Gmail **David Jansen <david.awam.jansen@gmail.com>**

Fwd: Fw: Major Revision required for Manuscript ID JAE-2018-00697

Mercy Akinyi-Matindi <akinyimercy@gmail.com> Wed, Nov 28, 2018 at 2:51 AM
To: Beth Archie <beth.archie@gmail.com>, Susan Alberts <alberts@duke.edu>, "Laurence Gesquiere, Ph.D." <laurence.gesquiere@duke.edu>, David Jansen <david.awam.jansen@gmail.com>, Robert Habig <Robert.Habig.2@nd.edu>

Dear Coauthors,

I trust this email finds you well and that you had a wonderful thanksgiving last week. Please see below comments from the reviewers regarding our MS. Beth and I have started working on the responses and will share soon.

Have a good day,

Mercy.

From: Journal of Animal Ecology <onbehalfof@manuscriptcentral.com>
Sent: Tuesday, October 30, 2018 9:45 AM
To: Mercy Akinyi
Subject: Major Revision required for Manuscript ID JAE-2018-00697

Dear Dr Mercy Akinyi

Re: Costs and drivers of helminth parasite infection in wild female baboons. Akinyi, Mercy; Jansen, David; Habig, Bobby; Gesquiere, Laurence; Alberts, Susan; Archie, Elizabeth.

Thank you for submitting your manuscript to the Journal of Animal Ecology.

I have now received reviewers' reports and the Associate Editor's comments on your manuscript and looked at it myself. As you can see, there was a consensus that this has the capacity to be an interesting paper suitable for JAE but there are some issues with the clarity of the approach, manuscript structure and the scope of the narrative, which needs to appeal to the broad readership of our journal.

As a result, I cannot accept this current version of the manuscript but I am giving you the option to submit a revised version of the manuscript for consideration.

Although we are giving you the option to complete a major revision on this manuscript, please note that acceptance and publication is not guaranteed. Your revised manuscript is due on 28-Jan-2019. If you foresee any problems with meeting this deadline, please contact the editorial office at: admin@journalofanimalecology.org

It is important to respond to all comments in this decision letter, showing how you have amended your manuscript. Please remember that we expect your manuscript to be carefully re-worked with a detailed response as to how you have addressed the comments on a point by point basis.

Please note your resubmitted manuscript must contain a Data Accessibility section stating where you intend to archive your data associated with the manuscript. For details on the Journal of Animal Ecology's data archiving policy please see: https://urldefense.proofpoint.com/v2/url?u=http-3A__www.journalofanimalecology.org_view_0_authorGuideline.html-23Data&d=DwIFaQ&c=imBPVzF25OnBgGmVOlcsiEgHoG1i6YHLR0Sj_gZ4adc&r=SuFCzwCkGdyKB6dpH9FLEg&m=5whmkoNed202MusuXjWXS_4SXNbCuLTPDnRTbvn1dBg&s=F0a4KQ6tgptVwqUw4cngut91mpq7eS

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In your author centre, click on 'manuscripts with decisions' to bring up your decision letter and link to create your revision. Then, follow the on-screen steps for re-submitting your manuscript. Please ensure you have responded to all comments.

The system will have automatically copied your original files, cover letter and information from your original submission. Please ensure that you delete all the 'old' files and cover letter, for example, before uploading your fresh manuscript.

The new manuscript will be sent to the original Associate Editor who may or may not choose to send it out for Peer Review and may or may not ask the original referees.

I will then make a decision based on these reviews and my own reading of the manuscript.

Yours sincerely
Professor Ken Wilson
Editor, Journal of Animal Ecology

Associate Editor
Associate Editor Comments for Authors:
Both reviewers thought this was a generally good paper but raised a number of detailed comments on the clarity of the approach and results. They also made suggestions on alternative or complementary mechanisms that can generate the patterns observed, which should be discussed when explaining patterns and results. The paper structure should also be checked.

REFEREES'COMMENTS TO AUTHORS

(nb. If there is no comment from a Reviewer listed below, this probably means that they have uploaded a separate 'file for author' to the ScholarOne Manuscripts Site. You can see these comments in your Author's Centre by clicking ‘manuscripts with decisions’ and then using the 'files attached' link at the bottom of the decision letter)

Reviewer: 1

CONFIDENTIAL COMMENTS TO AUTHORS
The authors have undertaken extensive analyses of data collected from the outstanding Amboseli baboon population to show how helminth infection influences reproduction, and identify drivers of variation in parasite burden. Overall, I found this an interesting read and I think it will make a nice paper, although I do have quite a few comments regarding the explanation of the analyses and the presentation and interpretation of the results.

