The manuscript describes a simulation study of the effects of deployments of ontogenic resistances on disease development and pathogen evolution in cropping landscapes. The model used is strongly inspired by the case of wheat leaf rust caused by *Puccinia* fungal pathogens, but its predictions seem applicable to several other pathosystems. To my knowledge, this paper is a novel contribution in the field of evolutionary epidemiology. I found the paper interesting and the scenarios considered relevant. The methods seem also robust. I start this review with a quick summary of the ideas under consideration.

The model proposed and explored by the authors was implemented from an upgraded version of *landsepi*, a spatially explicit and demo-genetic modelling framework described in earlier papers. A thorough exploration of the model is conduced through three complementary main scenarios. Interestingly, the authors simulated, in each scenario, different times to resistance expression, varying degrees of efficiency of the resistance deployed, different levels of aggregation of resistant fields, varying levels of fitness costs undergone by the pathogen on the different cultivars, and different pathogen traits. In addition, effects of combinations of the adult plant resistance with a major gene resistance on disease development and resistances durability were investigated. As a consequence, the number of simulations realized and analyzed is very large.

Adult plant resistances impose an abrupt change in the selection pressures undergone by the expanding pathogen population. Importantly, depending on the time to resistance activation, such resistances become effective at more or less advanced stages of epidemics development within ongoing cropping season. Hence the size and the genetic composition of the pathogen population facing resistance activation strongly vary depending on the time to resistance expression. Note that unlike previous studies that accounted for the intensity of epidemics before resistance deployment (*e.g.* Fabre et al. 2015, Evolutionary Application; Djidjou Demasse et al. 2017, New Phythologist; Rousseau et al. 2019, Philosophical transactions of the royal society B), here gene resistance can be activated in hosts already infected by the pathogen. As indicated by the authors, this study is reminiscent of other studies focused on applications of chemical treatments against spreading pests and pathogens.

One important insight generated by the model is that the durability of adult plant resistance is globally decreased by the efficiency of the resistances (as predicted earlier), but increased by the time to resistance gene activation and the magnitude of the fitness costs undergone by the fungal genotypes. Similarly, the invasion of resistant and susceptible fields by the pathogen is constrained when substantial delays of resistance expression and relatively high efficiencies of adult plant resistance are simulated. Indeed, delaying the expression of the resistance allow genotypes of ‘wild type’ (not adapted to resistance) to infect many resistant cultivars before resistance activation. However, as the resistance activates wild type genotypes have almost no chances to further propagate into resistant fields. In addition, at this point, mutant genotypes (able to break the resistance) have also few chances to reach healthy resistant hosts, because (*i*) mutant genotypes are not frequent in the pathogen population when fitness costs are assumed and (*ii*) many resistant hosts are infected by wild type genotypes.
Although the paper was principally focused on adult plant resistance targeting the infectivity of the pathogen, parameters combinations leading to similar outcomes were also identified when other pathogen traits were targeted by the resistance, or when the adult plant resistance was combined with a major gene resistance.

I have only minor suggestions for improvement:

My main concern is that I find the current manuscript difficult to follow in places. The introduction is fine but the methods are too succintely explained. Current model description contains a short mix

of statements that do not allow the reader to understand and evaluate the model. I agree with the authors that there is no need to give, again in this new paper, all the details of the model. Yet, the model proposed in this paper is not exactly the same as the model by Rimbaud et al. (2018 c). Adding more information about the composition of the fields, the traits targeted by the resistance, the host pathogen interaction, and mutation is necessary (see some remarks below). Some elements, essential for model understanding, are given in Results section; they should be moved in Methods section.
As the model proposed is complex and the experimental design ambitious, it is not easy for the reader to get an integrative picture of all the results produces. That being said, the manuscript does a good job with regard to showing and commenting disease development in susceptible and resistant fields, and resistance durability. But I find the results section lacks of a more detailed presentation of the total damages caused by the pathogen (as given for instance in figure S5), that could help in the interpretation of the results, and that would inform the reader about the overall yield of the landscape depending on the deployment strategy implemented.
In the discussion, the authors propose comparisons with existing litterature and descriptions of the mechanisms leading to the patterns predicted. The discussion is overall efficient, although the part about the combination of major resistance genes with adult plant resistance genes is not easy to follow. The mention of a competition effect among the different pathogen strains was also sometimes a bit vague, as it does not always give the reader a good understanding of what happened in the simulations. This overall feeling may be the consequence of an insufficient understanding of the model.

