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Conservation biology

Age of first infection across a range of parasite taxa in a wild mammalian population

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Newborn mammals have an immature immune system that cannot sufficiently protect them against infectious diseases. However, variation in the effectiveness of maternal immunity against different parasites may couple with temporal trends in parasite exposure to influence disparities in the timing of infection risk. Determining the relationship between age and infection risk is critical in identifying the portion of a host population that contributes to parasite dynamics, as well as the parasites that regulate host recruitment. However, there are no data directly identifying timing of first infection among parasites in wildlife. Here, we took advantage of a longitudinal dataset, tracking infection status by viruses, bacteria, protists and gastro-intestinal worms in a herd of African buffalo (*Syncerus caffer*) to ask: how does age of first infection differ among parasite taxa? We found distinct differences in the age of first infection among parasites that aligned with the mode of transmission and parasite taxonomy. Specifically, we found that tick-borne and environmentally transmitted protists were acquired earlier than directly transmitted bacteria and viruses. These results emphasize the importance of understanding infection risk in juveniles, especially in host species where juveniles are purported to sustain parasite persistence and/or where mortality rates of juveniles influence population dynamics.

1. Introduction

Infection risk varies with age, particularly in mammalian hosts [1–6]. Newborns have underdeveloped adaptive immune systems and are assumed to be more susceptible to infection [3]. However, age structure of disease dynamics is not generalizable across parasite taxa (encompassing macro- and micro-). Mothers may transfer maternal antibodies to their offspring [2], where, if the antibodies provide complete protection, they may sharply decrease parasite susceptibility. This maternal protection depends on host and pathogen type [7,8] but is reported to last anywhere from approximately 3 [9] to 8.5 [10] months. However, effective maternal immunity necessitates that parasite resistance be mediated through a humoral response [3], which is not effective for all parasites [11]. Additionally, infection risk is not solely dependent on host immunity, but also on exposure likelihood. Exposure likelihood varies with parasite transmission mode [12], with various components of transmission sensitive to different abiotic and biotic environmental factors (e.g. vector abundance changes with rainfall [13]; parasite viability changes with moisture and humidity [14,15]; host contact rate changes with food abundance

[16]). As such, exposure likelihood may undergo temporal fluctuations that are synchronous or asynchronous with birthing seasons and across parasite taxa, depending on parasite biology. Thus, disparate host immune responses may combine with parasite exposure likelihood to drive differences in the relationship between age and infection risk across parasite taxa.

Determining the age at which animals first acquire infections provides important insight into ecosystem disease dynamics in two ways. First, knowing the age at which animals are at the risk of infection facilitates the identification of age classes that contribute to parasite spread and persistence. Indeed, estimating the force of infection can often be improved by including age structure into the model, accounting for variance among age classes in susceptibility and infectivity [17]. Second, the age at which animals are first at the risk of infection by particular parasites defines the diseases that impact recruitment to subsequent age classes (e.g. through mortality [18]). For many wildlife species, young animals represent the most vulnerable life stage [19], and population performance depends critically on parameters related to recruitment of juveniles into the adult age group [20]. Differences in the timing of infection risk (i.e. when animals are most likely to first be infected) could drive differences in the ability of particular parasites to influence recruitment and, ultimately, population health.

Despite the importance of elucidating how parasite communities are sub-structured by host age, the timing of infections across parasite taxa have not been evaluated explicitly. Although the wildlife studies of hosts and their parasite communities have the advantage of monitoring the natural occurrence of infections [5], their often cross-sectional design limits the ability to pinpoint the timing of first infection [2]. In the absence of longitudinal data, researchers have used age–prevalence curves to approximate the timing of first infection [21]. However, this indirect measure of timing of infection can be confounded by recovery and mortality. We had the opportunity to interrogate a longitudinal dataset on a herd of wild African buffalo, *Syncerus caffer*, and directly measure the incidence of a suite of parasite taxa in the same cohort of hosts, facilitating comparison of infection risk and parasite communities across taxa. We used this dataset to ask: how does median age of first infection differ between parasite taxa?

