Genes involved in resistance to powdery mildew in barley differentially modulate root colonization by the mycorrhizal fungus *Glomus mosseae*

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Mycorrhiza (1999) 9:237–240

SHORT NOTE

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Genes involved in resistance to powdery mildew in barley differentially modulate root colonization by the mycorrhizal fungus *Glomus mosseae*

Accepted: 16 September 1999

Abstract Arbuscular mycorrhizal fungi (AMF) and *Erysiphe graminis* are obligate biotrophic fungi with different outcomes in their interaction with plants, different targeted host tissues, but similar patterns of development and infection processes. These similarities raise the question of whether the two types of biotrophic fungal infections have common features in their regulation. To investigate this question, we compared a number of *Ror* and *Rar* barley mutants susceptible to *E.graminis* f. sp. *hordei*, as well as their resistant progenitors, for susceptibility to infection by the AMF *Glomus mosseae*. The two powdery mildew-resistant lines BC Ingrid and Sultan presented a similar reduction in *G. mosseae* development within roots when compared to the wildtype cultivar Ingrid, indicating a systemic effect of the altered genes in the plant. *Ror* and *Rar* mutants, in which susceptibility to powdery mildew is restored, showed increased resistance to AM fungal development in their roots when compared to their progenitors, which suggests that corresponding mutations must have affected genes which differentially modulate symbiotic and pathogenic biotrophic plant-fungus interactions.

Key words Barley · *Glomus mosseae* · Powdery mildew · Non-race-specific resistance · Race-specific resistance

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Introduction

Erysiphe graminis f. sp. *hordei* is an obligate biotrophic fungus responsible for powdery mildew disease in barley. It attacks leaf tissues (Freialdenhoven et al. 1994) and causes severe damage and sometimes death of the host (Kita et al. 1981; Koga et al. 1990; Freialdenhoven et al. 1994). The different outcomes of this pathogenic interaction, varying from susceptibility to race-specific resistance or non-race-specific resistance depending on plant and/or fungus genotype, have been studied intensively (Wiverg 1974; Sogaard and Jorgensen 1988; Görg et al. 1993; Peterhänsel et al. 1997). Mutation analysis has led to the discovery of genes required for resistance gene function in barley (Torp and Jorgensen 1986; Freialdenhoven et al. 1994, 1996). *Rar* genes (*Rar1* and *Rar2*) are required for the race-specific resistance controlled by the *Mla12* resistance gene, as well as for a number of other powdery mildew resistance interactions (Jorgensen 1996). *Ror* genes (*Ror1* and *Ror2*) are required for the non-race-specific *mlo*-controlled resistance response in barley. Mutations in these genes restore susceptibility to the pathogen with successful penetration of the host cell and completion of the fungal life cycle (Freialdenhoven et al. 1996).

Arbuscular mycorrhizal fungi (AMF), like *E. graminis,* are obligate biotrophic microorganisms. In contrast, they colonize root tissues and provide nutrients and water to their hosts, compensating their carbon cost to the plant and, hence, leading to a mutualistic symbiotic association which improves plant growth (Allen 1991; Bethlenfalvay and Linderman 1992; Smith and Read 1997). Although the host tissues targeted by AMF and powdery mildew fungi are different, the two types of fungi show similar patterns of development and infection processes. Both begin with the germination of spores and formation of a germ tube which comes into contact with the host cell surface. This induces differentiation of an appressorium from which a penetration hypha develops. Once inside the host tissues, an appressorial infection peg (*E. graminis*) or an

intraradical hypha (AMF) penetrates the plant cell and produces a branched haustorium surrounded by a host cell membrane, termed the extrahaustorial membrane or the periarbuscular membrane, respectively. The haustoria, called arbuscules in the case of AMF, are nutrient exchange organs essential to development of both types of biotrophic fungi, but which also function in the transfer of nutrients from the fungus to the host plant cell in the case of AMF (Kita et al. 1981; Koga et al. 1990; Giovannetti et al. 1994; Boyd et al. 1995; Gianinazzi-Pearson 1996).

These similarities raise the question of whether the two types of biotrophic fungal infections share common features in their regulation. Structural similarities in resistance gene products from different plant species imply the existence of common "downstream" biochemical defence mechanisms to diverse pathogens (Bowles1990). Numerous defence-related genes have been implicated in the host's defence response to *E. graminis* f. sp. *hordei* infection (Bryngelsson and Collinge 1991; Boyd et al. 1994; Büschges et al. 1997), and host plant defence responses are expressed during arbuscular mycorrhiza formation in different plants, although these are uncoordinated, weak and/or very localized (Morandi et al. 1984; Gianinazzi-Pearson et al. 1992, 1996; Harrison and Dixon 1993; Volpin et al. 1994). In the case of *E. graminis* f. sp. *hordei,* the products of the *Mla* and *mlo* alleles and the mechanisms by which they achieve resistance are not well known. This is also true for AMF interactions with resistant pea mutants (Gianinazzi-Pearson et al. 1991; Gollotte et al. 1993, 1996; Ruiz-Lozano et al. 1999).

