

Review of manuscript
“Impact of ploidy and pathogen life cycle on resistance durability”
DOI <https://doi.org/10.1101/2021.05.28.446112>

In this article, Saubin et al. developed a simulation model to evaluate plant resistance durability for different pathogen ploidies and life histories. In particular, analyses confront haploid versus diploid pathogens, as well as absence versus presence of an alternative host (in addition to both susceptible and resistant hosts). The model is stochastic, non-spatial and accounts for pathogen evolution via selection, genetic drift, and sexual reproduction. Results highlight crucial differences in the epidemiological and evolutionary dynamics between haploid and diploid pathogens: the latter being subject to higher stochasticity and probability to extinction due to the counter-selection of heterozygous individuals in resistant hosts. The presence of an alternative host speeds the probability of invasion of resistant hosts by virulent pathogens, but slows their subsequent establishment.

The durable deployment of plant resistance is a hot topic which has been explored via numerous models. However, very few of them explored the case of diploid pathogens or the impact of alternative hosts where sexual reproduction may occur (as highlighted in one of my own article: Rimbaud et al. 2021. Models of plant resistance deployment. Annual Review of Phytopathology 59: in press). Yet, these features are potentially very influent on the epidemiological and evolutionary trajectories of the concerned pathogens (e.g. potentially numerous fungi). There is thus a gap in the scientific literature, and this article is a very nice contribution to fill it. The model is sound, results and conclusions are interesting, and the text is well written. I am very convinced by this study and only have minor comments to make to further improve the manuscript.

Loup Rimbaud

General comments:

- the word “virulence” has different meanings depending on the scientific area, I suggest to give a definition at first occurrence.
- In this work, resistance durability is defined in several ways:
 - apparition of a virulent pathogen in resistant hosts (line 64-65)
 - time to fixation of the virulent pathogen (line 242). “Fixation” must also be defined (I supposed it is when the frequency of the virulent pathogen exceeds a threshold? If yes, this threshold must be explicit)
 - Invasion of the resistant host (1/1000 prevalence on resistant hosts)
 - Resistance breakdown (1/100 prevalence on resistant hosts). Is there a reason for the 1/1000 and 1/100 thresholds or is it an arbitrary decision?

I think the computation of these metrics should be more explicit in the text. In addition, the two last definitions are described as two components of durability, so the status of the two first metrics is not clear.

Line by line comments (suggestions for additions are in red)

Abstract

- #14 “a major component of deployment host strategies”: remove “host”

Introduction

- #42 “...the result of the infection is determined by **the interaction between** a locus in the plant...”
- Paragraph #44 to #51 needs to be slightly rephrased or re-organised. In lines #44 to #46, if the product of the avirulence gene is not recognised, it may be because the pathogen is virulent. So infection occurs, but the plant is actually resistant (broken down, though). Additionally, in lines #49 to #51, there is a mention of coevolution (which occurs in natural systems) whereas the beginning of the paragraph started with the breeding of resistance (thus cultivated crops).
- #57-58 “strategies like resistance deployment hinder this maintenance of polymorphism”: here I don’t think we can say it is a strategy, and it is definitely different from the strategies destined to improve durability. I would rather say “monocrops”, or “pure crops of resistant hosts”.
- #64-65 “the expected time until the apparition of a virulent population of pathogens on resistant hosts (Johnson 1984)”: I am ok with this definition but this is not the same as Johnson’s one (“expected time during which a cultivar stays efficient in spite of an environment favourable to disease”).
- #116 “Therefore, **in the absence of data**, it could be more insightful not to consider fitness cost...”. I also suggest to replace “insightful” by “conservative of the risk of breakdown” (because accounting for the fitness cost is highly relevant to pathosystems where there is one!).
- #127 it seems a bit contradictory to focus on simulations where the pathogen population “went extinct” to highlight “evolutionary rescue events”.

Model description

- #155 “the population size is considered constant”: do you mean before/after the reproduction event, or throughout the simulated period?
- #163-165: I suppose you assume here that all pathogen genotypes have the same reproduction rate. It could be worth mentioning it.
- #171-172: “even if the number of individuals on this compartment reached the maximum carrying capacity” seems contradictory with “...restricted to the carrying capacity at each reproduction event” in the sentence after. Please clarify.
- #183: on alternative hosts, there are two reproduction events (sexual and asexual). Is there a reason for simulating an asexual reproduction here?
- Paragraph starting #205: it is important to mention that homozygous avr-avr individuals are initially present.

Results

- #257: it could be worth mentioning here what are the sources of stochasticity (reproduction, migration, input parameters).
- #278: “axes” (instead of “axis”)
- #289-290 “higher mean generations of avr fixation”: maybe simplify by “longer time to fixation”?
- #356 replace “the GLM results” by “the results of GLM” for fluidity

Discussion

- The discussion is very interesting!
- #378-379 “as soon as one virulent individual invaded the resistant compartment, it was selected and the resistance breakdown occurred”. This is also because the bottleneck between seasons is large (and thus imposes soft genetic drift) and there is no host spatial structure (that could be amenable to more stochasticity and extinctions).
- #391 replace “cannot” by “poorly” and replace “only” by “mostly” (because homozygous avr-avr individuals exist).
- Paragraph #436 to #438: given Figures 6 and 7, I would have said the opposite (host alternation leads to more stochastic evolution of allelic frequencies and slower speed of virulence fixation).
- Paragraph 454 to 458: this situation may lead to high level of genetic diversity in pathogen population, as shown in a study which investigated the case where the time to immigration (“invasion” here) is well shorter than the time to colonisation (“breakdown” here) :
Wingen LU, Shaw MW, Brown JKM. 2013. Long-distance dispersal and its influence on adaptation to host resistance in a heterogeneous landscape. Plant Pathology 62: 9-20
- #523: for pyramids, the presence of an alternative host increases the probability of encounter of mono-virulent pathogens, and thus the probability of appearance of multi-virulent pathogens able to overcome the pyramid. It may be worth mentioning it.

Figures

- Figure 1: I suggest to indicate when (or how often) does migration occur close to the arrow, or to specify this in the caption.
- Figure 2: why freq_avr is not negatively correlated with freq_Avr?
- Figure S1 (caption): “Sobol’s indices” (with a capital letter)
- Figure S2: I hardly see the 5th colour...consider limit to 4!
- Figure S3: year of “invasion” (instead of “invision”).
- Figure S5 and S6: is the left part the same as in the main document? If yes, please mention it.