Dear Recommender,

Thank you for the careful evaluation of our manuscript. We have utilized the reviewers' exceptionally thorough feedback, as well as yours, and we a provide brief description of the changes together with our responses below. We hope you find our response satisfactory, allowing for recommendation of the manuscript.

With best regards,

Petteri Karisto, on behalf of the authors.

Recommender:

Dear authors,

I have now received three expert reviews on your preprint. As you will see below, all reviewers agree that your manuscript addresses an interesting question, that the paper is well written, and that the analysis seems to be correct. However, they also raise several questions and comments that suggest different ways of improving the analysis and presentation of your results. Having read your paper, I agree with the reviewers' assessments, and see many opportunities for improvement.

In particular, and in line with some of the reviewers' comments, I would like you to pay particular attention to (i) the possibility of adding a figure illustrating cytoplasmic incompatibility and the workings of the model (as suggested by reviewers 2 and 3), (ii) being more concise in pages 7-10 (as suggested by reviewer 2), and (iii) the comment on the (unnecessarily complicated?) use of matrix calculus (suggested by reviewer 1).

I therefore ask that you revise the manuscript to take into account these and other reviewer's comments. Please also provide a detailed point-by-point response to all comments. After this, I will be able to further consider the preprint for recommendation.

> Thanks a lot for this positive and constructive feedback. We have followed the suggestions as specified below, and we feel that the manuscript has benefited substantially from the edits.

In addition to the reviewers' specific comments, I have some of my own. First, I would like you to pay attention to a typographical detail. You often write 0.5 for either "1/2" or "one half". The form "1/2" looks nicer in math mode or in equations; the form "one half" looks nicer when writing sentences. For instance, in line 29 you could write "can produce low frequency ($<1/2$)" and in line 31 you could write "any stable equilibrium close to one half".

> Done.

Second, I found some of the choices for naming variables a bit strange. In the kind of population dynamics models that you study, time is usually denoted by (lower case) t, not (upper case) T, as you do in the manuscript. This choice might be due to the fact that you already use t for one of the parameters of the model. My suggestion would be that you stick to the standard norm of using t for time but use an alternative symbol for the transmission parameter (that you now represent by t).

There is some historical legacy here. On the one hand it would be nice to adhere to common norms about *t* denoting time, on the other hand we were also hoping to not deviate from the notation of earlier modelling on the present topic, where Engelstädter & Telshow had used *t* to denote transmission. Our hope was that *T* is close enough to 'time' that this would as a whole make sense. We now solved this together with the issue below, see our next comment.

Third, you write down equations (1) and (2) in the form $p_{T+1}=g(p_T)$ for a given function g, but then in Fig. 1 you illustrate the dynamics by plotting Δp_T as a function of p_T. I would stick to one or the other way of writing these recursions and would try to be consistent, i.e., I would either stick to the way equations (1) and (2) are written now, but then change Fig. 1 to show p_{T+1} as a function of p_T, or I would keep Fig. 1 as it is but then rewrite equations (1) and (2) in difference form. When making one change or the other, please also try to be consistent with the way you write down the equations for the haplodiploid cases.

Thanks for these useful suggestions. We solved this inconsistency (together with T vs t issue) by introducing the models in the difference form $\Delta p = …$. This way we give the function for Δp explicitly and also got rid of the time T everwhere else than in Appendix B, where it was still needed. We hope that it does not disturb too much there – as stated above, our desire to follow 'traditional' notation is not easy to do when there are two different uses of *t*, depending on how narrowly we define the relevant literature.

Fourth, many equations in the Appendix seem to be copy-pasted directly from (I guess) Mathematica, leading to cumbersome expressions that are difficult to parse. As an example, the factor $f(-1+(-1+k)L(-1+t)t)$ appearing in p. 39 could be rewritten as $f[L(k-1)t(t-1)-1]$ (note also the use of square brackets, consider using e.g., $\left($ and $\right)$ instead of simply $($ and $)$), which is shorter and easier to parse. I encourage you to make these changes throughout the manuscript.

