Response to reviewers

We thank the editor and reviewers for their detailed and constructive analysis of our manuscript. A primary concern from multiple reviewers was that the manuscript both over-generalized the results and over-diminished the trade-off hypothesis. We agree with this sentiment, wholeheartedly. As of today, a mechanistic trade-off has been a necessary constraint in nearly-all models that can account for the evolution of the intermediate virulence strategies observed in parasites and pathogens. Our work presents an exception to this rule such that an alternative mechanism can account for intermediate virulence strategies under some conditions. Although the prior writing did not make this clear, we agree that mechanistic trade-offs are currently the only mechanism that can explain intermediate virulence strategies in the majority of disease systems. We have re-written much of the introduction and discussion to clarify these points. We have also added discussion of several examples of disease systems that conform to the assumptions of this model to specify the types of systems where our results suggest that phenology could account for intermediate virulence. We also discuss extensions to this modeling framework that could increase the range of systems potentially impacted by host phenology. Thus, we have re-written this manuscript to represent our current thinking: a mechanistic trade-off is still essential in most, but not all, disease systems to account for intermediate virulence strategies. We have addressed each of the comments from the reviewers and the editor. We have used **bold font** to distinguish our responses from the original comments.

Editor comments: The study has potential but it needs to be presented in the context of host-parasitoïd literature.

The authors investigate the evolution of parasite life-history trait in the context of a specific kind of host-parasite interactions. The originality of the study is that they include seasonal host activities to determine optimal parasite strategies.

The recommendations from the reviewers are balanced but I think they agree on several points.

First, it is important to clarify the terminology. Indeed, the parasite is here assumed to only be transmitted after killing the host. Such a life cycle is often referred to as that of an "obligate killer" and is known to greatly affect the constraints on virulence. This should be stressed very clearly (ideally in the title) and I think it also calls for strong rewriting of the Introduction.
Thank you for this comment. We agree that the terminology needed to be clarified and we state in the introduction and model description that the parasite species under study is an obligate-killer. We have re-written the introduction to address this comment and others. We also changed the title to better reflect the conclusions that can be drawn from the presented results.

Furthermore, I found the model notations difficult to follow, probably because many are missing in Table 1 (adding units in the table would also be a plus).
We apologize that the model notations were difficult to follow. All parameters are now de-

fined in Table 1 with units.

Finally, as pointed out by one of the reviewers, the definition of fitness is central and perhaps deserves a more careful justification.
We fixed the typos in the analytical solution for mutant invasion fitness and are confident it is correctly specified.

Second, the biological relevance could be improved. One of the reviewers suggested mentioning specific host-parasite systems for which this model could apply (especially with regards to some of the strong assumptions it makes). Discussing the parasitoïd literature also seems important given the key assumption of the model (parasites do not transmit unless they kill their host).
We agree that the relevance of this work would be much better supported with an increased discussion of the biological relevance. We now discuss and cite multiple systems that conform to the assumptions of the model as presented including Baculoviruses/*Lepidoptera*[1–5] and ichneumonids parasitizing univoltine insects.[6–8]

We have also added a discussion of extensions to this model that are unlikely to change the qualitative results and would extend the range of systems to which this model could be applicable beyond monocyclic obligate-killer parasites (line 228). Briefly, relaxing the obligate-killer assumption could still result in selection for intermediate virulence in some cases. That is, longer latency periods that result in progeny release near the end of the season would still be adaptive for parasites that reduce host fecundity or increase the host death rate. Longer latency periods are equivalent to lower virulence in this system as infected hosts have more time to reproduce and thus have higher fitness. This extension is not expected to qualitatively alter the results if the parasite transmission period is short relative to the season length. The assumptions of this extended model likely conform to many additional parasite-host systems such as soil-borne plant pathogens, demicyclic rusts, post-harvest diseases, and many diseases systems involving univoltine insect hosts.[9–12] Although speculative, this discussion broadens the potential impact of host phenology on virulence evolution and could motivate future research in this area.

