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Major gene and polygenic resistance to *Leptosphaeria maculans* in oilseed rape (*Brassica napus*)

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Abstract

The most common and effective way to control phoma stem canker (blackleg) caused by *Leptosphaeria maculans* in oilseed rape (*Brassica napus*) is through the breeding of resistant cultivars. Race specific major genes that mediate resistance from the seedling stage have been identified in *B. napus* or have been introgressed from related species. Many race specific major genes have been described and some of them are probably identical in *B. napus* (allotetraploid AACC) and the parental species *B. rapa* (diploid AA). More work is needed using a set of well-characterised isolates to determine the number of different major resistance genes available. In some *B. napus* cultivars, there is resistance which is polygenic (mediated by Quantitative Trait Loci) and postulated to be race non-specific. Many of these major genes and Quantitative Trait Loci for resistance to *L. maculans* have been located on *B. napus* genetic maps. Genes involved in race specific and polygenic resistance are generally distinct.

Abbreviations: LG – Linkage Group; QTL – Quantitative Trait Loci; RGA – Resistance Gene Analogues

Introduction

This review focuses on resistance of oilseed rape (*Brassica napus* L.) to *Leptosphaeria maculans* Desm. (Ces & de Not) since this pathogen causes more damage to oilseed rape than that caused by other members of the *Leptosphaeria* species complex, such as *L. biglobosa* (Shoemaker and Brun, 2001), that are found on Brassicaceae species. Furthermore, only the genetics of the interaction between *L. maculans* and *Brassica* spp. has so far been studied.

Different sources of resistance to *L. maculans* have been identified and introduced into *B. napus* breeding lines and cultivars. Many studies on the inheritance of resistance have been done at both seedling and adult plant growth stages. Two types of resistance are usually distinguished. The first type is a qualitative resistance, which is expressed from the seedling to the adult plant stage in cotyledons and leaves and is generally considered as single-gene race specific resistance. The second type is a quantitative adult-plant resistance, which is a partial resistance usually thought to be race non-specific

and mediated by many genes. In Europe, Canada and Australia, many resistant cultivars have been registered but there is evidence of breakdown of race specific resistance in response to rapid evolution of *L. maculans* populations. Therefore, understanding the genetic basis of resistance in oilseed rape is strategically important for management of resistant cultivars. This paper reviews knowledge on race specific and race non-specific resistance and the relationship between them.

Race specific resistance genes in different Brassica *species*

Differential interactions in the *Brassica – L. maculans* pathosystem were first studied at the seedling stage using a cotyledon inoculation test (Williams and Delwiche, 1979). The first *B. napus* differential set consisted of three cultivars, 'Westar' (susceptible control, spring oilseed type), 'Quinta' and 'Glacier' (winter oilseed types) (Mengistu et al., 1991). Using this differential set, *L. maculans* isolates were classified into three Pathogenicity Groups (PG), i.e. PG2 (avirulent on 'Quinta' and 'Glacier'), PG3 (avirulent on 'Quinta' but virulent on 'Glacier') and PG4 (virulent on all three cultivars). Badawy et al. (1991) replaced 'Westar' with winter *B. napus* cultivar 'Lirabon' and added 'Jet Neuf', leading to the description of six PG, termed A1-A6, resulting from a subdivision of each of the previous groups into two PG (virulent or avirulent on 'Jet Neuf', respectively). Other race specific interactions were described using other differential sets including other *Brassica* species (Cargeeg and Thurling 1980; Ballinger et al., 1991; Kutcher et al., 1993; Kuswinanti et al., 1999). Genetic studies demonstrated a number of gene-for-gene interactions between *B. napus* and *L. maculans* and both avirulence genes (*AvrLm*) in the pathogen and their corresponding resistance genes (*Rlm*) in the host have been identified. Race specific resistance to isolates of *L. maculans* with the corresponding avirulence allele results in an incompatible interaction that inhibits infection from germinated ascospores or conidia and subsequent development of leaf lesions.