Thank you for pointing this out. We have reorganized the terms and adjusted bracing to make the equations more readable. We realized that simple \left(+ \right) did not solve the issue with braces, since those dynamically-sizing braces stay small when all content is on single line. Hence, we manually sized the braces to help reader to recognize the level of bracing (\bigl(, \Bigl[etc).

Finally, although all reviewers agree that the manuscript is well written, I found some grammatical mistakes and typos---please revise the writing carefully and edit the manuscript accordingly.

I look forward to receiving your revised manuscript.

Best regards,

Jorge Peña

Reviewer 1:

The authors re-investigate and extend the qualitative dynamical behavior of Wolbachia prevalence in populations. Classical models of Wolbachia population dynamics assume a fitness disadvantage of infected host individuals. Together with a positive frequency-dependence caused by cytoplasmic incompatibility, this results in bistable dynamics. The two stable equilibria, separated by an instable equlibrium, are the Wolbachia-free state and a high frequency of Wolbachia-carrying host individuals. Here, the authors relax the assumption of negative fitness effects and show that positive fitness effects can change the model behavior substantially. Positive fitness effects have already been studied by Zug & Hammerstein (2018). However, the focus of that study was predominantly on invasion dynamics of cytoplasmic incompatibility and male-killing parasitic invasion behavior. In contrast, the present study focuses exclusively on cytoplasmic incompatibility in diplodiploid and haplodiploid populations and adds analytical justification to undermine the results.

First, the authors study the Wolbachia dynamics in diplodiploid populations. After reviewing the classical theory, they add a rigorous analysis of the case of positive fitness effects of a Wolbachia infection. This assumption may result in situations without an invasion threshold, which cannot be the case if infection comes with a fitness disadvantage for the host. The authors do a great job explaining the new findings both mathematically and biologically. Second, the authors apply the same methodology to Wolbachia dynamics in haplodioploids. Essentially the same conclusions as in the case of diplodiploidy hold. Unfortunately though, in the case of haplodiploidy the results cannot be shown completely analytically. Instead, the authors conduct a large numerical study that supports their claims.

Overall, the manuscript is well written. The authors have reviewed and acknowledged the vast existing theory very nicely. The new contribution of the manuscript is highlighting that stable intermediate and low frequency equilibria of Wolbachia prevalence in host populations can be explained by positive fitness effects of the infection. The rigorous analysis is, as far as I can say, correct, though I have a question regarding the haplodiploid case (see comment 12 below). Besides this, I only have a small number of minor comments that I list now.

Thank you for your kind words and thorough evaluation of the manuscript.

Comments

1. Terminology `low frequency' (first in line 29 and throughout the manuscript): I personally would prefer the term `intermediate frequency' instead of `low frequency'. To me, low frequencies are very close to the extinction boundaries, e.g. maintained by mutation-drift-balance. The manuscript, however, studies and emphasizes the possibility of frequencies far from the zero frequency boundary, which is why I would prefer the term `intermediate'. (I am aware though that technically `low' can be interpreted as closer to zero than to one, which is how the authors seem to use the term.)

Thanks for this note. We have added explanation of the specific use of word "low" in Abstract and few places in the main text, to clarify the meaning.

2. Line 220: two → three

Done.

3. Lines 222-.: I am not sure to understand that paragraph correctly. The authors first state that changing the parameter f may lower the equilibrium frequency of Wolbachia. Then the authors state that increasing the value of f and all else remaining equal, the equilibrium frequency increases, which is in line with the biological intuition. This is in contradiction to the introductory sentence of the paragraph. I suggest to rephrase it or to clarify what exactly is counter-intuitive.

> We clarified the explanation. Lowering f indeed does not lower the equilibrium, but it *allows for* a lower equilibrium frequency. We now write:
>
> "It is worth clarifying the statement that introducing benefits can allow for a lower equilibrium frequency of *Wolbachia*, as it is counter-intuitive at first sight. The meaning of the statement is not that higher *f* lowers the equilibrium frequency when other parameters are kept constant; this does not happen, instead, higher *f* increases the frequency. The statement refers to the fact that this very effect (high *f* improves the prospects for *Wolbachia*) can, under parameter settings that are a priori unfavourable to *Wolbachia*, shift a situation where no *Wolbachia* persist to one where some spread is possible, and the system finds its equilibrium at low *p* (the light-coloured curves in Fig 2e; Fig 3)."

