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PUTTING PLANT DISEASE RESISTANCE GENES TO WORK

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1. INTRODUCTION

Semi-dominant plant disease resistance ($R$) genes confer recognition of and response to specific races of pathogen that carry a corresponding Avirulence ($Avr$) gene. $R$ proteins are presumed to recognise pathogen $Avr$ gene-encoded products, or compatibility factors, that are likely to be involved in pathogenicity on the host. $R$ genes against various important diseases have been used by plant breeders, but when deployed in monocultures, resistance frequently breaks down as races of the pathogen emerge that can overcome the $R$ gene through recessive mutations in the corresponding $Avr$ gene. Nevertheless, in nature, $R$ genes have been maintained. In Arabidopsis, ~164 homologs of the largest class of $R$ genes exist. These $R$ genes encode proteins of the nucleotide binding-leucine rich repeat (NB-LRR) class (Dangl and Jones, 2001).

In natural populations and environments, in which nitrogen is limiting, plants have been selected to only turn on the defence response upon attack. Constitutive expression of defence mechanisms is costly and likely to be deleterious, even after application of nitrogen fertiliser. The $R$ genes encode the plant “antennae” to ensure defence mechanisms are only expressed when needed. However, if it is so easy to evade detection by mutating an $Avr$ gene, why maintain them? The answer lies in the fact that $R$ gene loci are usually extremely polymorphic compared to other loci. $R$ gene polymorphism in a population, with each $R$ gene allele present at a low frequency, reduces selection in the pathogen population to overcome any particular $R$ gene, especially if the corresponding $Avr$ gene contributes to pathogenicity on plants that lack that $R$ gene. This “balancing polymorphism” is disrupted in conventional agriculture. It is worthwhile to consider possible approaches to deploying $R$ gene polymorphism in agriculture, using the example of potato late blight caused by the oomycete Phytophthora infestans.