2. Material and methods

(a) Study site

African buffalo included in this study were located in a 900-ha enclosure within the Kruger National Park (KNP) a 19 000 km^2 preserve, located in northeastern South Africa (S 24 23′ 52″, E 31 46′ 40″) (electronic supplementary material, S1). The enclosure was entirely within KNP. Numerous other wild animals typical of the ecosystem (e.g. giraffe, zebra, warthogs, small mammals and small predators) were present, excluding mega-herbivores (rhino, hippo, elephant) and large predators (lion, leopard). Study animals grazed and bred naturally and found water in seasonal pans and man-made (permanent) water troughs. The KNP experiences pronounced seasonal variation in rainfall and temperature [22]. We condensed patterns into a binomial seasonal variable: hot, wet season (October–March) and warm, dry season (April–September). In extremely dry conditions, supplemental grass and alfalfa hay was supplied. At any given time, depending on births and deaths, the herd consisted of around 65 buffaloes.

(b) Health and infection status

The herd was a sampled at two- to three-month intervals totaling five time points per year (immobilization methods outlined in [23]) from February 2015 to June 2017. Body condition was measured during immobilization by visually inspecting and palpating four areas on the buffalo where fat is stored: ribs, spine, hips and base of tail. Each area was scored from 1 (very poor) to 5 (excellent) and an overall body condition score was calculated, as the average of all four areas [24,25]. At each capture event, blood and faecal samples were collected to assess infection status of macro- and micro-parasites. The presence of three taxa (strongyles, *Trichuris* and Coccidia) of gastro-intestinal parasites were estimated from faecal egg/oocyst counts [26,27]. The presence of tick-borne pathogens *Anaplasma marginale*, *Anaplasma centrale* and nine *Theileria* subtypes were obtained through conventional PCR (*Anaplasma*; electronic supplementary material, S2 [28]) and high-throughput sequencing (*Theileria*; electronic supplementary material, S3 [29]) of DNA extracted from whole blood. The presence of respiratory pathogens, including bovine herpes virus III (BHV), adenovirus III (Ad-3), parainfluenza III (Pi-3), *Mannheimia haemolytica* (MH), *Mycoplasma bovis* (MB), bovine viral diarrhoea virus (BVDV) and bovine respiratory syncytial virus (BRSV), were detected by calculating the incidence of new infections using seroconversion calculations outlined in Glidden *et al.* [23]. Infections are summarized in table 1.

(c) Statistics methods

All statistical analyses were conducted using R (v. 3.5.1, [31]).

(i) The timing of first infection of pathogens and parasites

We used time-to-event models of interval censored data as our data were formatted so that we observed the time at which an animal was uninfected (t_0) and time when the pathogen was first detected (t_1)—the time of first infection lies between the t_0–t_1 interval. Specifically, we calculated survival curves for time of first infection using the Cox proportional hazard or proportional odds models in the R package 'icenReg' [32]. We specified the parametric family that the baseline distribution belongs to as a Weibull distribution, thus the Cox proportional hazard and proportional odds models used were parametric models run using the 'ic_par' function. We determined which model (Cox proportional hazard or proportion odds) to use for each analysis using the 'diag_covar' function. We used baseline survival curves to calculate 25%, 50% (median) and 75% quantiles for age of first infection for each parasite (electronic supplementary material, S4).

Bovine calves acquire maternal immunity through the consumption of colostrum [33], with maternal immunity lasting up to eight months [8]. As such, animals were included in this analysis if their age at first capture was less than eight months of age. We included 32 animals total with a mean age at first capture of 3.7 months (min = 0.05 months; max = 7.8 months; median = 3.05 months). Sample sizes were smaller within each parasite taxon depending on availability of adequate samples for analysis. Finally, we supported these results by conducting an analysis to evaluate age structuring of the parasite community composition (electronic supplementary material, S5) and richness (electronic supplementary material, S6).