4. Fig. 1: I suggest to add a legend with the different values of L directly to the plots. Additionally, I think it would be helpful to zoom into the low frequency range of pT in subfigure b) because the very low stable frequency mentioned in the main text (line 215) is barely visible.

> Both done. Added new panels to show the differences close to zero better.

5. Lines 361-363: My (very small) literature research also resulted in small evidence for Wolbachia evolution towards a mutualistic, hence positive fitness effect, lifestyle, e.g. Weeks et al. (2007), PLoS Biology, From Parasite to Mutualist: Rapid Evolution of Wolbachia in Natural Populations of Drosophila. It is maybe worth mentioning this biological possibility of parasite evolution that enable stable intermediate frequencies.

> Very interesting, thanks! Added in the suggested place.

6. Line 317: I think in the Section `CI infection dynamics' in the paper by Zug & Hammerstein they show (or claim?) that the invasion threshold vanishes if ft > 1 (even though I am not sure if they show this analytically or numerically).

> Correct, Zug & Hammerstein 2018 indeed state that in the mentioned section of their paper and we now note it in the third paragraph of discussion.
>
> Interestingly, we could not find proof for that statement in their paper, except for the figures with certain parameter values (numerical, not exhaustive). They also state that the threshold exists whenever ft<1 as do also Engelstädter & Telschow 2009 (Box 1). Neither paper provides proof of this claim either. It seems to hold, if one first makes the assumption that we are in a parameter region where the two non-trivial equilibria exist in the first place (obviously not

all ft < 1 qualify, since some are too low for any spread of Wolbachia). We now note this additional condition (that the non-trivial equilibria have to exist) in the text. We also note that with ft>1 there is no invasion threshold *because* that condition implies unstable equilibrium at p0.

7. Line 424: inequation → inequality

Done

8. Line 437: I suggest to add a sentence about the purpose of the analysis, e.g. `we show that ^pF > 0:5 whenever f <= 1.

Done.

9. Line 440: I suggest to repeat that the case f <= 1 is considered.

Done.

10. Line 457: I think the title should rather be Local stability analysis?

Good point, thank you, we changed it.

11. After Eq. (B.3): I suggest to give a reference, where an interested reader could look up the standard technique of local stability analysis, e.g. the book by Otto & Day.

Done.

12. Matrix calculus and the analysis of the haplodiploid case: First, I need to admit that I have not seen the framework of matrix calculus before. This might be the reason for my confusion. Basically, I was wondering why the authors take the detour of matrix calculus instead of conducting a `standard' local stability analysis of the polymorphic equilibrium, i.e. studying the eigenvalues of the Jacobian of the model dynamics? Maybe this approach would yield analytical results? Or could the authors explain why they used the matrix calculus framework and how it is different from the `standard' local stability analysis approach?

Thank you for pointing out that this is a bit non-standard and might be confusing. We found it convenient to use matrix calculus to derive an expression for the Jacobian matrix, but we accept that this may appear to be an overkill. We now instead derive the Jacobian matrix through the more usual route by directly calculating the partial derivatives that make up the Jacobian matrix.