Third, related to the previous point, many important references from the literature seem to be missing. The reviewers mentioned relevant articles on parasitoïds and on seasonality. I would also add the study by King et al (2009, Am Nat, DOI: 10.1086/597217) about persistence constraint.
We agree with this comment and have included a discussion of the literature suggested by the editor and the reviewers throughout the discussion.

Reviewer #1: The study investigates the impact of host phenology on the evolution of virulence using the evolutionary invasion analysis. The study finds that the duration of the host activity period and synchronicity of the host emergence timing influences virulence. The authors present a mathematical model that is biologically interesting while being mathematically tractable. The manuscript is concise and

very well written. Below I outline my comments regarding the interpretation of modelling assumptions and provide minor suggestions.

I appreciate that the introduction provides a thorough general context to the virulence evolution. Having said that, the modelled system is a fairly specific one: a plant virus that is environmentally transmitted. The readers may benefit from a further background of the modelled system as the majority of the literature on evolutionary epidemiology employs rather generic direct transmission models. For example, it might be useful for the readers to know that environmental transmission lowers the cost of host exploitation in comparison to direct transmission.

Thank you for this comment. We agree that the modelled system is specific and now state in the introduction and model description that the model represents an obligate-killer parasite. We also agree that it is useful to discuss that our results show that seasonal host absence is a factor that can increase the benefit of remaining in hosts for environmentally transmitted parasites by decreasing environmental exposure. We now state this on line 181.

More importantly, the parasite considered in this manuscript is, in an ecological sense, a parasitoid.

We agree that our model applies to parasitoids such as ichneumonid wasps but it also applies to other systems such as seasonal baculovirus outbreaks in univoltine insects in cases where the baculovirus is monocyclic.[1–5]

I believe that the fact that the parasite needs to kill the host to complete transmission is a form of trade-off – not unlike the classic virulence-transmission trade-off – that selects against avirulence. As such, I tend not to agree with the assertion made by the authors that they investigate virulence evolution "in the absence of any explicit mechanistic trade-off," (L38). If this claim is crucial to the main thesis of this manuscript, I recommend the authors provide further clarifications as to how the "kill-to-transmit" does not qualify as an evolutionary trade-off. Specifically, the readers may benefit from discussions on whether the key findings are applicable outside "parasitoid" systems.

Thank you for these comments. We agree with the reviewer that transmission for obligate host-killer parasites requires host death, an obvious byproduct of virulence. However, the transmission-virulence trade-off is defined by the constraint that the transmission rate (infectious parasites released per infected individual) cannot increase without a correlated increase in virulence (time to host death in this model) due to the mechanistic link between the two. In the current model, the number of infectious parasites released from each infectious individual remains constant regardless of the level of virulence, such that the transmission rate and virulence are not mechanistically linked in this system. In the absence of any other selective pressures, killing the current host as quickly as possible would be advantageous for an obligate killer-parasite to maximize transmission opportunities in new hosts. In contrast, we find that seasonal host absence can serve as a major selective force that drives the evolution of intermediate virulence in an obligate-killer parasite, at least in systems that conform to the assumptions of our model. We have edited the manuscript considerably to limit generalizations of our results beyond what the results can support.

We have also added a discussion of extensions to this model that are unlikely to change

the qualitative results and would extend the range of systems to which this model could be applicable beyond monocyclic obligate-killer parasites (line 228). Briefly, relaxing the obligate-killer assumption could still result in selection for intermediate virulence in some cases. That is, longer latency periods that result in progeny release near the end of the season would still be adaptive for parasites that reduce host fecundity or increase the host death rate. Longer latency periods are equivalent to lower virulence in this system as infected hosts have more time to reproduce and consequently higher fitness. This extension is not expected to qualitatively alter the results if the parasite transmission period is short relative to the season length. The assumptions of this extended model likely conform to many additional parasite-host systems such as soil-borne plant pathogens, demicyclic rusts, post-harvest diseases, and many diseases systems involving univoltine insect hosts.[9–12] Although speculative, this discussion broadens the potential impact of host phenology on virulence evolution which could motivate future research in this area.