Genes identified in B. napus

The first race specific resistance genes were identified in 'Quinta' and 'Glacier' cultivars in the original differential set (Rimmer and van den Berg, 1992). Gene-for-gene *B. napus*/*L. maculans* interactions (*Rlm1*/*AvrLm1* in 'Quinta'-PG3; *Rlm2*/*AvrLm2* in 'Glacier'-PG2 interactions) were demonstrated through the use of segregating populations of both plant and pathogen (Ansan-Melayah et al., 1995, 1998). Other dominant race specific resistance genes have been described through genetic studies involving different oilseed rape cultivars/lines and different *L. maculans* isolates (Table 1). Some of these genes have been positioned on *B. napus* linkage maps (Ferreira et al., 1995; Mayerhofer et al., 1997; Delourme et al., 2004; Rimmer, 2006).

Mapping studies showed that some of the resistance genes are organised in clusters. Zhu and Rimmer (2003) found two closely linked but distinct loci mediating resistance at the seedling and adult plant stage, respectively in two *B. napus* breeding lines ('RB87-62' and 'DH88-752'). These genes all mapped to Linkage Group 6 (LG6) of the genetic map published by Ferreira et al. (1994). The two resistance loci in line 'RB87-62' mapped more than 40 cM away from those in line 'DH88-752', but only 5-10 cM separated the seedling and adult plant resistance loci of each line (Rimmer, 2006). Three other resistance genes (*LEM1*, *LmR1* and *cRLMm*, present in 'Major', 'Shiralee' and 'Maluka', respectively) have also been mapped onto this linkage group (Ferreira et al., 1995; Mayerhofer et al., 1997; Rimmer, 2006). From comparing their locations on the LG6 linkage group, it seems that *LmR1* is different from *LEM1* but *LmR1* could be identical to *cRLMm* since 'Shiralee' and 'Maluka' share a similar pedigree and produce similar interactions with *L. maculans* (Mayerhofer et al., 1997). Based on differential interactions with a series of *L. maculans* isolates, it seems likely that the seedling resistance genes in 'Maluka' (*cRLMm*) and 'RB87-62' (*cRLMrb*) are equivalent (Rimmer, 2006).

Delourme et al. (2004) have mapped five race specific resistance genes (*Rlm1, Rlm3, Rlm4, Rlm7* and *Rlm9*) on LG10 and one gene (*Rlm2*) on LG16 of the genetic map published by Lombard and Delourme (2001). *Rlm1* is clearly distinct from *Rlm3* and *Rlm4* because they both occur in one cultivar and they map to different positions. *Rlm3* and *Rlm4* are found in many cultivars but rarely seem to be present together in a single cultivar. Similarly, *Rlm3* and *Rlm7* have not been

Table 1. Genetic interactions between *Brassica napus* (allotetraploid AACC) and *Leptosphaeria maculans* including race specific resistance genes identified in *B. napus*

Cultivar/line	*L. maculans* isolate		Resistance gene	Location in *B. napus* LG[c]	Reference
	Name & information[a]	Genotype[b]			
Quinta	11.26.11 (PG3; A2)	***AvrLm1*** *avrLm2 avrLm3 avrLm4 AvrLm7 avrLm9*	*Rlm1*	LG10 = N7	Ansan-Melayah et al. (1998) Delourme et al. (2004)
Maxol	v11.1.2 (PG3; A2)	***AvrLm1*** *avrLm2 avrLm3 avrLm4 AvrLm7 avrLm9*	*Rlm1*	LG10 = N7	Balesdent et al. (2002) Delourme et al. (2004)
Glacier	14.3.01 (PG2; A4)	*AvrLm1* ***AvrLm2*** *avrLm3 avrLm4 AvrLm7 avrLm9*	*Rlm2*	LG16 = N10	Ansan-Melayah et al. (1998) Delourme et al. (2004)
Major	PHW1245 (PG2; A3)	*AvrLm1 AvrLm2 avrLm3* ***AvrLm4*** *AvrLm7 avrLm9*	*LEM1 = Rlm4*	LG6 = N7	Ferreira et al. (1995)
Shiralee	Canadian isolates	–	*LmR1 = Rlm4 ?*	LG6 = N7	Mayerhofer et al. (1997)
Crésor	Field population (Saskatchewan)	–	$LmFr_1$	–	Dion et al. (1995)
Crésor	Field population (Saskatchewan)	–	*aRLMc*	LG6 = N7	Rimmer (2006)
Maluka	Pl86.12 (PG2; –)	–	*CRLMm = Rlm4?*	LG6 = N7	Rimmer (2006)
RB87–62	Pl86.12 (PG2; –)	–	*CRLMrb = Rlm4?* *aRLMrb*	LG6 = N7	Rimmer (2006) Zhu and Rimmer (2003)
DH88–752	Pl86.12 (PG2; –)	–	*cRLMj* *aRLMj*	LG6 = N7	Rimmer (2006) Zhu and Rimmer (2003)
Quinta	v23.2.1 (PG4; A5)	*avrLm1 avrLm2 avrLm3* ***AvrLm4*** *AvrLm7 avrLm9*	*Rlm4*	LG10 = N7	Balesdent et al. (2001) Delourme et al. (2004)
Maxol	19.2.01 (PG4; A1)	*avrLm1 avrLm2* ***AvrLm3*** *avrLm4 avrLm7 avrLm9*	*Rlm3*	LG10 = N7	Balesdent et al. (2002) Delourme et al. (2004)
23.1.1	A290 (PG4; A1)	*avrLm1 avrLm2 avrLm3 avrLm4* ***AvrLm7*** *avrLm9*	*Rlm7*	LG10 = N7	Balesdent et al. (2002) Delourme et al. (2004)
Darmor	IBCN56	*AvrLm1? AvrLm2? AvrLm3? avrLm4 avrLm7* ***AvrLm9***	*Rlm9*	LG10 = N7	Balesdent et al. (2002) Delourme et al. (2004)