Line 60-61: I think the costs of infection should be reworded to reflect the fact that there are both (1) pathology/damage and (2) resource costs of fighting infection; currently this paragraph makes it sound as though only number 2 is being invoked. There are additionally costs of immunopathology and co-infections, which could also be mentioned in passing.

Lines 64-74: I find that the treatment of the expected association between GC and helminths is being made to sound a little simplistic (even if this is not the authors’ intention). In this paragraph, GC is suggested to mediate energetic expenditure, which is true. However, an association between GC and infections may be observed for many reasons and the association may be observed to be either positive or negative. For example, one could imagine high GC individuals with lower resistance due to the stress of reproductive investment, or high GC individuals with higher resistance because the fact they’re making a greater reproductive effort indicates they’re in better condition than individuals making a lesser reproductive effort.

Then the paragraph seems to say that the GC-helminth link is because of energetic reserves, but then if there's no link, it could be due to one of two reasons (due to tolerance or weak effects of parasites). I think there are many other possible reasons for not finding a link (positive and negative associations cancelling each other out, for example) and it's (inevitably!) a bit more complicated than portrayed here.

Lines 75-93: It isn't clear here whether the authors are simply talking about infection (is an individual infected or not, which is how this paragraph is written) or whether the material in this paragraph is also relevant to quantitative resistance (the ability to limit parasite burden once infected). I'd like to see the distinction either made, or it to be made explicit that both resistance to infection and ability to limit burden are included here.

Lines 95-106: This is doubtless a difficult issue- social aspects may have all sort of effects on exposure/resistance to parasites. Could a sentence be added to suggest how we might go about untangling all of these conflicting effects (better yet if it's an approach used in this study!).

Lines 186-197: I think it would be useful to predict which direct the associations between parasite measures and reproductive traits are expected to be in. I say this because I think it's far from obvious what to expect. For example, I think most readers would assume that a lower IBI is better since it reflects a faster reproductive rate: hence, we'd expect a higher parasite burden to be associated with a higher IBI (more parasites; lower reproductive rate). However, I'm not sure what I'd predict regarding pregnancy. Is a longer pregnancy a good thing or a bad thing? Do we expect there to be much variation in length of pregnancy, or do we expect most of the variation in IBI to be because of variation in PPA? I wonder whether it might be worth discussing the relative importance of the effects of parasites on the different components of IBI- for example, is the effect on pregnancy duration small compared to the effect on PPA?

Line 172: The mean number of samples per female is given as 8, but 745 / 122 = 6.11.

Line 215: How was age treated in the analyses? Was it a covariate or a categorical variable, and were non-linear effects considered?

Line 263: It's not clear here whether each of the five parasite measures were added to the same model, or whether each of the five measures was tested in a separate model. Could you make this clearer?

Lines 271-275: were random effects included in models of GC? Presumably there are multiple measures across females and years?

Line 290: I can't see any information in the Methods or Results on how the final models (e.g. in Table 3) were arrived at. The initial models contained a lot of terms which do not seem to appear in the final model. How was model selection performed? Was stepwise deletion used? AIC comparison of a range of fitted models? Please provide more information. This is true for every stage of the analysis.

On a similar note, why are some factors denoted as 'NS' in the Table? Clearly they're non-significant, but there must have been other terms tested (based on the description of what was included in each model) which were also non-significant. What's so special about these terms?

Line 310: A key result is that individuals which have higher parasite species richness have longer inter-birth intervals, with the causation implying that richness IBI. However, I could also imagine that a longer IBI might lead to greater richness simply as an artefact: more time to accumulate more parasite species will lead to greater richness (even if the mean richness is taken). Perhaps an extra 35 days IBI allowed a mean of one more parasite species to be encountered? Unless the authors consider this possibly to be remote, I'd suggest perhaps acknowledging this in the Discussion.

Line 314: There's no need to quote the F-stats and P-value here, since they already appear in Table 3, to which the reader is directed. This is true throughout this section.

Lines 332-338: I think it's probably worth examining in the Discussion (e.g. lines 428-432) why this association was no longer supported once reproductive status was accounted for in the model. Presumably this is because pregnancy = higher Tt and pregnancy = higher GC, and thus higher Tt = higher GC when pregnancy is not included in the model.

