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REVIEW ARTICLE

Powdery mildew control in pea. A review

Sara Fondevilla · Diego Rubiales

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Abstract Pea powdery mildew is an air-borne disease of worldwide distribution. It is particularly damaging in late sowings or in late maturing varieties. It is caused by *Erysiphe pisi*, although other fungi such as *Erysiphe trifolii* and *Erysiphe baeumleri* have also been reported causing this disease on pea. The disease can cause 25–50% yield losses, reducing total yield biomass, number of pods per plant, number of seeds per pod, plant height and number of nodes. The disease also affects green pea quality. Current powdery mildew control methods include early planting, the use of fungicides and of resistant cultivars. Chemical control is feasible with a choice of protective and systemic fungicides. However, public attitude and environmental concerns towards the use of pesticides as well as the development of powdery mildew strains resistant to different fungicides have reduced the appeal of chemicals and have led to the search of alternative control methods. The present review summarises the current control strategies and highlights future challenges for efficient and sustainable powdery mildew management. Non-fungicide products, such as soluble silicon, oils, salts and plant extracts are under study but are not fully ready yet for commercial application. Attempts have also been made to control powdery mildews with mycolytic bacteria, mycophagous arthropods, fungi, yeasts and other possible non-fungal biological control agents, but more efforts are still needed to prove the efficacy of these methods in agricultural practice. Genetic resistance is acknowledged as the most effective, economic and environmentally friendly method of control. However, only three genes (*er1*, *er2* and *Er3*) have been described so far in *Pisum* germplasm and only *er1* has been widely used in breeding programmes, what is very risky. Expansion of cultivation areas of pea varieties harbouring the same resistance gene could promote the occurrence of new races of the pathogen that would lead to a breakdown of the resistance. The use of polygenic resistance or combining several major genes could enhance the durability of the resistance.

Keywords *Erysiphe pisi* · *Erysiphe trifolii* · *Erysiphe baeumleri* · *Pisum sativum* · Biological control · Chemical control · Disease resistance

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1 Introduction

Pea powdery mildew caused by the obligate biotrophic fungus *Erysiphe pisi* DC is an air-borne disease of

worldwide distribution, being particularly important in climates with warm dry days and cool nights (Smith et al. 1996). The disease can cause 25–50% yield losses (Munjal et al. 1963; Warkentin et al. 1996), reducing total yield biomass, number of pods per plant, number of seeds per pod, plant height and number of nodes (Gritton and Ebert 1975). The disease can also hasten crop maturity, rapidly raising tenderometer values beyond optimal green pea harvesting levels (Falloon and Viljanen-Rollinson 2001). Severe pod infection leads to seed discolouration and downgrading of seed quality. It can also damage quality of processing pea giving tainted and bitter characteristics. Conidias and fungal debris from heavily infected crops can cause breathing and allergy problems for machinery operators. Powdery mildew is particularly damaging in late sowings or in late maturing varieties. The earlier the disease occurs the more severe the damage.

Powdery mildew affects all green parts of pea plants. The first symptoms are small, diffuse spots on leaflets and stipules, usually first appearing on the lowest part of the plant. These lesions grow and became white to pale grey powdery areas (Fig. 1) that later coalesce and completely cover plant surfaces (Falloon and Viljanen-Rollinson 2001).

Current powdery mildew control methods include early planting, the use of fungicides and of resistant cultivars. The present review summarises the current control strategies and highlights future challenges for efficient and sustainable powdery mildew management.

2 The pathogen

Powdery mildew of pea is caused by *E. pisi* in the past, often reported as *Erysiphe communis* auct. p.p. or *Erysiphe polygoni* auct. p.p. Braun (1987) differentiated *E. pisi* var. *pisi* infecting species in *Pisum*, *Medicago*, *Vicia*, *Lupinus* and *Lens*, and *E. pisi* var. *cruchetiana* infecting *Lathyrus* and *Ononis* species. Recently, *Erysiphe baeumleri* and *Erysiphe trifolii* have also been reported causing disease on pea in both field and glasshouse conditions in the Czech Republic and US Pacific Northwest, respectively (Ondřej et al. 2005; Attanayake et al. 2010). These *Erysiphe* species can be differentiated from *E. pisi* by rDNA internal transcribed spacer sequences, in combination with assessment of morphological characters of chasmothecial appendages, being typically mycelioid type in *E. pisi* and dichotomously branched in *E. trifolii* and *E. baeumleri*. *E. trifolii* can be distinguished from *E. baeumleri* by its horizontally spread and coloured appendages. The existence of distinct powdery mildew species infecting pea in both glasshouse and field environments may interfere with the powdery mildew-resistance breeding programmes and possibly explains putative instances of breakdown of resistance in previously resistant pea breeding lines.