[a]Pathogenicity groups are indicated as PG2-PG4 (Mengistu et al., 1991) and as A1-A6 (Badawy et al., 1991).
[b]*AvrLm* genes matching the *Rlm* genes studied are indicated in bold typeface.
[c]Linkage groups LG10 and LG16 are from the Lombard and Delourme (2001) genetic map; LG6 is from the Ferreira et al. (1994) genetic map; LG N7 and N10 are from the Parkin et al. (1995) genetic map.

found in the same cultivar. Thus, *Rlm3, Rlm4, Rlm7* and *Rlm9* could be a cluster of tightly linked genes, or a single gene with different alleles, or a combination of both. Both LG6 of the genetic map published by Ferreira et al. (1994) and LG10 published by Lombard and Delourme (2001) seem to correspond to LG N7 of the genetic map described by Parkin et al. (1995). Thus, some of the genes described on these LG might be the same. The genes *LEM1* and *cRLMm* are almost certainly identical to *Rlm4*, present in 'Major' and 'Maluka' (Rouxel et al., 2003). Additionally, *LmR1* in 'Shiralee' and *cRLMrb* in 'RB87-62' might also correspond to *Rlm4*. The different locations of *LmR1* and *LEM1* (Mayerhofer et al., 1997) might be due to the homeologous reciprocal translocation that can occur between LG N16 and LG N7 close to the position of *LEM1* (Osborn et al., 2003). Such homeologous reciprocal translocation can affect recombination and precise mapping in this region using parents with or without the translocation.

Definite conclusions on identity of or distinctness between these *Rlm* genes will be possible only through a precise characterisation of *B. napus*/*L. maculans* interactions using differential *L. maculans* isolates selected or genetically bred to carry single (or as few as possible) identified avirulence (*Avr*) genes (Balesdent et al., 2002), through allelism tests or, in the longer term, by cloning and sequence comparison of the resistance genes. An improved host differential set comprising fixed cultivars or lines possessing a minimum number of *Rlm* genes has been developed (Balesdent et al., 2005). It consists of 'Westar' (no R genes, susceptible control), 'Columbus' (*Rlm1*-*Rlm3*), 'Bristol' (*Rlm2*-*Rlm9*), '22-1-1' (*Rlm3*), 'Jet Neuf' (*Rlm4*), '150-2-1' (*B. juncea* line, *Rlm5*, not yet characterised at the *Rlm9* locus), 'Darmor-MX' (*Rlm6*, not yet characterised at the *Rlm9* locus), '23-1-1' (*Rlm7*), '156-2-1' (*B. rapa* line, *Rlm8*, not yet characterised at the *Rlm9* locus) and 'Goeland' (*Rlm9*). The host genotypes carrying genes originating from *B. napus* are freely available to the scientific community, so that a common nomenclature can be used to simplify the identification of genes for resistance to *L. maculans* in different genotypes.