Reviewer 2

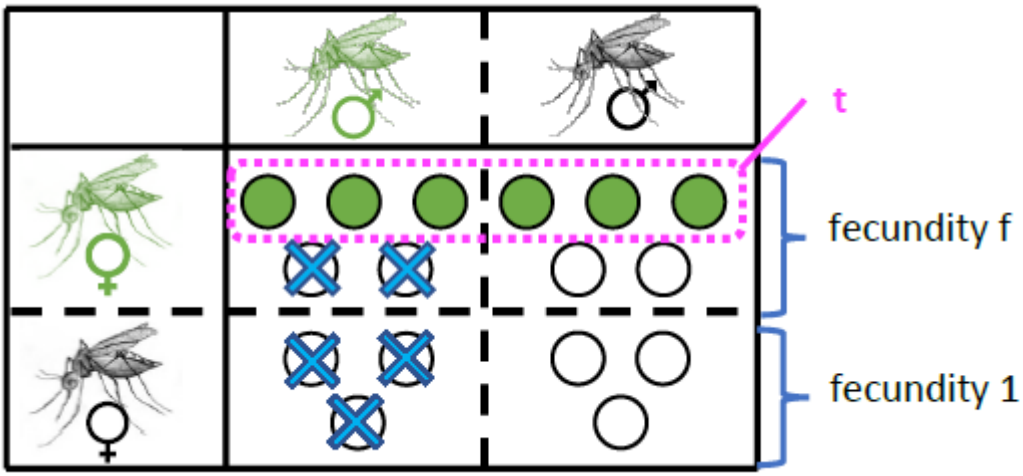
Karisto et al. revisit models of propagation of a Wolbachia endosymbiont within a host population. They consider a pleiotropic effect of the symbiont on the host fitness, namely a direct fitness effect manifested in a relative fecundity rate f of females carrying the symbiont, and a cytoplasmic incompatibility that results in either a reduced survival or a masculinization of uninfected eggs

fertilized by infected males. Classical models have focused on deleterious direct fitness effects (f < 1), that necessarily result in either extinction of the symbiont from the host population, or an equilibrium frequency of symbiont-carrying host close to fixation. In contrast, Karisto et al. propose to focus on beneficial direct fitness effect. They study the fixed points (values and stability) of three different models (diplo-diploid, haplo-diploid with female killing and haplo-diploid with masculinization) and robustly conclude that a beneficial direct fitness effect (f > 1) provides a plausible and potentially testable explanation for the low frequencies of symbiont-carrying hosts sometimes observed in nature. I find the manuscript very interesting and well written, and have no scientific concern to raise. In the following, I only ask a few minor remaining questions and provide a few suggestions to streamline the manuscript, which could gain in conciseness in order to be more easily accessible to a broader audience.

Thanks a lot for your kind words.

General comments & broader questions

I would strongly advise to add a figure with drawings recapitulating the key parameters of the model, for example in the line of this:



Thanks for this extremely useful suggestion! We have added a figure very similar to this one.

I understand that this goes beyond the scope of this paper, but for my own curiosity: what about the evolution of the total size of the host population, not only of the frequency of symbiont-carrying hosts? I was wondering if you could comment on that, as I am assuming that this is a key parameter in using *Wolbachia* CI as a potential way to control mosquito populations?

This is indeed interesting but we believe it does, indeed, go beyond the scope of this paper – based on our analysis we don't have good answers right now. It would be an interesting avenue for further work, for sure!

In the paper you are focusing on the fixed points and their stability. But what about the dynamics to approach the fixed points? Do we know anything about the time to fixation? If dampening oscillations are to be expected? This is also probably going beyond the scope of the

paper, but I am also expecting this to be of importance, again, if CI is to be used as a controlling tool.

> Again, a very interesting point that is beyond the scope of the current analysis. Deriving such expressions would certainly be of interest when *Wolbachia* is used for control purposes. Once again, an avenue for further work in this area!

From what I understand, Wolbachia has the ability to "jump" from one host species to another quite easily. In these conditions, the initial symbiont-carrying host frequency will always be low. Are there mechanisms other than stochastic fluctuations that are being proposed to explain how the invasion threshold can be overcome?

> It's indeed a puzzle how the invasion threshold is/was overcome in nature, and mostly the ideas turn around some kind of stochasticity. As our MS does not really shed new light on this question, we have not expanded it in this direction.

Abstract

I think the use of the term "pleiotropy" in the abstract is unnecessarily complicated for non-specialists and would rather suggest something in the line of "additional direct fitness effect", as is otherwise used in different sections of the manuscript.

> Agreed, and changed.