Fig. 1 Powdery mildew symptoms in a pea leaf

3 Cultural practices

The most adopted practise to escape from powdery mildew infection is to plant early in the growing season or use early maturing cultivars. Early seeded crops and early maturing cultivars are often less affected by this disease than late-harvested crops because the fungus has less time to spread and affect yield.

Infection of most powdery mildews increases with soil nitrogen availability due to its effect on host growth rate. On the contrary, phosphorous reduces the incidence of the disease (Jarvis et al. 2002). Powdery mildew is often more severe in a lush pea stand. The fact that powdery mildew is more severe in conditions that favour growth and productivity of the host implies that crop management practices to create sub-optimal host growing conditions in the hope of reducing powdery mildew and severity is not an attractive proposition for farmers.

Crop rotation is of limited usefulness in managing powdery mildew. Powdery mildew epidemics sweep large areas with ease, and the separation of crops in time and space can delay epidemics but not prevent them (Viljanen-Rollinson et al. 1998a). Weather factors are more important than tillage regime in the incidence of powdery mildew. Use of disease-free seed does not have a significant effect in managing the pathogen as the possibility of transmission of *E. pisi* through infected seeds is remote (Tiwari et al. 1999a).

4 Chemical control

Sulphur and dinocap formulations have been successfully applied in protective schedules (Sharma and Mathur 1984;

Singh and Singh 1978; Warkentin et al. 1996). However, the cost and logistics of repeated applications of protective fungicides preclude their extensive use in many countries. Application of fungicides only when disease is observed (reactive programme) is more realistic and cost-effective than routinely applications (preventative programme). Fungicide must be applied when the number of plants infected is still low and infection level on each plant is minimal (<5% infection). Success is dependent on effective monitoring and timely application. Pea growers are reluctant to follow a spray schedule requiring delivery of chemical through ground rigs at late stages of crop development since crop damage is not compensated by yield increases. This makes the use of wettable sulphur unattractive to many growers who prefer aerial applications. However, because of its low cost, sulphur remains an economic alternative to modern fungicides where aerial application is restricted by regulation, topography or proximity to housing. Sulphur is also allowable under some biodynamic and organic horticulture systems.

Generally, only one application is required, unless infection comes in very early and/or conditions conducive to infection persist. In this case, follow-up applications may be required. Preventative programmes are more appropriate when powdery mildew is known to occur regularly. The first spray should be applied at flowering and then followed with additional sprays at 14-day intervals depending on disease presence.

Extensive research throughout the agrochemical industry expanded options for powdery mildew control in the 1980s through introduction of several triazoles (sterol demethylation inhibitors) and two additional members of the morpholine group, fenpropimorph and fenpropidin. These have proven very effective in controlling pea powdery mildew (Ransom et al. 1991; Warkentin et al. 1996). Triazoles are reputed to have some translaminar systemic activity and are suited to the low-volume aircraft applications favoured by green pea growers. A single application of triadimefon at early flowering prevents mildew infection of pods, increases yield and evens out maturity, thus improving crop quality. A second application may be necessary 2 weeks after the first if the anticipated date of harvest is more than 5 weeks or the risk of disease is high. Good plant coverage with the fungicide is essential. Over time, triadimefon accumulates at the leaf margins, leaving other parts of the leaf more open to infection. Tebuconazole is active over the whole leaf for a longer period, giving more sustained control.

More control options are recently available with the broad-spectrum fungicides strobirulins and anilinopyrimidines and the powdery mildew specifics spiroxamine and quinoxyfen (Hollomon and Wheeler 2002). New mixtures are continuously being tested and approved for powdery mildew control in pea, such as the formulation mixture of the strobirulin pyraclostrobin plus the carboxamide boscalid.