Genes identified in other Brassica *species*

Resistance to *L. maculans* in germplasm of other Brassicaceae species related to *B. napus* has also been studied. Few resistance genes were found by screening different accessions of the two diploid progenitors of oilseed rape, *B. oleracea* (CC, $2n = 18$) and *B. rapa* (AA, $2n = 20$). An extensive screening of *B. oleracea* germplasm in the main European Gene Banks was done at 'Instituto Superior de Agronomia' (ISA Lisbon). The differential isolates were BBA62908, harbouring *AvrLm1*, *AvrLm2* and *AvrLm4* alleles (Rouxel et al., 2003), and three 'PG4' European isolates harbouring none of these avirulence alleles. Of the 392 accessions tested, a few occasionally reacted to one of the 'PG4' isolates, but none was resistant to the isolate BBA62908, suggesting the absence of *Rlm1*, *Rlm2* or *Rlm4* in *B. oleracea* genotypes (JS Dias, unpubl.). These data, which are consistent with the data of Mithen et al. (1987) and Rimmer and van den Berg (1992), confirm that no major resistance genes to *L. maculans* originate from *B. oleracea*. However, in one closely related species, *B. insularis* ($2n = 18$), two dominant resistance genes were detected in a segregating population obtained from a *B. oleracea* × *B. insularis* hybrid (Mithen and Lewis, 1988).

This screening of genetic resources also encompassed 555 accessions of *B. rapa*, including accessions of vars *chinensis*, *japonica*, *parachinensis*, *pekinensis*, *perviridis*, *rapifera* and *trilocularis* and a few wild accessions. Most (95.5%) of these accessions were fully susceptible to all four *L. maculans* isolates. However, 12 (2%) accessions were resistant to all four isolates and 10 (1.8%) accessions were resistant to isolate BBA62908 and susceptible to the three 'PG4' isolates (JS Dias, unpubl.). These data suggest that the resistant accessions of *B. rapa* could harbour genes previously identified in *B. napus* such as *Rlm1*, *Rlm2* or *Rlm4*. To test this hypothesis, limited screening was done through collaboration between IPK Gatersleben and INRA-PMDV (T Rouxel and E Willner, unpubl.). Sixty-two *B. rapa* var. *oleifera* accessions, a few *B. rapa* var. *sylvestris* accessions and wild accessions were inoculated with differential isolates BBA62908 [race Av1-2-4-5-6-7-(8)], v11.1.1 [Av5-6-7-8], v11.1.2 [Av1-5-6-7-8] and v23.2.1 [Av4-5-6-7-8]. Twenty-two percent of the accessions were susceptible to all isolates and 48.3% of the accessions showed either a heterogeneous or a homogeneous resistance to all four isolates (T Rouxel and MH Balesdent, unpubl.). Of these, four accessions have *Rlm1*, three accessions have *Rlm4* and two accessions have both genes. The resistant accessions were investigated using a wide range of differential isolates. In at least one accession (CR1478), self pollination of one fully resistant plant generated a line expressing the *Rlm7* resistance. Screening of progeny of another resistant accession (156.1.1) showed monogenic control by *Rlm8* interacting with the novel single-gene avirulence *AvrLm8* (Balesdent et al., 2002). In a few accessions, resistance was observed against all or most isolates tested, suggesting occurrence of undescribed major resistance genes.

Dominant resistance genes were also identified in two *B. rapa* cultivars (Crouch et al., 1994; Chèvre et al., 2003) and a cluster of race specific genes, effective at the cotyledon stage, was identified in one source (Chèvre et al., 2003). These *B. rapa* genes were introduced into the *B. napus* genome either through production of a synthetic