L34: I think the use of "infection rates" here is not exactly correct, because it suggests an evolution in time. I think just removing "rates" restores what I think you mean.

> True, done.

Introduction

L64: the spread of the *Wolbachia* → the spread of *Wolbachia*

> Done.

L74-76: At first read, the meaning of this sentence was unclear (in particular, do they also consider CI in the mentioned paper?)

> Clarified this part. (Yes, they do.)

Models and analysis

L130: I think it should be the contrary ($t \leq 1, L > 0$) to prove the point that $f \geq 1$ (even though both are true).

> I think this is a misunderstanding of the notation. We mean that both t and L are in the range from 0 to 1.

Pages 7-8-9-10 in particular would benefit from being more concise. What could help is to follow more closely the order of the panels of the figures in their textual description (or alternatively, reorder the panels). More particularly, L142-148 and L164-181 could be shortened, I think.

> We checked the panel referring order now with the new fig 2.
>
> More concise writing is often preferred, but here we wanted to really explain what happens in the models and why. We found it hard to make sense of it (with the earlier literature) and thought there might be room for a bit more detailed explanations. This was noted as a positive aspect by R3: "outlining not only the results of their models, but much of the intuition behind these results too. This personally gave me a much richer understanding of the population dynamics, which I really appreciated".

L209-210: I did not understand *"this case is fully captured by the analogy to the asexual lineages A and B above"*, could you please clarify?

> This sentence was an orphan from a previous version, thanks for noting. Removed.

L227: Why only 2c? From my understanding I would have said that the whole of figure 2 is relevant here?

> Correct.

L283: I would suggest to make the change (many eggs → many fertilized eggs) for clarity.

> Done

L297 and following: here I would suggest to make it clearer that you speak only of the masculinization version of the haplodiploid model. In general, I think the manuscript might gain in clarity if these two cases are more clearly separated.

> We added clarifications.

Discussion

L 339-345: here it is not clear to me how would a model with two different *Wolbachia* strains, one with $f_1 = 1$, the other with $f_2 \neq f_1$ would be any different from the current model with infection vs. no infection. Could you maybe comment on this?

> With two strains, there might still be uninfected individuals, so that there are three different fitness levels in the population. Additionally, with multiple strains there is potential for bidirectional or unidirectional CI (i.e. they inhibit each other, or only one inhibits the other), though this would likely have no effect for the initial dynamics governed by *ft*.

L361-363 I would suggest to move this sentence by one paragraph, to keep all the comments on empirical data together (this sentence looks more specifically linked to L377-378 in my opinion).

Makes sense, done.

L390-392: Could you please elaborate on mtDNA genotype diversity? I am not familiar with how this could help elucidate temporal evolutions in the context of endosymbionts. Is that because the host's cells are expected to lose redundant genes in streamlining coevolution?

Mitochondrial DNA will "follow" the infection since both of them are maternally inherited. We added a bit more explanation in the text.

L394-396: I am assuming that the studies mentioned here refer to dengue-carrying mosquitoes' populations monitoring. I would explicitly mention the host species to make it immediately crystal clear.

Done

Figures

Figure 1: On panel 1b, it could be useful to have a zoom insert of what happens close to zero. I would add on the figures 1b and c $ft > 1$ and $ft < 1$, respectively, to make the comparison easier without refering to the text of the caption.

Done

Figure 4: In the caption, I don't think "FK" and "MD" have been defined before, and I am still unsure what the D stands for.

Thanks for having sharper eyes than we did! This notation was orphan from an earlier version (Female Killing, Male Development). Now fixed. We also searched for "fk" and "md" and fixed a couple of occurrences in the appendix.

I think it should be capital M and F in the equilibria notations (but this also occurs in the caption of figure 5, and I might have missed other occurrences – be sure to make the notation uniform).

Fixed the lower case f and m in p_f & p_m to upper case across the manuscript.

I would recommend to write the f values on top of the columns to ease comparison of the panels (and since most parameters are conserved). Would it be possible to also take L=0.65 in d) for consistency?