Line 343: Helminths aren't linked to lower fertility here, they're linked to longer inter-birth intervals and its

components. These associations may be due to effects on fertility, or they could be driven by other causes. I think you could say ‘measures of fertility’.

Line 346: Once again, no need to include estimates and P values, given they appear in Table 4; this applies throughout the paragraph.

Line 413: Strongyles weren’t associated with IBI in Table 4- only two of its constituent parts.

Lines 419-421: This doesn’t seem for an argument as to why parasites prolong IBIs- it more seems an argument for why parasites limit the resources for immune responses. Instead, I would say that parasites cause damage which limits reproductive ability, or elicits immune responses which limit reproductive ability: these are both reasons why infection would prolong IBI.

Line 429: I don’t think that you can be as confident that the association works this way round ‘GCs rise in response to infection’ implies ‘infection CAUSES GC to rise’. This is a correlative analysis (never mind which is the response variable) and so is unable to determine causation. Equally, individuals with chronically-high GC may be less able to resist infection. The experimental study that you cite here CAN say that removing parasites drives GC, but the correlative analysis here cannot be so confident. Both possibility should be discussed.

Line 443: A third explanation is that infection begets poor condition, which begets further susceptibility to similar parasites, which begets poorer conditions, and even higher susceptibility…

Line 503: The Discussion ends rather abruptly. It would be nice to see some material summing-up the findings, and/or the advances made, and/or the future work which is needed to build upon the study.

Figure 2: Please show the data: it would be nice to see the raw data as well as the means and SE. For B, C, D, E, and F, it would be better to show simple scatterplots of the raw data overlain with regression lines and SE for the model predictions, since this is how the data were analysed. Showing the data divided into arbitrary bins could mask the real pattern: for example, C, E and F look non-linear, but I didn’t see any evidence that you modelled the associations with anything other than a linear function (e.g. there was no mention of a quadratic function being fitted).

Fig S1: Since a regression is used for the analysis of the association between GC and T. trichuria egg counts, it would be nice to see the data represented this way, rather than in the categorical format currently used in this figure. What’s wrong with a simple scatter plot showing all the hard-earned data?

Table 3: If (for example) T. trichuria was log-transformed, it should be denoted as such in the Table.

Random effects were fitted in all of the analyses conducted (as far as I can tell) and yet nothing is given in the Results regarding whether these effects were significant, nor how large these effects were. It would be very interesting to know, for example, how much variation there was between individuals in reproductive and parasite traits: it would help to establish how repeatable the traits are. At least showing random effect estimates in the model results tables would be a start.

Reviewer: 2

CONFIDENTIAL COMMENTS TO AUTHORS
This research investigates the forces that drive parasite risk and the costs of those same parasites to individual fitness. It is unique in its multi-scale approach, considering host, group, and population- level factors to understand how parasites act as selection pressures that shape behavior and life-histories. In such, this paper offers an unusually holistic understanding of the drivers and costs of parasitism within a population. By spanning 5 years, this research is also able to demonstrate fitness consequences of parasitism in long-lived social species, making it a valuable contribution to the literature.

The authors found that parasites were associated with reduced fertility and that female infection risk was predicted by factors acting at the level of the host (age and co-infection), group (social connectivity) and population (dry conditions). Individually, results predicting infection risk are mostly confirmatory of previous studies. However, as a whole, they offer a uniquely holistic picture of primate parasite ecology within a single population. This is valuable as it has proven difficult to assess the interplay between factors in light of

observed variation across group, species, and habitats in the literature.

As a consequence of their multifaceted approach, the authors must deal with interpreting (and interrelating) outcomes across scales. I believe the paper could be improved through better organization of results and discussion of the interplay of factors across these scales (the real strength of their approach). The methods are appropriate, but there needs to be more information on the sampling protocol so that we can evaluate the relevance of infection parameters to individual, group, and population level factors that vary in scale.

Overarching Issues

Organization

1. The paper spends a long time reviewing the diverse drivers (and competing hypotheses) explaining primate parasitism across multiple scales, which has been done elsewhere. I suggest removing much of the review and including the key references in Figure 1 (which nicely summarizes competing hypotheses), coupled with a brief summary paragraph that explicitly states the directional hypotheses of the research at hand. The most salient debates and supporting literature can then be revisited in greater depth within the discussion. As is it now, there is a lot of repetition between the introduction and discussion (and even results).
2. There is quite a bit of data interpretation (and repetition) between the results and the discussion sections, much of which would be more appropriate in the discussion.
3. Given the multitude of factors being tested, it is easy to get lost in this paper. I suggest the authors stick to an organization scheme throughout. For example: 1) costs (reproductive, glucocorticoid), and 2) predictors (individual, group, population). In the results section, the paper switches to organizing results by parasite type (e.g. section 4.4). I find this very confusing. These types of cross-level analyses and interpretations would be very beneficial as part of a synthesis in the discussion.As it stands now, the discussion reads like a laundry list of individual interpretations rather than a synthesis of results across multiple levels of analysis.