oilseed rape crossed to *B. napus* (Crouch et al., 1994) or by direct crosses between *B. napus* and *B. rapa* (Chèvre et al., 2003) (Table 2). The efficiency of the different introgression methods is under study (AM Chèvre, unpubl.). Genetic studies with lines obtained from the synthetic *B. napus* indicated the presence of three genes introgressed from *B. rapa* var. *sylvestris* on different *B. napus* linkage groups; *LepR1* and *LepR2* were mapped, respectively, onto *B. napus* A-genome LG N2 and LG N10 of the Parkin et al. (1995) genetic map (Yu et al., 2005), and *LepR3* was identified from new commercial cultivars Surpass 400 (Li and Cowling, 2003) and Hyola 60. *LepR3* was mapped onto *B. napus* LG N10 about 15 cM below *LepR2* (Yu et al., 2004). The LG N10 is the LG where *Rlm2* mapped (Delourme et al., 2004). In seedling assays, *LepR1* behaved as a dominant allele and was resistant to all except one *L. maculans* isolates. The *LepR3* gene was described as a dominant gene (Li and Cowling, 2003), whereas *LepR2* was incompletely dominant to most isolates, with the phenotype of the heterozygotes more similar to that of the susceptible parent than to that of the homozygous resistant lines. Isolates virulent on *LepR2* have been identified (F Yu, SR Rimmer and DJ Lydiate, unpubl.). Resistance conferred by *LepR3* has been overcome in some parts of Australia (Li et al., 2003; Sprague et al., 2006). Thus these three genes are race specific. A recessive gene has also been identified in *B. napus* lines derived from *B. rapa* var. *sylvestris*. Mapping of this locus is in progress (S R Rimmer, unpubl.). The cluster of race specific dominant *B. rapa* resistance genes (Chèvre et al., 2003) has been transferred into *B. napus* genetic backgrounds with or without polygenic resistance and is being tested under field conditions. This cluster was introgressed into a different *B. napus* linkage group (AM Chèvre, unpubl.).

The *Brassica* species with the B genome, *B. nigra* (BB, $2n = 16$), *B. juncea* (AABB, $2n = 36$) and *B. carinata* (BBCC, $2n = 34$) have been described as highly resistant to *L. maculans* under field conditions (Rimmer and van den Berg, 1992). Based on cotyledon and stem resistance ratings, Keri et al. (1997) suggested that resistance in *B. juncea* is mediated by two genes. This is consistent with genetic data obtained with *L. maculans*, which showed that the interaction was governed by two avirulence genes termed *AvrLm5* and *AvrLm6* (Balesdent et al., 2002). The corresponding resistance genes were fixed, respectively, in a *B. juncea* line originating from 'Aurea' (*Rlm5*) and in the series of introgressed *B. napus* MX lines developed at INRA Rennes (*Rlm6*) (Chèvre et al., 1997; Balesdent et al., 2002, 2005). The resistance genes were introgressed into *B. napus* either by hand pollination between the donor species and *B. napus* cultivars/lines or by symmetric or asymmetric protoplast fusion (Table 2) and the resulting hybrids were backcrossed to *B. napus*. Whatever the screening methods used, all genes detected were dominant, except for one recessive gene introgressed from *B. juncea* (Saal et al., 2004) and three genes acting in a complex interaction (Pang and Halloran, 1996a). Evaluation of different *B. napus*-*B. nigra* addition lines carrying resistance has suggested that a number of different resistance genes occur in the B genome (Zhu et al., 1993; Chèvre et al., 1996). Resistance genes, introgressed from *B. nigra, B. juncea* or *B. carinata* into the *B. napus* genome are all on the same B genome region (Dixelius, 1999). Furthermore, Plieske et al. (1998) found that resistance genes from these three species all introgressed into the same *B. napus* linkage group. In all the introgression lines obtained by sexual crosses, resistance genes from the B genome were introgressed into A genome linkage groups of *B. napus* (Roy, 1978; Barret et al., 1998; Plieske et al., 1998). However, from their location on *B. napus* genetic maps, it seems that different genes were introgressed. This result was confirmed by the different interactions from different introgressed lines (Saal et al., 2004; AM Chèvre, unpubl.).

Other sources of resistance are available in less closely related species such as *Arabidopsis thaliana, Sinapis arvensis, Coincya monensis, Diplotaxis muralis, Diplotaxis tenuifolia* or *Raphanus raphanistrum* (Chen and Seguin-Swartz, 1999; Winter et al., 1999; Snowdon et al., 2000; Bohman et al., 2002). Some gene introgressions have been attempted by crosses to *B. napus* or by asymmetric protoplast fusion for *Arabidopsis* (Table 2). Resistant addition lines have been obtained from *B. napus–S. arvensis* hybrids (Snowdon et al., 2000). Bohman et al. (2002) showed that an introgression of genes carried by chromosome 3 of *A. thaliana* confers adult leaf resistance in *B. napus*.