In the paragraph starting at L111, the readers may benefit from explanations with more specific references to different parts of the figures (e.g. Figure 3A,ii).

Thank you for this suggestion. We now reference specific parts of Figure 3 in this paragraph and the paragraph following it.

I appreciate the authors' intent for transparency, but I noticed that the Github link is not working as of March 30th 2021.

Sorry for the confusion. If the link is copied directly from the manuscript with line numbers, the line number is inserted into the Github link. The correct link is: https://github.com/hannelorem phenology-drives-the-evolution-of-intermediate-parasite-virulence

Reviewer #2: This preprint addresses the evolution of the latent period of monocyclic parasites in a seasonal environment. Hosts have non-overlapping generations and are alive for one season. The latent period is defined as the time between host infection and the release of free-living forms that may survive and infect hosts of the next season/generation. Monocyclic parasites can perform only one round of infection per season. That is, there are no secondary infections (host-to-host contaminations) within a season/generation. The authors show that if hosts emerge synchronously at the beginning of the season, then the optimal latent period is roughly equal to the length of the season. This is because survival as a free-living form is less likely than within-host survival. However, if the host emergence time is spread over a time interval, the optimal latent period is shorter. It is roughly equal to the mean time between host emergence and the end of the season.

I have several concerns with this preprint:

1. The authors interpret their results in terms of virulence rather than latent period. I realize that for obligate killers the latent period is the same as the time to host death (the inverse of virulence), but obligate killers are a very specific case. The authors should not draw general conclusions from such a very

specific case.

We agree completely with this statement. There are several statements in the original manuscript that appear to make more generalized conclusions than are warranted by the results. The primary conclusion that our data do support is that a mechanistic trade-off (*e.g.* instantaneous transmission rate increases as virulence increases) is not *always* necessary for intermediate virulence to evolve. We agree with the reviewer that the latent period (time to host death) is the metric of virulence in this system, which is not generalizable across systems. Thanks to this and other related reviewer comments, we have added a discussion of extensions to this model that are unlikely to change the qualitative results and would extend the range of systems to which this model could be applicable beyond monocyclic obligate-killer parasites. We hope this speculative discussion will motivate future research in this area.

Moreover, the authors do not provide a single example of an obligate killer parasite which would be monocyclic, and that would infect a host with non-overlapping generations.

We agree that the relevance of this work would be much better supported with an increased discussion of the biological relevance. We now discuss and cite multiple systems that conform to the assumptions of the model as presented including Baculoviruses/*Lepidoptera*[1–5] and ichneumonids parasitizing univoltine insects.[6–8]

Furthermore, the authors argue that there is no trade-off (unlike previous studies on the evolution of virulence) while there is by essence a trade-off in obligate-killers, since they cannot transmit without killing their host. The sooner they kill their host, the sooner they can infect new hosts in principle. Unfortunately, obligate killers are one of the few examples clearly supporting a trade-off between transmission and virulence. The authors may argue that this trade-off does not hold for monocyclic obligate-killer parasites, which leads me to my second point.

We agree with the reviewer that there is good theoretical and empirical support for mechanistic trade-offs in obligate killer parasites. The reviewer's explanation of how a negative correlation between instantaneous transmission rate and virulence can result in the evolution of extremely high virulence when the parasite is not monocyclic is also spot on. We argue here that a mechanistic trade-off between virulence and instantaneous transmission rate (or other transmission related trait) is not essential to select for an intermediate virulence strategy in monocyclic, obligate-killer parasites. We have edited the manuscript considerably to limit generalizations of our results beyond what the results can support.

2. The authors assume that there are no secondary infections without any justification (not even a single example clearly checking all model assumptions). The naïve reader may believe that the focus on monocyclic parasites was made for simplicity only.

We agree that this work is relevant only as it applies to real biological systems, several of which we now discuss and cite.

However, this assumption is all but unimportant. If parasites can perform secondary infections, then parasites that infect early hosts can also infect later hosts and preempt infections from a parasite that would be more prudent and would keep alive its host longer. This would select for increased virulence despite the fact that this strategy does not maximize survival at the end of the season (sort of tragedy of the commons).