Below are my specific comments on the paper:

l. 106, 108, and further. «hard» and «soft» selection recalls the dichotomy proposed by Wallace (1975, Evolution) (see also Reznick, 2015, Heredity)). If the aim of the authors is to quantify the strength of the selection undergone by the pathogen, I recommend to use « strong » and « weak » selection, largely used in the litterature (*e.g.* Whitlock 2008, Molecular Ecology).

l. 108. The constraint imposed on pathogen populations by delayed resistances and partial resistances are not exactly the same. Delayed resistances induce, at the plant scale, a sudden change in the direction of the selection pressure, with an intensity that can be strong. By contrast, partial resistances impose constant but relatively weak selection pressure on the pathogen population.

l. 131. As sexual reproduction is not simulated here, I would remove the beginning of the sentence.

l. 136. Unlike the model by Rimbaud et al. (2018c), I understand that there is here no aggressiveness quantitative component, is it correct? Few sentences describing the traits, the interaction between host and pathogen and how mutation affects the expression of the traits are necessary here. For instance, does the transformation of a ‘wt’ genotype into a ‘rb1’ a genotype or a ‘rb12’genotype require the same number of mutations?

l. 142. I understand that each field is assumed to be perfectly mixed, and that the density of hosts is assumed to be globally homogeneous. Therefore, the probability that a fungal genotypes carried by a spore reaching field *i* penetrates an healthy host depends on the proportion of healthy hosts in field *i* and the composition of the cloud of spores. I would add these important elements to the text.

l. 177 to 181. This part is also at the heart of the model and not specific to the experiment. I suggest to transfer these lines to model description. How resistance activation operate on latent period duration is well explained, but how does resistance activation influence other traits ? For instance I

am not sure to understand correctly: what happens when the resistance targeting pathogen infectivity is activated within a host already infected? Nothing?

l.186 : « off season survival » is mentionned for the first time here. Mentionning this important component of the model earlier in overall model description (l. 144?) would facilitate the overall understanding of the structure of the model.

l. 250 to 258. I would move these lines to Methods.

l. 285 to 287. I would move these lines to Methods.

l. 304 « Critical zone ». Please define here this term, using for instance the definition given l. 465.

l. 306. Unremarked on here is the fact that the overall epidemiological control (damage in S and R fields) is worst when the activation of strong resistance is delayed (If I understand correctly figure S5...). My impression is that this result, difficult to extrapolate from the main figures, deserves more comments.

l. 354 to 361. Should be explained in Methods section.

l. 426 to 434. These sentences about the generality of the predictions could be moved to the last paragraph of the discussion.

l. 446. Is there a difference between the severity of epidemics and the level of epidemiological control monitored in the simulations?

l. 462 and l. 486. As mentioned by the authors, the infection of many resistant cultivars (before resistance activation) decreases the quantity of available host tissue, because of many infections and as a consequence of an indirect effect on hosts growth (equation 2 Rimbaud et al. 2018 c). After resistance activation ‘wt’ genotypes have few chances to infect resistant hosts, while both the frequency of ‘rb’ genotypes in pathogen population and the availability of healthy resistant hosts are low. I agree with the authors that there is, formally, competition between the two pathogen strains, but, If my understanding is correct, this competition takes different forms depending on the delay of resistance expression, the efficiency of the resistance, the mutation rate and the range of dispersal of spores. As this competition effect is mentionned several times in the discussion, I think the paper would benefit here from a slightly more mechanistic and detailed description of how competition occurs.

l. 551 « hard selection » should be replaced by « strong selection ».