Table 2. Introgression of resistance genes from related species into a *Brassica napus* genetic background

Donor species		Resistance tests	Genetic control	References
Diploid species	*B. rapa* (AA, $2n=20$)	Cotyledon, leaf, field	Dominant gene	Crouch et al. (1994)
		Field	Dominant gene	Li and Cowling (2003)
		Cotyledon	Dominant genes	Chèvre et al. (2003)
		Cotyledon, field	Two genes, *LepR1* and *LepR2*	Yu et al. (2005)
	B. nigra (BB, $2n=16$)	Leaf, stem	–	Sjödin and Glimelius (1989)*
		Petiole	Three additional chromosomes	Zhu et al. (1993)
		Cotyledon, field	One additional chromosome	Chèvre et al. (1996)
		Petiole	Dominant gene, *PhR1*	Plieske et al. (1998)
		Leaf	Two independent dominant genes, *LmBR2* and *LmBR3*	Dixelius (1999)*
		Cotyledon, leaf	Three independent genes	Dixelius and Whalberg (1999)*
		Stem, field	–	Ogbonnaya et al. (2003)
	Sinapis arvensis (SarSar, $2n=18$)	Cotyledon, stem	One additional chromosome	Snowdon et al. (2000)
	Arabidopsis thaliana (AtAt, $2n=10$)	Leaf	–	Bohman et al. (2002)
Allotetraploid species	*B. juncea* (AABB, $2n=36$)	Cotyledon, field	Dominant gene(s)	Roy (1978, 1984)
		Seed, cotyledon, leaf	Dominant character	Sacristan and Gerdemann (1986)
		Leaf, stem	–	Sjödin and Glimelius (1989)*
		Stem	Three genes with interaction	Pang and Halloran (1996a)
		Cotyledon, field	Dominant gene, *JLm1*	Chèvre et al. (1997), Barret et al. (1998)
		Petiole	Dominant gene, *PhR2*	Plieske et al. (1998)
		Cotyledon, stem	–	Winter et al. (1999)
		Leaf	Dominant gene, *LmBR1*	Dixelius (1999)*
		Cotyledon, leaf	Three independent genes	Dixelius and Whalberg (1999)*
		Cotyledon	Recessive gene, r_jlm2	Saal et al. (2004)
	B. carinata (BBCC, $2n=34$)	Leaf, stem	–	Sjödin and Glimelius (1989)*
		Petiole	Dominant gene, *PhR3*	Plieske et al. (1998)
		Cotyledon, leaf	Three independent genes	Dixelius and Whalberg (1999)*

*Material produced from symmetric and asymmetric protoplast fusions; – no information.

Correlation between race specific resistance at seedling and adult stages

Comparisons between seedling (cotyledon test) and adult (petiole or stem inoculation in glasshouse or field tests) resistance screening tests have produced either significant positive (McNabb et al., 1993; Bansal et al., 1994) or non-significant (Ballinger and Salisbury, 1996; Pang and Halloran, 1996b) correlations. These differences may be explained by differences between sources of resistance studied (conferring either race non-specific quantitative resistance *versus* race specific resistance or a combination of both resistance types) and differences in combinations of avirulence genes between *L. maculans* isolates used in controlled environment tests and *L. maculans* populations in field tests. Another explanation is that isolates may interact with each other. For example, Mahuku et al. (1996) reported that the weakly virulent *L. biglobosa* can induce resistance in *B. napus* to the highly virulent *L. maculans*.

The effectiveness of race specific resistance genes at growth stages later than seedlings has been clearly demonstrated. The effect of *LEM1* in 'Major' was detected using a stem inoculation test (Ferreira et al., 1995). The *LmFr1* gene from 'Crésor' accounted for 57–84% of the variation in resistance in a segregating doubled haploid (DH) population in field trials, depending on the year/location of the trial (Dion et al., 1995). The *Rlm1* gene in 'Maxol' explained 70% of the phenotypic variation for resistance in a field trial (Delourme et al., 2004). Currently, the *Rlm7* resistance is 100% effective in France because nearly 100% of field isolates of *L. maculans* harbour *AvrLm7* (Balesdent et al., 2005). Similarly, cultivars/lines with race specific resistance genes introgressed from *B. rapa* var. *sylvestris* (*LepR1, LepR2* and *LepR3*) were highly resistant to *L. maculans* in field trials (Li and Cowling, 2003; Yu et al., 2005), except for those with *LepR3*, which has been overcome, so that large yield losses have occurred in regions of south eastern Australia (Sprague et al., 2005). Furthermore, resistance genes introgressed into *B. napus* from *B. nigra* (Chèvre et al., 1996) or *B. juncea* (Roy, 1984; Chèvre et al., 1997) are generally effective in field trials.