5 Control with natural products

Public attitude and environmental concerns towards the use of pesticides as well as the development of resistant powdery mildew strains have reduced the appeal of chemicals fungicides and have led to the search of alternative methods to control powdery mildews. Non-fungicide products, such as soluble silicon, oils, salts and plant extracts, inducing resistance in plants infected with powdery mildews or acting as prophylactic and/or curative factors are in focus (Bélanger and Labbé 2002).

Several crude plant extracts or substances isolated from the plants in vitro have proven effectiveness for controlling pea powdery mildew in vitro, in glasshouse or under field. Powdery mildew of pea has also been controlled experimentally with spraying of azadirachtin EC, a natural product of neem (*Azadirachta indica*) (Singh and Prithiviraj 1997), with bergenin, a natural product from *Flueggea microcarpa* (Prithviraj et al. 1998), with ajoene, a constituent of garlic (*Allium sativum*; Singh et al. 1995; Prithviraj et al. 1998), with extracts from ginger (*Zingiber officinale*; Singh et al. 1991) or with exudates from *Scletorium rolfsii* (Pandey et al. 2007). Also, α-hydrastine (Goel et al. 2003) isolated from *Corydalis longipes*, berberine and (+)−bicuculline (Basha et al. 2002), isolated from *Corydalis chaerophylla* and venenatine isolated from *Alstonia venenata* (Singh et al. 2000b) reduced germination of *E. pisi* conidia.

A weekly application of a formulation developed from a methionine–riboflavin mixture was sufficient in controlling the disease as effectively as most conventional fungicides (Tzeng et al. 1996). The formulation is unique because it contains mainly food constituents and biodegradable ingredients. Other substances such as calcium silicate, potassium silicate, phosphorous acid, potassium bicarbonate or oils have proven successful in controlling some powdery mildews (Moyer and Peres 2008).

6 Biological control

Biological control of powdery mildews remains a challenge for future research and development. Results obtained so far are promising for practical biocontrol of a number of powdery mildew diseases, but more efforts are needed to prove the efficacy of these methods in agricultural practice. Attempts have been made to control powdery mildews with

mycolytic bacteria, mycophagous arthropods, fungi, yeasts and other possible non-fungal biological control agents (see reviews by Paulitz and Bélanger 2001 and by Kiss 2003). However, these studies have provided little practical control to date. The most promising biological control trials have involved a number of antagonists to powdery mildews and have resulted in the development of several biofungicide products. AQ10 Biofungicide®, containing conidia of a strain of a pycnidial fungus *Ampelomyces quisqualis* (Hofstein et al. 1996), Sporodex®, based on the conidia of a basidiomycetous yeast *Pseudozyma flocculosa* (Paulitz and Bélanger 2001), Serenade®, a formulation of *Bacillus subtilis* and Sonata® a formulation of *Bacillus pumilus* (Marrone 2002) have been tested for powdery mildew control and commercialised in some countries.

Potential of other biocontrol agents have also been studied such as the fungi *Acremonium alternatum*, *Irpex lacteus* (Koitabashi 2005), *Paecilomyces fumosoroseus* (Kavková and Curn 2005), *Verticillium lecanii*, *Sporothrix rugulosa* (Verhaar et al. 1996), *Trichoderma harzianum* and the yeasts *Stephanoascus* spp. and *Tilletiopsis* spp. (Hijwegen 1992). Also, seed bacterisation by *Pseudomonas fluorescens* and *Pseudomonas aeruginosa* alone and in combination with aerial spray of their cell suspensions can control powdery mildew (Singh et al. 2000a).

Other fungi such as *Acrodontium crateriforme*, *Dissoconium aciculare* and *Ramichloridium apiculatum* have been reported as natural antagonists of *E. pisi* (Hijwegen and Buchenauer 1984), but their potential as practical biocontrol agents has not been further investigated.

7 Induced resistance

It is well established that many factors may act on plants to induce high levels of systemic resistance to subsequent pathogen attack. These include prior pathogen attack and various chemical and environmental stimuli. Induction of systemic resistance is associated with gene induction (Ward et al. 1991), the activation of a wide range of disease resistance mechanisms and the production of a wide range of defence compounds; it is race non-specific and is often effective against a broad spectrum of pathogenic agents (Kuc 1995).