We agree that altering or relaxing this assumption would lead to different behavior. In particular, the reviewer's intuition that high virulence is adaptive for polycyclic parasites is correct. In follow-up work we are finding that relaxing the assumption that parasites can only complete one round of infection per season results in bistability of parasite virulence strategies. We are finding that it is adaptive for parasites to kill their host and release new parasites just before the end of the season (same strategy as found in the current manuscript) and that a high virulence strategy that can complete multiple infection cycles within the host activity period season can also be favored in many cases.

3. The authors do not cite previous studies on the topic (van den Berg et al 2011; Hamelin et al 2011), which take into account secondary infections, and different trade-offs between transmission and virulence, and transmission and survival. Some of the results are reminiscent of these studies.

We agree with the reviewer that the study by van den Berg et al 2011 is an important study to discuss in this manuscript. We now discuss this study, as well as several others noted by the editor and other reviewer, to better place our results in context.

4. The mathematics are unclear:

We thank the reviewer for their careful attention to the mathematical derivation. We went over the maths carefully, clarifying and fixing several typos.

4.1. The initial conditions are never specified. Around equation (1), it should be specified $s(0) = 0, v1(0+) = v2(0-)$ (or any other notation dealing with the discontinuity), $v_2(\tau) = 0$.

We thank the reviewer for catching this. We have added the initial conditions to line 54.

4.2. In equation (2), gamma has never been defined.

This was a typo; γ was not meant to be in this equation. We have deleted it.

4.3. In equation (3), if $\alpha = 0$, one should recover the equation expressing $v_2(T)$ on page 3, right? That does not seem to be the case.

Thank you for noticing this typo, now corrected. In the correct version of Equation 3, we do indeed recover $v_2(T)$ when $\alpha = 0$ as expected.

4.4. In equation (3), s1 and v* have never been defined.

Another typo: $s1$ should have been s. We now define $\hat{v}^*$ in line 86.

4.5. It is unclear whether evolution maximizes (3) (see Lion and Metz 2018 for a review). For an optimization principle to hold, one must show that the invasion criterion of the mutant depends in a one-dimensional way of the resident, which is all but obvious from equation (3) (in which there are several implicit functions). However, I guess this is true when tau is the only trait that differs between the resident and the mutant.

We agree that this may not have been clear, however we are confident that (3) is the maximand in an optimization principle. Specifically, in system (2), the resident and mutant parasite only impact one another through competition for susceptible hosts such that the mutant dependence on the resident is one-dimensional. Regardless, equation (3) incorporates all the environmental effects of the resident pathogen on the host population, so the quantity $v_{2m}(T)$ corresponds to $\mathcal{R}$ in Lion and Metz's notation, rather than R_0. Therefore, it is a proper maximand. We now make this point clear after equations (4) in the text.

4.6. Where is the difference between equation (3) and equation (5)? When β_m differs from β, there is likely no optimization principle although an ESS indeed satisfies (4) (if correct). I assume that the authors performed correct computations, but they should be much more careful and explicit in describing what they did (at least in appendix).

The only difference between (3) and (5) is that β_m is constant in (3) while in (5) $\beta_m(\tau_m)$ is a function of τ_m. As we mention above, since $v_{2m}(T)$ takes into account the environment created by the resident, it can still be used as a maximand in an optimality calculation.

4.7. In appendix A, the first row of equation v_2 should be defined for $\tau < t < t_l$ (not $0 < t < t_l$). This would have been clearer if you had specified initial conditions, i.e. $v_2(\tau) = 0$.

We added initial conditions to Appendix A. We also fixed v_2 so that it is now correctly defined from $\tau < t < t_l$ and then from $t_l < t < T$.

4.8. In appendix A, there is a mistake in the second row of equation v_2, which is defined for $t = t_l$ and depends on the future ($v_2(t_l + \tau)$). I think there is a mess with the handling of the delay (τ), which is the trait under consideration. Consequently, I cannot be confident in the central equation (3).