The present study was stimulated by the striking similarities in plant cell responses to *E. graminis* f. sp. *hordei* and to AMF by recessive EMS-induced resistant barley and pea mutants, respectively. In both cases, incompatibility reactions are characterized by the elicitation of defence-related fluorescent host cell wall appositions adjacent to fungal appressoria (Gollotte et al. 1993, 1996; Freialdenhoven et al. 1994; Smith and Read 1997). The aim of the present work was to see whether genes involved in biotrophic pathogen interactions could also affect biotrophic symbionts like AMF. For this, a number of *Rar* and*Ror* barley mutants susceptible to *E.graminis* f. sp. *hordei*, as well as their resistant progenitors, were compared for their susceptibility to the AMF *Glomus mosseae*.

Materials and methods

Clay loam soil from Epoisses experimental domain (INRA, Dijon) was sieved (2 mm), sterilized by γ-irradiation (10 Gy) and mixed with autoclaved calcined clay (Terragreen®) (2:1, v:v). The soil had a pH of 8.1 (water), 2.3% organic matter and 18 ppm available P (Olsen). Pots were filled with 1 kg of the sterilized soil/terragreen mixture.

Seeds from nine different barley lines or cultivars were used in this study: [1] cultivar Ingrid carrying the *Mlo* wild-type allele, [2] BC Ingrid into which the non-race-specific resistance mediating *mlo-5* allele was transferred from the genetic background of cultivar Ingrid by backcrossing (Jorgensen 1983), [3] two susceptible *Ror1* mutants, A39 and C36, and a susceptible *Ror2* mutant, A44, obtained by mutagenesis of a backcross line of BC Ingrid (Freialdenhoven et al. 1996), [4] cultivar Sultan, a resistant double-haploid containing the *Mla-12* wild-type allele, responsible for race-specific resistance to powdery mildew, and [5] two susceptible *Rar1* mutants, M82 and M100, and a susceptible *Rar2* mutant, M22, isolated after mutagenesis of cultivar Sultan (Torp and Jorgensen 1996).

Surface-disinfected seeds (10 min 7% Ca hypochlorite) of *Hordeum vulgare* L. (9 different lines) were sown in the soil/terragreen mixture in the presence of an inoculum of *G. mosseae* (Nicol. and Gerd.) Gerd. and Trappe (BEG 12), as described previously (Dumas-Gaudot et al. 1994). Treatments were replicated four times for a total of 36 pots arranged in a randomized block design (three plants per pot). Plants were grown in a greenhouse with a 16/8-h day/night cycle and 80% RH. Day temperatures did not exceed 28 °C and night temperatures did not fall below 19 °C throughout the experiment. Water was supplied daily to maintain soil at field capacity throughout plant growth.

Six weeks after sowing, parameters of mycorrhizal colonization were determined, after trypan blue staining of roots (Phillips and Hayman 1970). Thirty 1-cm root fragments from the mixed root system contained in each pot (three plants) were examined with a compound the microscope. Four replicates per treatment were used. The colonization frequency (F%) is the ratio between colonized root fragments and the total number of root fragments examined. It gives an estimation of the length of root colonized by the fungus. The colonization intensity (M%) is an estimation of the amount of root cortex colonized. Finally, the arbuscule abundance (A%) is an estimation of the arbuscule richness in the total root system. These parameters were calculated as described by Trouvelot et al. (1986). Percentages were arcsin transformed and the data were subjected to ANOVA. Differences amongst means were analysed for significance by Duncan's multiple range test.

Results and discussion

Three levels of specificity for resistance to powdery mildew infection have been described in barley (Jorgensen 1994). First, race-specific resistance mediated by the *Mla* locus, which is effective against individual isolates of *E. graminis* f. sp. *hordei* causing barley powdery mildew, follows the rules of the gene-for-gene concept (Floor 1971). Second, a non-race-specific or broad spectrum resistance, mediated by recessive (*mlo*) alleles at the *Mlo* locus, is effective against almost all tested isolates of *E. graminis* f. sp. *hordei* (Jorgensen 1977; Lyngkjaer et al. 1995). Finally, a non-host resistance protects barley from infection attempts by several other formae speciales of *E. graminis*; little is known about this kind of resistance (Tosa et al. 1990). Development of the AMF *G. mosseae* varied among the different barley genotypes (Table 1). Wild-type barley plants (cultivar Ingrid) showed the highest levels of root colonization in terms of percentage of root fragments showing AMF structures, percent of root system cortex colonized and frequency of arbuscules present in the root system.