Some chemicals like benzothiadiazole, β-aminobutyric acid, chitosan, salicylic acid or even some plant extracts have been reported to induce resistance in a number of pea–pathogen interactions that can be very effective (Frey and Carver 1998; Dann and Deverall 2000; Bélanger and Labbé 2002; Barilli et al. 2009, 2010). They may provide commercially useful broad-spectrum plant protection that is stable, long-lasting and environmentally benign. Exogenous application of salicylic acid solutions to pea leaves induced systemic resistance to *E. pisi* reducing by 20–30% the percentages of fungal germlings that successfully infected untreated leaves (Frey and Carver 1998).

Resistance to *E. pisi* has also been induced by prior inoculation with spores of pea non-pathogenic powdery mildews *Oidium* sp., *Phyllactinia corylea* and *P. delbergiae* (Singh et al. 2003a). *T. harzianum* fungus has shown potential as biocontrol of several powdery mildews including pea powdery mildew, but its main mechanism of action does not seem to be mycoparasitism or antibiosis but induction of resistance in the infected plants (Elad 2000).

Reduction of pea powdery mildew by Neemazal, the above-listed natural product made from neem, has been ascribed to induction of resistance (Singh and Prithiviraj 1997). Also, reduction of pea powdery mildew by aqueous extracts of vermicompost has been ascribed to induction of resistance (Singh et al. 2003b). Soil amendment with vermicompost (1–5%) induced synthesis of phenolic acids in pea, what was correlated with the degree of resistance in treated as compared with non-treated (control) pea plants.

8 Genetic resistance

Only two recessive (*er1* and *er2*) and one dominant (*Er3*) genes for powdery mildew resistance have been described so far in *Pisum* germplasm (Harland 1948; Heringa et al. 1969; Fondevilla et al. 2007a). Gene *er1* provides from complete to moderate levels of resistance (Cousing 1965; Heringa et al. 1969; Tiwari et al. 1997a,b; Fondevilla et al. 2006) and is in widely used in pea breeding programmes. A recent study indicates that resistance provided by *er1* is due to a loss of function of PsMLO1, a MLO (Mildew Resistance Locus O) gene (Humphry et al. 2011). Gene *er2* (Heringa et al. 1969) confers complete resistance that was effective in some locations but ineffective in others (Tiwari et al. 1997a, b; Fondevilla et al. 2006). This suggests the existence of races of *E. pisi*, although, to date, races of *E. pisi* races have not been described unambiguously. Tiwari et al. (1997a) found that the reaction of a set of pea genotypes to powdery mildew in divergent locations in North and South America and Asia was similar. An alternative explanation for the differences of powdery mildew susceptibility conferred by gene *er2* at different locations may be the effect of different environment or other factors on the expression of the resistance gene. This hypothesis is supported by a study showing that the level of resistance of line JI2480 (carrying the gene *er2*) is strongly influenced by temperature and leaf age (Fondevilla et al. 2006). An alternative explanation might be that different powdery mildew species are predominant in the different locations.

Gene *Er3* was recently identified in *Pisum fulvum* and has been successfully introduced into adapted *Pisum sativum* material by sexual crossing (Fondevilla et al. 2007a, 2010).

An isoline of cv. Messire containing this gene is available and could be used to introduce this gene into any other interesting cultivar. *Er3* gene confers complete resistant against the Spanish, English and Canadian *E. pisi* isolates which have been tested (Fondevilla et al. 2007a, b; Valarmathi G personal communication) and is currently being tested against local Indian isolates.

Molecular markers linked to these three genes in coupling and repulsion phase are available. Thus, simple sequence repeat and SCAR (sequence-characterised amplified region) markers linked to *er1* (Timmerman et al. 1994; Tiwari et al. 1998; Janila and Sharma 2004; Ek et al. 2005; Pereira et al. 2010), amplified fragment length polymorphism, SCAR markers linked to *er2* (Tiwari et al. 1999b; Katoch et al. 2010) and SCAR markers linked to *Er3* have been reported (Fondevilla et al. 2008). *er1* gene is located in pea LG VI (Timmerman et al. 1994) while *er2* gene is in LG III (Katoch et al. 2010).