Thank you for catching this. We fixed v_2 so that it is now correctly defined from $\tau < t < t_l$ and then from $t_l < t < T$.

4.9. Appendix B does not bring much compared to the text (and contains the same typos or mistakes). One would expect much more rigor and details in the appendices.

Thank you again for catching the typos. We have fixed them.

While I do not think the above possible mistakes would qualitatively change the results (which are expected), I am not 100% confident that the mathematics are correct.

Other remarks:

- Page 7, line 107: By contrast?
  We apologize, but we are not sure what the reviewer's issue is with this phrasing.

- Page 10, lines 160-161: I do not agree with "this phenomenon could occur more broadly than the specific system investigated here"
  We agree that this and several other statements suggested that the conclusions are generalizable to more systems than the results suggest. We have re-written the majority of the manuscript to qualify or remove these types of statements.

- Page 10, lines 167-168: "avirulence is maladaptive as..." is simply an assumption of the model (obligate-killer parasite)
  We agree that it is a model assumption that avirulence is maladaptive. We have rewritten the sentence as " Low virulence is maladaptive for parasites in this system as they do not kill their host before the end of the season and create no progeny which results in no evolutionary fitness." (now on line 178).

- Fig. 3, panel B: one of the ii's should be replaced with iii
  Thank you, we fixed this in Figure 3.

- Page 12, line 191: the reference to *T. parva* comes out of nowhere
  We apologize that the link between our work and *T. parva* was not clear. This is an empirical example where increasing the time between transmission events (similar to season length in our model) can select for decreased virulence in a different type of system.

Overall, although I have been quite critical in my review, it might be that the authors could revise their manuscript to make it recommendable. I would recommend tuning down the results and even changing the focus of the manuscript. If the host-parasite species the authors have in mind are e.g. baculoviruses of forest Lepidoptera, they should clearly describe how their mathematical model fits these species, and introduce the research question with respect to these species only. The results would be much less general, but much more convincing, especially if they challenge previous ideas concerning these species (which remains to be shown, as the results are quite intuitive thus far).

References

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[11] Ivan H Crowell. The hosts, life history and control of the cedar-apple rust fungus gymnosporangium juniperi-virginianae schw. *Journal of the Arnold Arboretum*, 15(3):163–232, 1934.

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Reviewer #3: The manuscript examines a model of host-parasite interactions where hosts have non-overlapping generations and a fixed 'emergence period' each generation. Parasites must infect hosts and have produced progeny before the end of the season for those progeny to be carried forward to the next generation. The focus of the study is on how the parasite might evolve the duration of infection to maximise its fitness. The key result is that even without any trade-off, an intermediate infective duration - linked to virulence - is selected for.

The manuscript is really nicely written and the results presented pretty clearly. The code is made available, which is great (I don't know how many people use Mathematica, but still, it's there). The existing literature is mostly well covered, but there are a few papers I think should be added. I've enjoyed reading the preprint and it got me thinking, which is perhaps the biggest compliment I can give.

Below are some mostly minor comments.

- P2 L16 - The main empirical example in the cited paper, that of myxamatosis, actually considers a recovery-virulence trade-off. I wonder if it is worth tweaking this sentence to stress that.
  Thank you for catching this, we changed the sentence.

- P3 below L54 - This is the first time you have referred to 'the resistant life stage'. Please could you expand on what you mean.
  Thank you for bringing this up. We added a clarification in the previous paragraph on line 46 that the host progresses to a later developmental stage that is not susceptible to parasite infection.

- P3 above L55 - I'm trying to decompose the parasite progeny production term. Am I right that it is the product of:
  - the density of newly infected hosts at $(t-\tau)$ [the $\alpha(s(t-\tau) * v1(t-\tau)$ term],
  - the density (number?) of parasites produced upon *parasite-induced* death [beta],
  - the number of hosts that survive infection between $t-\tau$ and t [the $\exp(-d*\tau)$]?
- If this is correct, I have a couple of questions:
  - Why do infected hosts who died from background mortality not release parasites into the environment?
    Thank you for bringing up that we did not explain this assumption. We assumed that background mortality arises from predation, herbivory, or some other natural cause. We assume that infected hosts that die from background mortality do not release parasites because the parasites are either consumed or the latency period corresponds to the time necessary to develop viable progeny. We added a justification for this assumption on line 68.

