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Modelling the effect of gene deployment strategies on durability of plant resistance under selection

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ABSTRACT

Genetic resistance in crop plants is a cornerstone of disease management in agriculture. Such genetic resistance is often rapidly overtaken due to selection in the pathogen population, resulting in an arms race between plant breeders and the pathogen population. Here we ask whether there are strategies that can prolong the useful life of plant resistance genes. In a modelling study we compare three basic strategies: gene pyramiding, sequential use, and simultaneous use, and combinations of these. We furthermore explore the effects of fraction of host area, fraction of resistant host and the threshold fraction of virulence in the pathogen population at which resistance is considered overtaken on the useful life of resistance genes. We found that pyramiding is not always the most durable solution. Model results indicate that the most durable deployment strategy depends on the threshold fraction at which resistance is considered overtaken. This threshold fraction will depend on the economic value of the crop, and whether damage is acceptable. Pyramiding is only the most durable solution if the threshold is low. Otherwise, simultaneous use of single-gene resistant varieties is the most durable solution.

1. Introduction

Using genetic resistance is a cost-effective and environmental friendly way to control plant diseases. Plant resistance genes are known for numerous plant-pathogen systems, including pathogenic fungi and oomycetes, viruses and bacteria (Dangl and Jones, 2001). Major resistance genes play an important role in preventing disease in cereals like wheat, rice and maize, but also in potato and tomato (Ballvora et al., 2002; Gururani et al., 2012; Miah et al., 2013; de Vallavieille-Pope et al., 2012). Host plant resistance to plant pathogens is one of the key components that can be used to prevent disease in an Integrated Pest Management (IPM) system. IPM is designed to minimize pesticide use through the complementary use of alternative methods to control pests, diseases and weeds. There are two types of genetic resistance, complete resistance (or major resistance genes) and partial resistance (QTLs). Complete resistance inhibits growth of pathogen phenotypes that don't possess the matching virulence gene, while partial resistance does not completely restrict colonization by the plant pathogen but limits pathogen growth and reproduction, thereby limiting damage of the crop. Plant resistance genes, however, may be overtaken due to selection in the pathogen population, making the major resistance genes ineffective against the virulent genotype at substantial cost to growers, breeders and society at large (McDonald and Linde, 2002; Parlevliet, 2002; Zhan et al., 2014). Selection pressure for resistance breaking genotypes in agricultural systems is strong, due to cultivation of genetically uniform crops across large areas this has been a serious concern for decades and is still a serious problem to date (Buddenhagen, 1977; Fisher et al., 2012; Parlevliet, 2002; Stuckenbrock and Bataillon, 2012; Stuckenbrock and McDonald, 2008; Zadoks and Kampmeijer, 1977). Furthermore, plant resistance genes are a scarce resource, therefore it is important to use them prudently, i.e. in a way that increases their durability. There is a keen interest in strategies to prolong the useful life of plant major resistance genes.

Here, we use modelling to explore the factors that affect durability of resistance in different deployment strategies in a selection context. Modelling can elucidate emerging outcomes by embedding selection for virulence in a dynamic framework for trait selection in the pathogen population (Leonard, 1977). Plant pathologist and entomologist have devoted considerable time to study whether pyramiding (stacking two or more resistance genes in one variety), sequential use or use of multilines or cultivar mixtures is the most durable deployment strategy to prolong useful life of plant resistance genes (Gould, 1986; Kiyosawa, 1982; Leonard and Czochor, 1980). Gene pyramiding is generally considered the

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most durable solution with many known successes (Mundt, 2014). Molecular breeding techniques have identified many resistance genes and QTLs. Moreover, DNA markers techniques provided a rapid tool to select for multiple resistance genes without phenotypic disease screening of the progeny (Ashkani et al., 2015). Molecular techniques have facilitated the development of varieties with stacked resistance genes (Ashkani et al., 2015; Collard and MCKill, 2008; Mundt, 2014). The question remains whether pyramiding is the best solution for all situations.

Most models in plant pathogen evolution assume that there are fitness costs associated with loss of avirulence (i.e. stabilizing selection, Van der Plank, 1968). Some resistance genes in the host are associated with fitness costs in the pathogen (Bahri et al., 2009; de Vallavieille-Pope et al., 2012). However, recently computational genomics has demonstrated that avirulence genes also serve as effectors of pathogen virulence and that there is substantial redundancy among effector genes (Jones and Dangl, 2006). There are several examples (even within the same pathogen) where virulence in the pathogen genotype has a neutral effect or even has a fitness benefit (Bahri et al., 2009; de Vallavieille-Pope et al., 2012; Leach et al., 2001). Furthermore, it has often been observed that removal of the variety with the corresponding resistance gene rarely results in lower frequencies of virulent genotypes in the pathogen population (Mundt, 2014). Moreover, several crop pathosystems occur with several to many redundant virulence genes (Parlevliet, 1981). If there is no selection against redundant virulence genes, virulence genes for new resistant varieties can already be present in the population. This not only affects the useful life of a deployment strategy, it potentially also affects which deployment strategy is most durable, therefore, in this paper therefore we study what deployment strategy is most effective in managing pathogen evolution in the absence of stabilizing selection, i.e., without costs for virulence.