We would prefer stating all parameters in the caption to avoid a false impression that the other parameters are the same. We tried many combinations and found these ones being suitable for the visualization; the curves are quite sensitive and we wanted to avoid too cluttered drawings. Unfortunately we could not keep the other parameters constant and only vary f. However, we managed to unify the parameters within columns and now make this explicit in the caption.

Figure 5: as before, I think it should be capital M and F in the equilibria notations.

Done.

In addition, I think it might be nice to have the shades of grey also directly explained on the figures, without having to refer to the caption text (this is also valid for figure 3).

> Introducing the grey scale legend into the figures decreases the size of the panels or overlaps with them. We haven't found a pleasing solution for that. Hence, we prefer to keep the explanation in the caption.

Annexes

L422: "the real part" here is not clear (only if the determinant is negative are the solutions complex conjugates).

> We agree that 'the real part' is not really an exact term – we used this wording for the part that is necessarily real. Added this explanation in the text.

L432: I think there lacks one final parenthesis in the denominator of p_F (which is correctly written in r_F a few lines down the page). Maybe the expression for p_F and p_M could also be squeezed into a single line to not have to repeat the same denominator twice.

> Fixed this. The squeezing was not possible due to space restrictions.

Page 27 might benefit from a couple of drawings to follow the reasoning more easily. Also on this page, "when $f \leq 1$" L 442 should be made clearer from the beginning of the argument and be stated explicitly that it is a proof by contradiction.

> We clarified the explanations in writing.

L536: I think there is a parenthesis error (it should be after the first $p_S L$, not the second, like in B9).

> Thank you for spotting this, we fixed it.

L544: I think there is a sign error (it should be a + instead of the first -, as in L546).

> Again, thank you for spotting this! We fixed it.

L554: notations should be standardized ("x" or ","), also with the following section.

> Thanks, both are now x.

Reviewer 3

Summary

Cytoplasmic incompatibility is a reproductive strategy used by certain strains of Wolbachia (and other matrilineally transmitted bacteria), whereby infected males only produce viable offspring with infected females. This means of reproductive manipulation has frequency dependent effects, as benefits to infected females increase with the number of infected males in the population.

This paper studies the ecological dynamics of Wolbachia spread and – in particular - considers the consequences of the Wolbachia providing direct fitness benefits to the female host, which have previously been ignored. Contrary to previous models, they find that stable, low frequency equilibria of Wolbachia infections can be maintained provided such fitness effects are positive. Moreover, when there are direct fitness benefits, then there needn't be an infection threshold that initially must be crossed. They suggest that this might provide a parsimonious explanation of the low Wolbachia frequencies seen in some natural populations. In addition to an investigation of diplodiploids, they also consider this effect under haplodiploidy, considering two distinct types of cytoplasmic incompatibility, a direct male-killing strategy, and a masculinising male strategy.

General comments

Overall, I thought this paper was excellent, and would be of great interest to the readers of PCI Ecology. I thought both the empirical and theoretical motivation for the question was clearly laid out, and the authors choice of approach appeared appropriate. The models were well explained, notation was clear, and I was able to recreate the key results from the descriptions provided. In addition, the authors did a thorough job outlining not only the results of their models, but much of the intuition behind these results too. This personally gave me a much richer understanding of the population dynamics, which I really appreciated. Moreover, the additional investigation of the two forms of cytoplasmic incompatibility in haplodiploids was incredibly interesting, and I think lends itself to some neat empirical tests. Generally, I think the paper is well written, clear, and correct, and so I see little need for substantial changes. The suggestions I do make, I hope the authors find useful.

Thank you!

Specific comments

Description of cytoplasmic incompatibility.

Whilst many of the readers of this paper may already be well acquainted with the workings of cytoplasmic incompatibility, nonetheless I feel like a small diagram would be of great help to succinctly explain the phenomena. This is particularly true for the two haplodiploid cases. Perhaps punnet square like crosses showing viable/non viable offspring (e.g. similar to Engelstädter & Telschow (2009), Figure 1)?