3. Results

Overall, we found the median age of first infection to be lowest for tick-borne (*Anaplasma* and *Theileria*, except *A. centrale*) and environmentally transmitted (gastro-intestinal: Coccidia and strongyles, excepting *Trichuris*) parasites, with the median age of first infection commonly less than 12 months (figure 1*a*,

Table 1. Summary information of buffalo parasite community (adapted from Beechler *et al.* 2019 [30]) and prevalence in adult animals (greater than 1.5 years old). Prevalence was calculated at each capture time point by dividing the number infected animals by the total number of animals captured during that time point. Prevalence is reported as a range, including the minimum and maximum prevalence observed during the study.

parasite		type	macroparasite/microparasite	region of infection	mode of transmission	infection length	prevalence in adults
adenovirus type 3	Ad-3	virus	microparasite	lung	contact	acute	0–64.10
parainfluenza 3	Pi-3						0–56.41
bovine respiratory syncytial virus	BRSV						0–13.73
bovine herpes virus	BHV					chronic	0–43.40
bovine viral diarrhoea virus	BVDV			multi			0–56.36
Mycoplasma bovis	MB	bacteria		lung			0–37.50
Mannheimia haemolytica	MH				contact, environmental	acute	0–36.73
Anaplasma marginale	*A. marginale*			red blood cells	vector (ticks)	chronic	16.36–39.22
Anaplasma centrale	*A centrale*						21.28–44.64
Theileria velifera	*T. velifera*	protist		red and white blood cells			100
Theileria velifera B	*T. velifera* B						86.49–100
Theileria mutans	*T. mutans*						24.39–47.37
Theileria mutans-like 1	*T. mutans*-like 1						100
Theileria mutans-like 2	*T. mutans*-like 2						100
Theileria mutans-like 3	*T. mutans*-like 3						97.22–100
Theileria mutans MSD	*T. mutans* MSD						2.44–20.00
Theileria parva	*T. parva*						87.50–100
Theileria (sp.) bougasvlei	*T. bougasvlei*						94.59–100
Coccidia	Coccidia			intestines	environmental	acute	10.87–82.00
strongyles	strongyles	helminth	macroparasite			chronic	16.67–60.53
Trichuris	*Trichuris*					both	0–12.77

