et al. 2016), as well as to more SB bears scavenging ashore than historically (Rode et al. 2014, Atwood et al. 2016). Such effects are

expected to worsen under ongoing climate change (Stirling and Derocher 2012), and we expect that hungry bears will be less discriminatory in their feeding habits, consuming both fat and muscle from seal kills (Stirling 1974) and increasing intraspecific feeding events (Amstrup et al. 2006). Both mechanisms would increase the contact rate with *T. nativa*, thus increasing parasite prevalence and the energy drained to parasitism within an already energetically stressed population (Liu et al. 2005, Worthington et al. 2013).

Most helminth parasites have life cycles that span across multiple trophic levels (Benesh et al. 2014). In the case of the nematode *T. nativa*, our model suggests that lower trophic levels are a critical component of its sylvatic cycle in the Arctic, thus emphasising the need to explore transmission pathways across the entire food web (cf. also Lafferty et al. 2006). For example, encapsulated *T. nativa* typically remain infectious to vertebrate hosts after consumption by amphipods, suggesting that Arctic detritivores might play a key role in maintaining the sylvatic *T. nativa* cycle (Fay 1968). Furthermore, experimental studies have shown that some bird species that consume infected meat are able to shed a decapsulated larval form that remains viable when consumed by lower level invertebrate hosts such as molluscs and crustaceans (Odoevskaya et al. 2013). These transport hosts are then able to transmit the parasite to higher trophic levels (Odoevskaya et al. 2013). As the decapsulated portion of the *T. nativa* life cycle has largely been ignored in previous discussions of the *T. nativa* dynamics, future studies should focus on interactions of the parasite with hosts at lower trophic levels so to allow complete characterization of this parasite's life cycle and transmission pathways.

Given the influence and pervasiveness of parasites across all ecosystems, it is critical to unravel their transmission pathways and means of persistence (Lafferty et al. 2006). Models are particularly useful for this purpose, highlighting system components that are data-poor and emphasising components that a system is sensitive to, while also providing formalised frameworks for hypothesis testing (Restif et al. 2012, Sures et al. 2017). Models allow evaluating the plausibility of hypothesized but difficult-to-observe transmission pathways by examining a pathway's influence on measurable phenomena, such as parasite prevalence. Such approaches have, for example, also been used in invasion biology to disentangle the effects of spatial variation in invasion success (Walter et al. 2015), and determine potential drivers of heterogeneity in rapidly spreading populations (Brown et al. 2013). Mortality, like prevalence, is another measurable output that is the net result of underlying processes, and has for example been used to determine the underlying mechanisms driving historic plague dynamics (He et al. 2011). As such, we suggest that the utility of our approach – synthesizing existing data in mechanistic models to explore the feasibility of hypothesized drivers of observed phenomena – extends beyond parasite ecology. It is valuable for studying complex systems where key data are unavailable to shed light on competing hypotheses of possible system drivers, either due to a lack of knowledge on what the key data are or logistical and ethical barriers to their collection.

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Author contributions – PKM and SRP conceived the study. All authors contributed to the development and parameterization of the model. SRP, KB and JSVS performed and analysed model simulations. All authors contributed to the writing of the manuscript.
Conflicts of interest – The authors have no conflicts to declare.

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Supplementary material (available online as Appendix oik-07458 at <www.oikosjournal.org/appendix/oik-07458>). Appendix 1.

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