The powdery mildew resistant lines cultivar BC Ingrid (*mlo*-5 backcross) and cultivar Sultan (*Mla* 12) both showed a similar reduction in *G. mosseae* development inside roots in comparison to the wild-type cultivar Ingrid (*Mlo* wild-type). Although the frequency of root fragments colonized by *G. mosseae* was not af-

Table 1 Development of *Glomus mosseae* in roots of different barley (*Hordeum vulgare* L.) lines. Calculated mycorrhizal parameters are F% (colonization frequency), M% (colonization intensity) and A% (arbuscule abundance). Within each parameter, means followed by the same letter are not significantly different ($P<0.05$) as determined by Duncan's multiple-range test ($n=4$)

Line/ ultivar	Genotype	Resistance to powdery mildew[a]	F%	M%	A%
Non-race-specific resistance					
Ingrid	*Mlo* wildtype	No	65.0a	35.7a	32.4a
BC Ingrid	*mlo*-5 backcross	Yes	60.0a	10.8 cd	10.3c
A39	*Ror1* mutant	No	33.3c	10.6 cd	1.3d
C36	*Ror1* mutant	No	14.3d	2.5d	0.2d
A44	*Ror2* mutant	No	31.3c	2.4d	1.0d
Race-specific resistance					
Sultan	*Mla12* (2n)	Yes	65.0a	18.0bc	17.1bc
M82	*Rar1* mutant	No	14.3d	0.4d	0.2d
M100	*Rar1* mutant	No	64.7a	18.0bc	15.5bc
M22	*Rar2* mutant	No	47.1b	24.8b	22.3b

[a] Freialdenhoven et al. (1996)

fected in either case, the development of the fungus within the root cortex was significantly reduced in both cultivars so that M and A decreased by 70% in the case of BC Ingrid and by 50% in cultivar Sultan compared with the wild-type cultivar. Because the *mlo-5* allele in BC Ingrid and the *Mla12* allele in cultivar Sultan confer non-race- and race-specific resistance to *E. graminis* f. sp. *hordei*, respectively, it could be hypothesized that the decrease in AM fungal development results from a generalized effect of the two alleles in different parts of the plant. Resistance mediated by recessive *mlo* or dominant *Mla* alleles is characterized by the induction of multiple defence reactions, leading to host cell death, such as plant cell wall modifications and the accumulation of defence-related transcripts encoding for hydrolytic enzymes or involved in synthetic pathways of antifungal toxins and phytoalexins, (Bohlmann et al. 1988; Boyd et al. 1994; Freialdenhoven et al. 1996; Büschges et al. 1997). Because AMF can also elicit defence responses in plants (Gianinazzi-Pearson et al. 1996), enhanced expression of these may be active in the reduced mycorrhizal development of the two *E. graminis*-resistant barley cultivars. However, this explanation is more likely to apply to the non-race-specific resistant line (BC Ingrid) than to the race-specific resistant line (Sultan), which is active against specific isolates of *E. graminis* f. sp. *hordei*. A striking feature of non-race-specific resistance is spontaneous cell death after the appearance of cell wall appositions; this is different from the hypersensitive cell death typical of race-specific incompatibility (Wolter et al. 1993; Freialdenhoven et al. 1996). Cellular and molecular investigations of AM fungus-host cell interactions within these barley genotypes could give important clues as to the mechanisms in common with resistance to *E. graminis*.

The *Ror* and *Rar* mutants, which have restored susceptibility to the powdery mildew pathogen, did not show increased AMF development in their roots compared with their resistant progenitors. Strikingly, fungal spread inside the roots of the three *Ror* mutants (A39, A44 and C36) and one of the *Rar* mutants (M82) was strongly reduced and arbuscule development was inhibited, as shown by the extremely low M% and A% values (Table 1). The *Ror* and *Rar* mutants were obtained by chemical mutagenesis of BC Ingrid and Sultan, respectively. Genetic analysis demonstrated that susceptibility to *E. graminis* f. sp.*hordei* in *Ror* mutants is not due to a reversion event restoring the dominant *Mlo* wild-type allele, but instead reflects mutations in other genes required for *mlo* function (Freialdenhoven et al. 1996). The *Rar* mutants represent monogenetically inherited mutations in loci unlinked to *Mla12* (Torp and Jorgensen 1986; Freialdenhoven et al. 1994). The present observations suggest that these mutations affect genes which are also involved in arbuscular mycorrhiza regulation, but that their inactivation differentially modulates the latter and leads to greater resistance to *G. mosseae*, rather than increased susceptibility, as in the case of *E. graminis*. This may reflect pleotropic activities of the corresponding genes, as already inferred from mutants for plant genes controlling nodulation and mycorrhizal processes in *Pisum sativum* roots (Duc et al. 1989). Our hypothesis is supported by recent findings by Jarosch et al. (1999), who found that barley plants carrying an *mlo* allele for powdery mildew resistance were much more infected by the pathogen *Magnaporthe grisea* than susceptible *Mlo* plants.

In conclusion, mutation of genes involved in barley resistance/susceptibility to the pathogenic biotrophic fungus *E. graminis* f. sp. *hordei* can also differentially mediate arbuscular mycorrhiza formation by the symbiotic fungus *G. mosseae*. Elucidation of the cellular and molecular bases of these effects could shed further light on the rôle of plant genes in symbiotic fungal-root associations.

Acknowledgements This work was supported by an EU research training grant to J.M. R-L (Contract BIO4-CT96-5037). We thank Dr. A. Freialdenhoven for kindly providing the barley seeds and Dr. Paul Schulze-Lefert for critically reading an original version of the manuscript.

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