  - Does this imply that there is a sort of 'programmed death' type situation, where once beta parasite progeny are ready, the parasite kills its host? This value (β) is fixed for most of your study, so in effect it is parasite growth rate that evolves - how slow or fast does the parasite reach this magic number of beta. In that sense I see the link with more classic definitions of virulence as simply mortality rate, where faster growth leads to higher mortality. But it is still a bit different.
    Yes, you are correct. We added an explanation in the model description starting on line 90.

- P3 L61 - Does it make sense for $g(t,t_l)$ to be a PDF in terms of units? Assuming $\hat{s}$ is a density doesn't $g(t,t_l)$ need to be a per-capita rate for the units to balance?

We apologize for not explaining this more clearly. You are correct, $g(t, t_l)$ describes host emergence as a function of time. We clarified this on line 61.

- P5 L77 - Gamma has appeared in the equations which wasn't there before. Please add a definition.

  Thank you for catching this. We deleted all the γ terms as they were there on accident.

- P5 L87 - Since fitness is a function of both resident and mutant traits, it is usual to denote the fitness derivatives as partial derivatives.

  Thank you, we corrected the notation.

- P7 L95 - 'The duration of host activity can select against....' is a bit vague. I guess you mean 'Longer durations of host activity can select against...'?

  We agree that our original phrasing was vague. We changed this sentence to "Temporally constrained host activity periods within each season can select against both extremely high and extremely low virulence levels resulting in an intermediate optimal level of virulence", now on line 106.

- P7 L110 - This linear relationship is interesting. Does it imply that the parasite's best strategy is just to ensure (mean) death at a fixed time before the season ends?

  Yes, you are correct. We now state this more clearly in the results section on line 121.

- P7 L111 - Is t_l really the 'time at which each host first becomes active'? In what sense does it become active then? Is it not simply the length of the emergence season? Similarly, in figure 3 the x-axis label of 'host emergence concentration' isn't right I think - it is the *inverse* of this isn't it? Again, in my head it makes more sense to think of it as the duration of emergence.

  We agree that the language we used is confusing. t_l is a quantitative measure of how synchronous host emergence is; that is, a metric of variation in the time at which each host first emerges within a season. Small t_l corresponds to the entire host cohort entering the system (or becoming active and available to be infected) over a short time period. By contrast long t_l corresponds to a slower emergence rate and thus less synchronous emergence as a cohort. We changed the x-axis in Figure 3 to 'host emergence period length'.

- P12 L202 - In terms of discussion points, I find myself mulling over how much of your results rest on the model assumptions. I think the non-overlapping generations in particular has quite a big impact, as well as the nature of quite what virulence is in your model (effectively growth rate). As you describe, your key result stems from the combination of factors that parasites have to ensure they have produced progeny before the end of the season, and that they can only release those progeny all in one go. It seems likely that under different set-ups - fully continuous-time with

overlapping generations, for example - this results might disappear? I wonder if you might comment on other models of parasite evolution in seasonal environments where generations are overlapping. I'm particularly thinking of Koelle et al. (2005), Sorrel et al. (2009) and Donnelly et al. (2013) (all in Proc Roy Soc B). My reading of these is that you wouldn't get intermediate virulence without a further trade-off, though I'm not sure any specifically say it.

These are all great points and we thank you for suggesting we discuss these studies. Our results are driven by the fact that there are periods of host absence and thus the parasite needs to transmit within a set period of time. We agree that a fully continuous-time model with overlapping generations will likely require a trade-off for intermediate virulence to be adaptive, or at least we know of no work contradicting that result. We now discuss on line 214 the implications of overlapping parasite and host generations in context with Donnelly *et al.* and Sorrell *et al.* It would be interesting to study how environmental decay of released parasite progeny and potentially competing forces such as density dependent virulence would impact the optimal virulence strategy.