Methodology

4. It is difficult to evaluate the relevance of the infection parameters on the different host factors because the sampling scheme is not laid out clearly in the beginning. For example, the authors state that individuals were represented by 1-25 samples, which is highly variable across individuals. What were the criteria for a female to be part of the study? Were individuals represented by one sample only present for part of the study? One fecal sample offers incomplete information on infection due to intermittent shedding and these limitations in determining infection status should be acknowledged.
5. An explicit statement within each section describing how the different variables were constructed in relation to parasite values would be very helpful. How were host, group and population factors attributed to a single sample collected at one point in time, and what was the justification? For example, how was infectious status (frequency of collection unclear) related to dominance (assigned monthly; line 227), social connectivity (calculated for each year; line 231), group size (assigned from the day of sample collection), and temperature (average from three month prior)? How variable is group size and thus how representative is group size from a single point in time? The paper contains some of this information, but the reader has to really search for it. For example, it is not until the results (line 263) that the reader learns that parasite metrics from samples collected during each IBI and component phase were averaged.

Minor Issues

1. Line 70: “host GCs may decline after parasite removal” refers to a different study than the one cited (Friant et. al. 2016, Proceedings B)
2. Line 75: State why it is important to understand the drivers (e.g. selection pressures shaping behavior and life history), not just that it is important. I also suggest removing “Once the costs of helminth infection are known, an important next step is to”.
3. Line 80: (Fig. 1) underdeveloped immune systems in younger life stages may also increase infection risk and should be listed as an alternate hypothesis. Similarly, coinfection can have competing effects by facilitating or inhibiting infection.
4. Line 101: See recent review (and references within) Rushmore et al. 2017 Trends in Parasitology
5. Line 178: The methods section heading do not accurately match the associated text. Section 2.3 should read measuring parasite effects on female interbirth intervals, since the text discusses parasite methods as well. Same thing with the following sections.
6. Line 179: How was the variable “helminth burden” constructed? Is it an average over the 5 years, or specific to samples collected during that IBI? If it was the latter, were individuals excluded that did not have

a representative sample?
7. Line 203: Do GCs in this population increase throughout the day as is known to be the case in many social primate species? If so, was fecal collection restricted to the morning hours? If not, how might this affect the results?
8. Line 203: How many samples were collected per individual? Per year? How was the GC variable constructed? Were multiple samples averaged?
9. Line 212: It would be helpful to have the supplemental table (or a version of it) within the manuscript for reference
10. Line 252: Begin with a statement of what is being tested with the mixed models
11. Line 255: For clarity, rephrase to indicate that these are your response variables
12. Line 271: Change “are” to “were”
13. Line 271: Please justify the basis for the decision to include bivariate and multivariate tests for GCs only.
14. Line 288: Please justify your decision not to nest individual as a random effects within group when testing for host-level factors, as was done for previous models.
15. Line 307: Authors state there was no relationship between helminth parasitism and female glucocorticoid levels in the abstract (line 36), which contradicts section heading 4.2 (line 307).
16. Line 317: The results showing different parasites were associated with each component phase of the IBI is not discussed further. Do the authors have any ideas to explain this finding?
17. Line 361: The first sentence does not accurately reflect the results outlined in the paragraph. Shouldn’t this read, “low rainfall and low temperatures predicted helminth infection”?
18. Line 372: The beginning of this section would be more appropriate in the discussion
19. Line 375: replace “increased risk of” with “was associated with” as to not infer causation.
20. Line 403: No need to state that this is the second study, just state whether your results confirm or contrast with results from previous work.
21. Line 425: It is not clear how the GC results support the idea that helminths rise in response to infection. Please provide a more detail discussion of this.
22. Line 435: The term “drivers” implies causality, but some of the results in this section could be a driver or a response (e.g. social isolation).
23. Line 485: Here it is stated that warm temperature elevates risk, but in the results it is stated females had higher egg counts and higher helminth richness during cooler periods (line 367).
24. Line 469: This first sentence is a little misleading, revise to indicate that group size predicted female parasite risk in conflicting ways.

End of Comments

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