In addition to pea lines containing genes *er1*, *er2*, and *Er3* that display complete or high level of resistance, many accessions showing uncharacterized moderate levels of incomplete resistance to *E. pisi* have been identified in *P. sativum* and wild relatives (Pal et al. 1980; Sharma 1992; Dang et al. 1994; Thakur et al. 1996; Fondevilla et al. 2007a).

9 Mechanisms of resistance

In susceptible pea genotypes, *E. pisi* conidia germinate producing a germ tube with a lobed primary appressorium. A penetration peg emerges from this appressorium and penetrates the epidermal host cells through the cuticle and cell wall. Furthermore, a primary haustorium forms within the epidermal cell (Fig. 2). Nutrient uptake from the plant cell through the haustorium supports development of secondary hyphae that radiate across the host epidermis forming hyphal appressoria from which secondary haustoria are formed (Falloon et al. 1989; Smith et al. 1996; Fig. 3). Finally, aerial conidiophores emerge from surface hyphae producing conidia capable of initiating a new cycle of infection (Falloon et al. 1989).

In pea lines harbouring *er1* gene, the vast majority of *E. pisi* conidia germinate and form appressoria. However, the pathogen is stopped soon after, and no secondary hyphae are formed (Fondevilla et al. 2006).

Resistance conferred by *er2* gene is influenced by temperature and leaf age so that complete resistance is only expressed at 25°C or in mature leaves. Resistance governed by *er2* gene at high temperatures is due to the occurrence of hypersensitive response in established colonies (Fig. 4). In adult leaves, resistance is due to a reduction of colony establishment in addition to the presence hypersensitive response in established colonies (Fondevilla et al. 2006). A proteomic study comparing the proteome of control and infected leaves of JI2480 (carrying *er2*) and the susceptible pea cv. Messire showed that JI2480 possessed constitutively a higher amount of proteins involved in defence than Messire, what could contribute to its resistance to *E. pisi* (Curto et al. 2006). These proteins included three proteins encoded by NBS-LRR resistance genes, PR proteins as PR1 and PR5, Kunitz–trypsin inhibitor that inhibit extracellular fungal proteinases, proteins associated with cell wall reinforcement, proteins involved in tolerance to oxidative stress caused by reactive oxygen species and proteins implicated in the synthesis of alkaloids compounds.

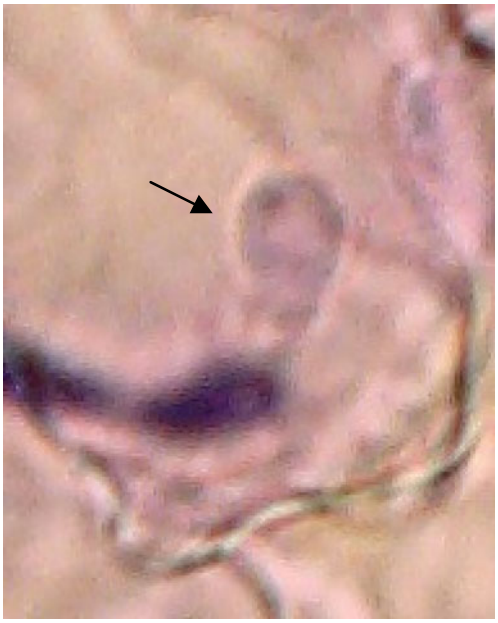

Fig. 2 *E. pisi* haustorium formed in a pea epidermal cell

In lines containing *Er3* gene, most of the *E. pisi* conidia are able to penetrate the epidermal pea cells and form