This is a good idea. We introduced an explanatory figure.

Fertilisation rate k

I thought that the parameter k (the fertilisation rate) could just be explained a little more. As far as I understood, this is just the proportion of eggs that a female allows to be fertilised? i.e. in a Wolbachia free population this would be the primary sex ratio? With the assumption that k might be much higher in an infected population as females attempt to counteract the sex-ratio effect?

We improved the explanation. The effect of *Wolbachia* on *k* is not known, but would be interesting to test with empirical data. Kawasaki et al 2016 (https://doi.org/10.1111/1758-2229.12425) found the haplodiploid species in

their study being female biased, regardless of their infection status, while *Wolbachia* infections were also more common among haplodiploids.

Masculinising vs female-killing CI

I thought that the differences between these two mechanisms of CI was very interesting, and thought that the results the authors presented might lend themselves to some clear empirical patterns, and so maybe these could be expanded upon a little more. Two points jumped out to me.

Firstly, from Figure 4, it appeared that the female-killing effect had a lower invasion threshold, and higher equilibrium frequency, than the masculinising strategy. This to me intuitively made sense, as by 'masculinising' the offspring from non-infected females rather than simply killing them, the infected males are diluted in the population, reducing their effects. Thus, one might expect those populations with female-killing CI to have higher Wolbachia loads than those with masculinising CI, although from Figure 5 it was difficult to see whether this was true. If this is correct, and the authors were able to expand upon this a little more – either in the discussion or in the haplodiploid section - then I think that would be worthwhile.

Nice explanation, this makes sense indeed! The *Wolbachia* frequency should be diluted when more non-infected individuals are produced. This seems to hold, looking from Figure 7 (old Fig. 6). The masculinization model produces slightly lower equilibrium frequencies (female infection frequency) than the female killing model with same parameters even with k=0.5 (comparing the first two rows of the fig, contours), and the effect is stronger with k>0.5. Additionally, the male infection frequency is even more lower than the female, which makes sense since they are "diluted" (darker green in the panels of masculinization model). We added this observation to the discussion.

Secondly, the sex-differences in the Wolbachia infection rate under haplodiploid CI seems a very interesting and clear empirical prediction, and a clear point of comparison to be made with the diplodiploid CI. Especially as the qualitative pattern of higher infection rates in females appears to hold pretty much under any of the specified parameters. I wondered if this point had previously been made, if not then I think is worthy of being made a bit more explicit, and worth noting any empirical work that had been done on this. This pattern might also provide a nice test of hypotheses to other explanations for low frequencies of Wolbachia, i.e. if low frequencies are maintained by gene flow with sex-biased dispersal.

Thanks! We added this to the discussion as well.

Sex-specific positive effects

As far as I can understand the positive (and negative) fitness effects investigated in this paper (and previous analyses) are exclusively restricted to females. I wondered whether this was worth the authors commenting on, and potentially speculating (or indeed modelling) the potential effects of positive and negative fitness effects falling upon males instead. Whilst I suspect that this would have relatively little consequence to the qualitative patterns previously shown (due to matrilineal nature of Wolbachia inheritance, and so is in effect a little bit more like just scaling L), I think it nonetheless

might be worthwhile, particularly as some of the potential sources of positive fitness effects may extend to both sexes, but nonetheless it is only the positive (or negative) effects that have any substantial bearing upon the ecological dynamics.

> This is an interesting possibility for extending the model analysis. The modifications to the current models should be relatively easy to do. However, it might well be a very minor effect as suggested. In any case, we prefer to keep it outside of the current manuscript.

Figures

On the whole I think the figures are clear and understandable. One comment would be that maybe Figure 5 could be explicitly split up into two figures (maybe restrict the male to the Appendix?), as I found it initially a bit of work to understand the male frequencies on the plot, and comes out relatively poorly in Grayscale.

> Thank you for the suggestion. We prefer to keep the figure with the main text as a whole, since both the female and the male frequencies are in the core of the presented analysis. This kind of figures presenting results across the parameter space are not the easiest ones to make sense.