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Fine Mapping of the Barley Locus *Rym11* Conferring Resistance to the Barley Yellow Mosaic Virus Complex

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1 Introduction

1.1 Disease resistance in plants

1.1.1 Broad-spectrum resistance

Broad-spectrum resistance is a defence strategy that plants harbour against a large range of natural enemies such as bacteria, fungi, nematodes and viruses. Two types of broad-spectrum resistance, active and passive are distinguished. With respect to passive resistance, toxic compounds are constitutive independently of the presence of the pathogen (Osbourn et al., 1996). The toxic alkaloids in potato are an example of a passive resistance mechanism.

In case of active broad-spectrum resistance, defence reactions are initiated only when the plant is facing a pestilent attack; the effectiveness of such reactions is, nevertheless, against various potential pathogens. An example of active defence mechanism is the induced resistance (IR) that is activated e.g. upon an inducer-pathogen attack, enabling the plant to drive an effective defence response against a second attacking pathogen, the challenger. The first induced resistance is called local acquired resistance because it is limited to the infection site. The resistance may then spread systematically through the entire plant body leading to a broad-spectrum, systemic resistance (Ryals, 1996) such as the systemic acquired resistance (SAR). A set of genes known as SAR genes or pathogenesis related (PR) genes because of their implication in the production of the pathogenesis related proteins (PR-proteins) have been identified to be associated with SAR mechanisms in dicotyledonous species. However, it has not been clearly elucidated how the SAR is involved in monocotyledonous plants.

For plant improvement, broad-spectrum resistance has the advantage of being effective against several pathogen species. Breeding to increase the level of this resistance may be of great profit. However, the level of toxicity associated with the broad resistance mechanisms may have negative side effects particularly in food and fodder crops. For instance, it may be unacceptably high to humans and cattle, or decrease dramatically the nutritional value. Another disadvantage is that broad-spectrum resistance against a wide range of generalist pathogens and pests may be associated with increased attractiveness to specialist species (Niks and Lindhout, 2000).

1.1.2 Hypersensitivity resistance

The hypersensitive response (HR) is classically defined as a locally triggered cell death in the host plant at the site of attack by a pathogen (Agrios, 1997). Even hypersensitive resistance is, in many cases, associated with other active defence mechanisms, such as the PR-protein production, one particular aspect is that the HR effectiveness is race-specific. This enables the activation of the defence only to certain genotypes of the pathogen. For this reason, this type of resistance is also known as vertical resistance. In fact, a graph with many vertical columns is obtained when the reaction of resistance of a host plant genotype is plotted against a set of pathogen genotypes (VanderPlank, 1963).

To explain the race-specificity feature of hypersensitivity resistance, Flor (1971) proposed the hypothesis of a gene-for-gene interaction: 'For each gene conditioning resistance in the host, there is a specific gene conditioning pathogenicity in the parasite'. Flor made the emphasis on pathogenicity, literally virulence, but currently the emphasis is on avirulence. The resistance is determined by an interaction between the product of the resistance gene of the host plant and the product of the avirulence gene of the pathogen. A model elicitor/receptor has been suggested to elucidate the molecular basis of the gene-for-gene interaction (Keen, 1982).

In this model the dominant allele *R* for resistance produce a receptor molecule that recognizes an elicitor molecule produced by the dominant allele Avr of the avirulence gene. This recognition event elicits the hypersensitive reaction, which consists of a signal transduction pathway leading to a cascade of physiological reactions in the plant that are responsible for cell-death in the infection site (Blumwald et al., 1998). In case the pathogen carries the virulence allele, avr, rather than the avirulence allele, Avr, there will be no, or a mutilated, avirulence gene product and therefore, no recognition event will take place.

During the last years, the isolation of several *R* genes and the characterization of their products permitted considerable advances in the knowledge of the molecular basis of specific resistance. The comparison of different gene products revealed a strong sequence homology as well as five types of conserved structural domains: LRR (Leucine-Rich Repeats), Ser/Thr kinase (serine/threonine kinase), NBS (Nucleotide Binding Sites), LZ (Leucine Zipp) and TIR (Toll Interleukin Receptor). The *R* products seem to combine a receptor domain with an effector domain ensuring two main functions: the recognition of elicitor molecules thanks to protein-protein interaction