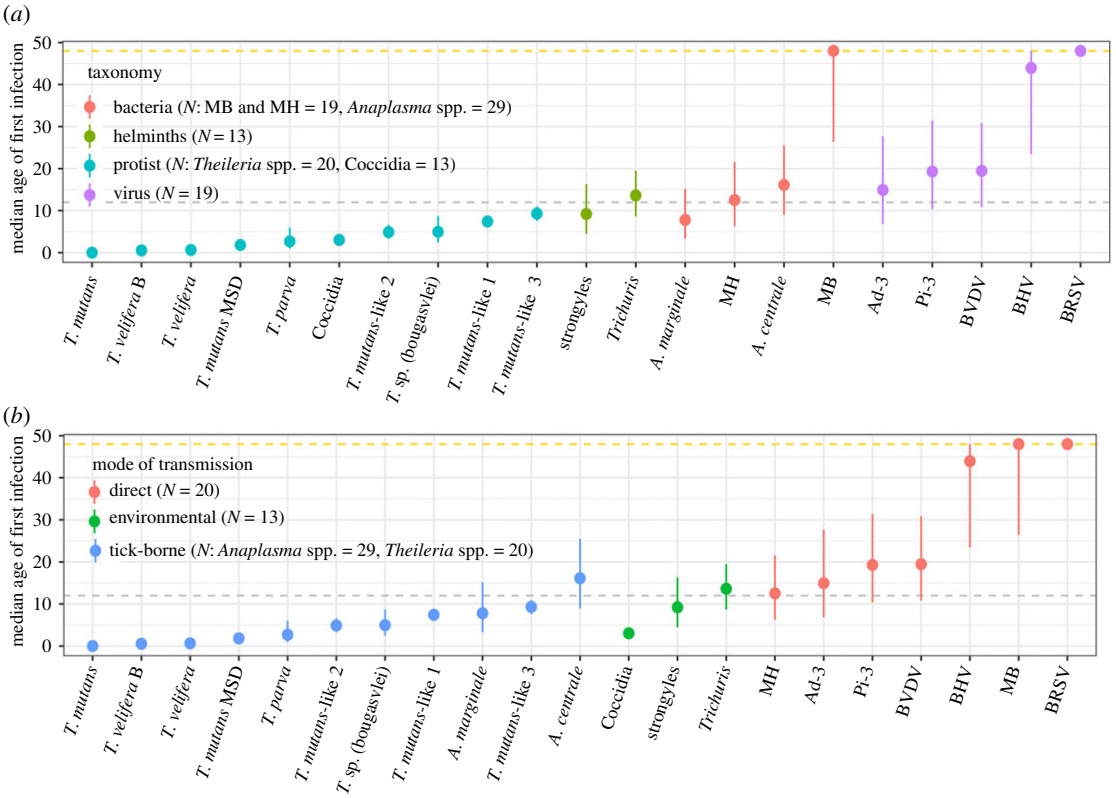

Figure 1. The median age (months) of first infection for buffalo parasites (micro- and macro-), showing the 25%, 50% (median) and 75% quantiles. The grey dashed line represents 12-month old, the yellow dashed lines represents estimates that were outside of the study's observed age range. (*a*) Orders parasites by taxonomic group (which is linked to effective maternal immunity) whereas (*b*) organizes parasites by mode of transmission.

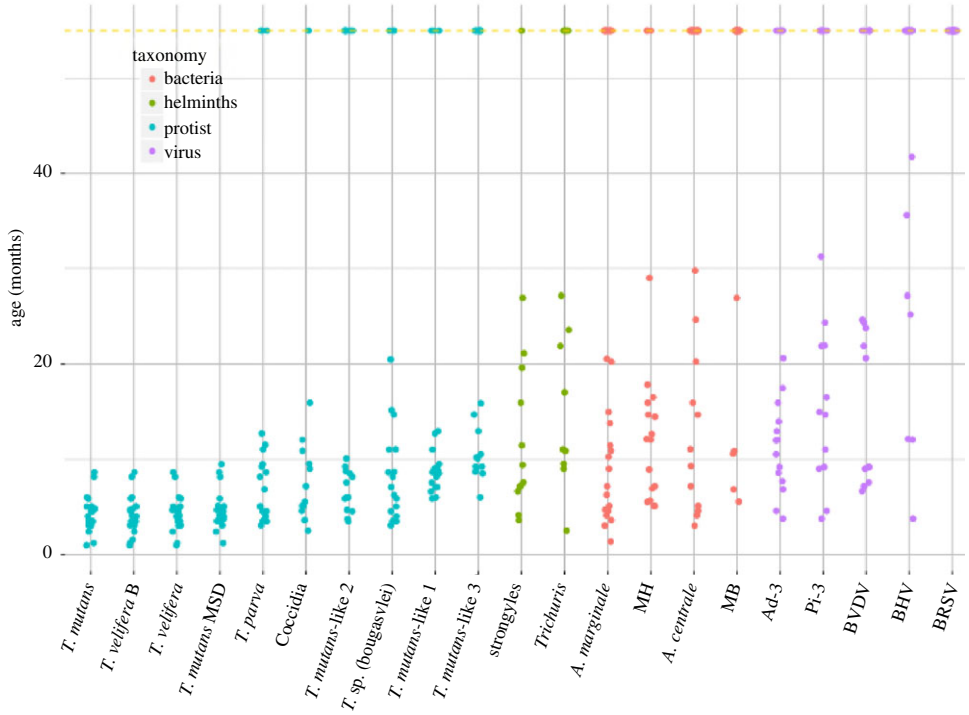

Figure 2. Raw data for the first time an animal was observed with an infection (t_1 in the t_0–t_1 censored interval). The dashed line at the top include animals in which the infection was unobserved throughout the study.

electronic supplementary material, S4). Interestingly, these parasites are also protozoa, rickettsia or helminths (figure 1*b*; electronic supplementary material, S4). We found the median age of first infection to be greater than 12 months for directly transmitted parasites (figure 1; electronic supplementary material, S4). These taxa are bacteria and viruses (figure 1*b*; electronic supplementary material, S4). We did not observe BRSV infections in any of our study animals. Raw data for the age at which an animal was first observed with the infection is reported in figure 2.

Age contributed to a significant portion of variation in pathogen community composition (electronic supplementary material, S5). Furthermore, age had a significant effect on pathogen richness, which increased until a peak at 24 months (electronic supplementary material, S6).