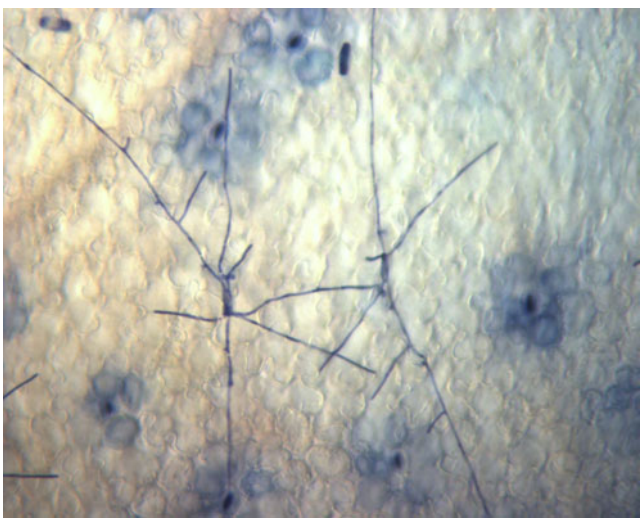

Fig. 3 *E. pisi* colony growing in a pea leaf

Fig. 4 Hypersensitive response observed in a JI2480 leaf inoculated with *E. pisi* and incubated at 25ºC for 7 days

secondary hyphae, but the growth of these established colonies is stopped by a strong hypersensitive response (Fondevilla et al. 2007a, b; Fig. 5).

In the case of other uncharacterized sources of incomplete resistance, different mechanisms can contribute to the reduction of disease severity. Thus, in lines showing incomplete resistance to *E. pisi*, mechanisms acting in almost all steps of *E. pisi* infection cycle have been identified. A certain reduction in germination have been observed in lines P6 (*P. sativum* ssp. *abyssinicum*), P17 (*P. sativum* ssp. *elatius*) and P637 (*P. sativum* ssp. *sativum* var. *arvense*; Fondevilla et al. 2007a). A small reduction in the percentage of germinated conidia that are able to form an appressorium have also been identified in lines P17 (*P. sativum* ssp. *elatius*), P635 and P637 (*P. sativum* ssp. *sativum* var. *arvense*; Fondevilla et al. 2007a) and cv. A $_{474\text{-}288}$ (Singh and Singh 1983). However, most differences between resistant and susceptible lines have been observed after appressorium formation. A lower succeed in colony establishment or a reduction in colony size have been reported in cv. A $_{474\text{-}288}$ (Singh and Singh 1983), cv. Quantum (Viljanen-Rollinson et al. 1998b) and accessions P6 (*P. sativum* ssp. a*byssinicum*), P629, P635, P636 and P637 (*P. sativum* ssp. *sativum* var. *arvense*; Fondevilla et al. 2007a). The smaller size of colonies in lines P629 and P637 and P642 was associated with a moderate percentage of hypersensitive response in established colonies showing that the death of host cells infected by *E. pisi* can contribute to incomplete resistance (Fondevilla et al. 2007a). A detailed histological study showed that hypersensitive response can result in complete resistance to this pathogen when it occurs fast and in a high proportion of colonies, as in lines harbouring *Er3* gene, or in incomplete resistance when it developed slower and take place in a lower proportion of established colonies, as in line P642 (Fondevilla et al. 2007a).

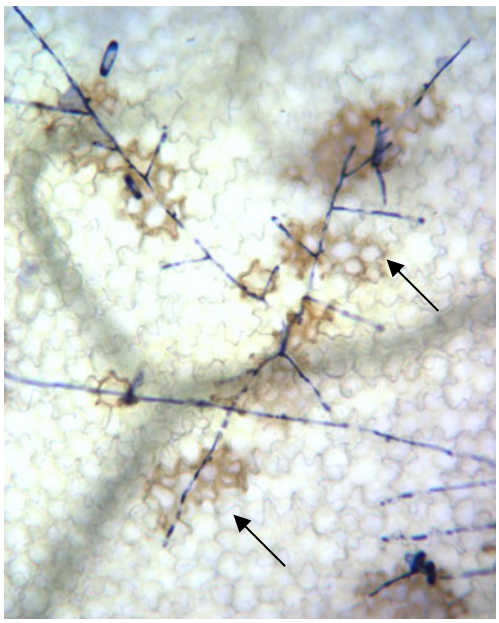

Fig. 5 Hypersensitive response observed in line P651 (containing *Er3* gene) 8 days after inoculation. Epidermal cells harbouring an *E. pisi* haustorium are dead

Differences in sporulation have also been reported. Thus, differences in the number of conidiophores bearing conidia per colony were also detected in a set of pea lines with variable reactions to powdery mildew (Banyal and Tyagi 1997). Similarly, the incomplete resistance shown by the pea cultivar Quantum was found to be based on a lower infection efficiency and conidial production accompanied by a longer time to reach maximum conidial production per day (Viljanen-Rollinson et al. 1998b).