The model used in this study is built on first principles, and considers the proportion of fields planted with the host, the proportion planted with different host genotypes, the proportion of propagules that is dispersed between fields during each generation of the pathogen, and the number of pathogen generations per growing season of the host. Our aim is to develop a general model that can be used for several host-pathogen systems. For transparency we considered a spatially implicit model with a minimal number of key parameters characterizing the life cycle of the pathogen and the spatial structure of the environment through its effect on movement of propagules between hosts.

With this model we study the durable life of major resistance genes under three basic use strategies for two resistance genes and one 'mixed strategy'. To begin with, we consider sequential use, in this strategy two single-gene resistant varieties are used sequentially, where the second one variety comes into use when the resistance of the first variety is overtaken. Then, we consider pyramiding, in this strategy two resistance genes are stacked into one variety. As the last basic strategy we consider simultaneous use, in this strategy two single-gene resistant varieties are used at the same time. Next to the three basic strategies, we study a 'mixed strategy' where a pyramided variety is deployed together with two single-gene resistant varieties that deploy the same genes affects the durability of the resistance genes. The latter strategy more closely resembles an actual landscape, where varieties with stacked resistance genes are often cultivated simultaneously with single resistant varieties that contain the same resistance genes. The aim is to test if using a pyramided variety together with single-gene resistant varieties could negatively affect the useful life of the variety with the stacked resistance genes. Furthermore, we consider four single-resistant varieties in 'simultaneous use' that all have one unique resistance gene and compare this to sequential use of four single resistant varieties and pyramiding of four resistance genes in one variety and study how that affects the durable life. The aim of this study is to identify the deployment strategy that maximize the useful life when there is no cost for virulence. Furthermore, we identify how characteristics of the pathogen affect the useful life.

2. Model description

We first introduce the basic model that will be used to explore the effects of resistance gene deployment strategy, i.e. fraction of host area and fraction of resistant host, on the useful life of resistance genes in a simple environment of a susceptible host grown together with one resistant variety. The pathogen population will consist of an avirulent genotype and a virulent genotype. In paragraphs 2.2 and 2.3 we expand the basic model to account for simultaneous growth of multiple resistant varieties.

2.1. Basic model

We assume a large number of fields with a fraction h of fields that are planted with a host, and a fraction r of the host fields are planted with a resistant host ("resistant fields") and the remaining fraction of host fields are planted with a susceptible host genotype ("susceptible fields"). The total pathogen population P (number of lesions (in the fields) or number of propagules (during dispersal)), is divided into a virulent subpopulation V and an avirulent subpopulation A. The virulent pathogen can infect the host both in the resistant and the susceptible fields, whereas the avirulent pathogen can only infect hosts in the susceptible fields. The population of virulent, $V(g)$, respectively the avirulent, $A(g)$, pathogen genotypes at the end of the first growing season g, are given by

$$V(1) = (1 - q + qh)^{\tau-1}\lambda^{\tau}h\theta(0)P(0) \quad (1)$$

$$A(1) = (1 - q + qh(1 - r))^{\tau-1}\lambda^{\tau}h(1 - r)(1 - \theta(0))P(0) \quad (2)$$

where, τ equals to the number of pathogen reproduction cycles during a growing season of the host, λ is the number of new lesions produced per parent per pathogen generation, we assume that there is no difference between the pathogen genotypes in the reproductive ability (e.g. λ is equal) and that, for the virulent genotype, there is no difference in the reproductive ability on the susceptible and the resistant host. Furthermore, q is the fraction of lesions formed by propagules that dispersed away from the field where they were produced. In our model, the parameter q can be used to reflect differences in field sizes. For larger fields a smaller fraction of the offspring will disperse outside the field, therefore a smaller value for q can be used. On the other hand, a larger value for q can be used when smaller fields are considered, to reflect that a larger fraction of the propagules move outside the field. Last, parameter θ is the fraction of the total pathogen population P that is virulent. The right hand side of the equations, reflects that at the start of a new growing seasons all fields receive a fraction θ virulent propagules and a fraction $(1-\theta)$ avirulent propagules of the total population P. Only the propagules that arrived in a suitable field can reproduce; the virulent genotype can reproduce in all host fields h, while the avirulent genotype can only reproduce in fields with susceptible hosts $(1-r)h$. The left hand side of the equations, reflects the dispersal process; a fraction $(1-q)$ of the propagules will remain in the field, and of the propagules that disperse between fields, a fraction qh of the virulent genotype will find a suitable field for reproduction, respectively, a fraction $(1-r)qh$ of the avirulent genotype will find a suitable field for reproduction. For an extensive model description see Electronic supplement.

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