4. Discussion

Our longitudinal study of infections in young African buffalo indicates that protists, mostly tick-borne, infect animals before hosts were 1-year old. Gastro-intestinal helminths, which are environmentally transmitted, were acquired around 1 year of age. Similarly, bacterial infections, with the exception of *Mycoplasma bovis*, were acquired around 1 year of age, regardless of transmission mode. Thus, calves may play a role in driving persistence of these parasites and infections by these groups have the potential to affect calf recruitment in buffalo. All directly transmitted viral infections were acquired after 1 year of age. As such, for these parasites, young calves are unlikely to contribute to population-level persistence. Neither should these parasites affect recruitment of calves into the juvenile age class.

Our results suggest that maternal immunity is short-lasting or ineffective for protists *Theileria* and Coccidia. Effective immunity to *T. parva* is T-cell mediated [34], and thus, it is plausible that antibody-based maternal immunity is ineffective at preventing *Theileria* infections. The major response conferring resistance to Coccidia is cell-mediated immunity [35]. Although maternal antibodies have been found in chickens infected by Coccidia (*Eimeria maxima*), these were not fully effective [36]. Exposure to *Theileria* and Coccidia should be high when calves are born (i.e. during the wet season), as tick abundances often increase with rainfall after periods of drought [13], and protist oocysts persist longer in wetter environments [37]. The lack of maternal immunity could combine with high exposure risk at birth resulting in the low median age of first infection.

In comparison, the antibodies of maternal origin have been found to be protective against a handful of viral infections in cattle: BVDV [38], Pi-3, BRSV and BHV [39]. However, maternal immunity to these viruses may not last beyond three to four months [38,39], and we found the median age of first infection to be greater than 1-year old for these parasites. Thus, maternal immunity may protect calves through the first season of high exposure, but may wane by the following wet season. Consequently, we observe risk of infection to be much later for these pathogens.

We conducted this analysis with a small sample size. The limited sample size notwithstanding, to the best of our knowledge, this is the first study to monitor infection status of a wild mammal to a broad range of parasites from birth or near birth. Our results generate interesting hypotheses in terms of how maternal immunity may pair with transmission dynamics to drive variability in the age structuring of infection risk across parasite taxa. In addition, our results suggest that buffalo population dynamics could be largely influenced by protists (tick-borne and environmentally contracted), as these are the diseases affecting young calves, and buffalo population dynamics are sensitive to juvenile recruitment [40]. With larger samples sizes, future research could measure the effect infection on calf survival and age at reproductive maturity. Furthermore, pairing findings with age-structured mathematical models could explicitly evaluate the importance of each age category in pathogen persistence. Notably, our study was conducted within the one population of African buffalo over a 3-year period. To increase the robustness of our findings, future work should identify age at first infection in a number of different populations and throughout different timeframes. Additional avenues of work include determining whether our conclusions hold for other hosts and parasite taxa. Investigating the timing of first infection across more parasites would better elucidate the relative contribution of maternal immunity versus exposure likelihood on the timing of first infection. In summary, our study provides an important foundation for understanding the age patterns of infection in multi-pathogen systems and reveals striking variation in the role of different parasite groups as potential mediators of host population dynamics.

Ethics. The study was conducted under South African Department of Agriculture, Forestry and Fisheries §20 permits Ref 12/11/1, ACUP project number 4478, Onderstepoort Veterinary Research Animal Ethics Committee project number 100261-Y5, and Kruger National Park Animal Care and Use Committee project JOLAE1157-12.

Data accessibility. Data are available from the Dryad Digital Repository: https://doi.org/10.5061/dryad.4f4qrfj7f [41].

Authors' contributions. C.K.G., L.C., B.R.B., B.C. and A.E.J. made substantial contributions to study conception. C.K.G., B.R.B., A.V.K., D.S., R.B.G. and A.J. made substantial contributions to acquisition of data. C.K.G. made substantial contributions to analysis. C.K.G., L.C., B.R.B. and A.E.J. made substantial contributions to interpretation of data. C.K.G. drafted the manuscript. All authors revised the manuscript critically, approved the final version and are in agreement to be accountable for all aspects of the work.

Competing interests. We declare we have no competing interests.

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