10 Future challenges in resistance breeding

Genetic resistance is an efficient, economic and environmentally friendly way of control, but the expansion of cultivation areas of pea varieties harbouring the same resistance gene could promote the occurrence of new races of the pathogen that would lead to a breakdown of the resistance. Therefore, although resistance conferred by the gene *er1* appears to be stable up to now against *E. pisi*, it is sensible to use additional genes for resistance to this pathogen in pea breeding programmes. In addition, *er1* might not be effective against *E. baeumleri* and *E. trifolii*, so identification of additional sources of resistance against these species is needed.

Different approaches can be attempted to obtain a durable resistance to powdery mildew. On one hand, incomplete polygenic resistance is expected to be more durable than single gene resistance, as it cannot be easily broken by a single mutation of the pathogen. As reported above, several sources of incomplete resistance displaying different mechanisms of resistance acting at almost all the steps of *E. pisi* infection cycle have been described. The combination of several of these mechanisms into a same cultivar would increase the level of resistance offered by each mechanism alone and would result in a durable resistance. However, polygenic resistance is difficult to use in breeding programmes, as the minor genes controlling resistance are difficult to identify.

Another approach to obtain a durable resistance is to combine several major genes into a variety. Knowledge on the mechanism of action of each gene will help in designing the gene combination most likely to result in a durable outcome. Combining resistances that act first to limit colony establishment (lines having *er1* gene) and, if this fails, to cause death of established colonies by hypersensitive response (lines having *er2* or *Er3* gene) would provide a double barrier to disease development that should enhance the durability of resistance offered by either gene alone. This strategy would provide a complete resistance and could be aided by the use of the available molecular markers linked to these genes.

Although some histological and proteomic studies have been carried out in the pathosystem *E. pisi–Pisum*, still little is known about the mechanisms of resistance acting against *E. pisi* at the cellular, molecular and biochemical level. Thus, still little is known about the mechanisms that result in incomplete resistance to *E. pisi*. A better knowledge of such mechanisms at the histological and molecular levels would facilitate the identification of the genes controlling these mechanisms and would be useful for combining several mechanisms into a same cultivar.

Medicago truncatula (barrel medic) is an annual, self-fertile, diploid legume species that has become a model for studying various aspects of legume genomics and biology (Ané et al. 2008; Young and Udvardi 2009; Rispail et al. 2010). The fact that *M. truncatula* is susceptible to powdery mildew (*E. pisi*; Prats et al. 2007; Ameline-Torregrosa et al. 2008) opens the way for its use to unravel pea–powdery mildew interaction. A range of resistance mechanisms against *E. pisi* are operative in *M. truncatula* accessions (Prats et al. 2007).

The transcriptomic and proteomic approaches developed for this *M. truncatula* can be used to understand the molecular components and identify candidate genes involved in defence against this pathogen. Microarray analyses have been performed to determine genes involved in defence mechanisms against *E. pisi* (Foster-Hartnett et al. 2007). In addition, Affymetrix chips with bioinformatically optimised oligonucleotides are now also commercially available for *M. truncatula* (http://www.affymetrix.com) and a novel generation of *M. truncatula* gene chip with probe sets for 1,850 *M. sativa* transcripts to facilitate transcriptomic analysis of closely related species will be soon available (Ané et al. 2008). In parallel, expression of more than 1,000 transcription factors (TFs) have been monitored by quantitative real-time polymerase chain reaction during resistance reaction to powdery mildew in *M. truncatula* (Curto et al. 2007), in order to refine hypothesis about possible TFs function in defence and in responses to powdery mildew. The range of application of proteomic approaches has been broadened to include *E. pisi* (Curto et al. 2008). All these genomic platforms will allow large improvements in our understanding of pea–powdery mildew interaction.

In addition to helping to identify new genes involved in plant biology, the chemical and insertional mutant collections of *M. truncatula* as well as the TILLING and RNAi methods can serve to identify the exact function of these genes, which is a pre-requisite step before gene transfer into other legume crops such as pea.

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