Conversely, Zhu and Rimmer (2003), comparing the results of cotyledon and stem inoculation tests on lines 'RB87-62' and 'DH88-762', concluded that distinct but linked genes were effective in each line at the seedling and adult stages. The effect of *LEM1* was not detected in field trials where *L. maculans* isolates were predominantly of the same pathogenicity group (PG2) as the isolate used to identify this gene at the seedling stage. However, some isolates in the *L. maculans* field population were highly virulent on 'Major'. A difference in avirulence allele composition between *L. maculans* isolates could explain the contrasting response of 'Major' in controlled environment and field experiments (Ferreira et al., 1995) since PG2 isolates can be either virulent or avirulent on lines with resistance conferred by *Rlm4* (Badawy et al., 1991). Consequently, in the field, the effect of a race specific resistance gene will depend on the *L. maculans* population structure, i.e. on the frequency of the corresponding avirulence allele. However, the threshold frequency of the virulence allele at which the corresponding resistance gene is no longer effective in protecting the crop is not known (Brun et al., 2004).

Quantitative resistance in B. napus

A high level of field resistance to *L. maculans* in the absence of effective race specific resistance genes has been observed in winter European *B. napus* cultivars such as 'Jet Neuf', which is one of the best known sources of quantitative resistance to *L. maculans*. Cultivar Jet Neuf was widely grown all over Europe during the 1970s and 1980s and is still very resistant to *L. maculans*. The major sources of resistance used in the Australian *B. napus* breeding programmes have been Japanese spring types and French winter types (Roy et al., 1983). Although Japanese lines such as 'Chikuzen' and 'Chisaya' are only moderately resistant to *L. maculans*, resistant selections from crosses between these and other lines were obtained. Two other Japanese cultivars ('Norin 20' and 'Mutu') also showed resistance and have been widely used in breeding programmes (Salisbury and Wratten, 1999). There is usually no difference in the development of phoma leaf spot symptoms on young plants between cultivars with quantitative resistance to *L. maculans* and cultivars without it, but later in the season stem cankers do not develop or are less severe on the cultivars with quantitative resistance than those without it. *L. maculans* can

survive and reproduce on even the most resistant lines (Marcroft et al., 2004). As quantitative resistance is partial, when *L. maculans* inoculum concentrations are high, it may not prevent large yield losses (Salisbury et al., 1995; Khangura and Barbetti, 2001; Marcroft et al., 2003).

Screening for quantitative resistance is primarily done by assessment of stem cankers on mature plants in field nurseries where plants have been exposed to the locally prevalent mixture of *L. maculans* races. Phoma stem canker severity is assessed using a disease index based on the extent of external and internal necrosis at the crown (stem base) of plants sampled just before harvest. Controlled environment tests for quantitative resistance using inoculation of leaves, petioles or stems with *L. maculans* have also been proposed (Newman and Bailey, 1987; Kutcher et al., 1993; McNabb et al., 1993; Bansal et al., 1994; Ballinger and Salisbury, 1996; Pang and Halloran, 1996b). With these tests, the correct evaluation of the quantitative resistance of a *B. napus* genotype depends on the *L. maculans* isolate used. Since the effect of a race specific resistance gene is detectable at later growth stages, *L. maculans* isolates that are virulent against any race specific resistance gene(s) present in the genotype to be tested must be used. Similarly, a cultivar carrying a new race specific resistance gene that is effective against all or most of the *L. maculans* isolates in a field population cannot be evaluated for quantitative resistance in that field. A controlled environment test can be done, provided a *L. maculans* isolate virulent against that particular race specific resistance gene is used.

Little information is available on the genetic control of quantitative resistance to *L. maculans*. Ferreira et al. (1995) detected two QTL, which were associated with field resistance in Manitoba, on LG12 and LG21. The genetic basis of quantitative resistance in the French winter oilseed rape 'Darmor', derived from 'Jet Neuf', has been studied. In the 'Darmor-*bzh*'×'Yudal' cross, Pilet et al. (1998) identified a total of ten QTL for resistance, of which four were associated with decreased stem canker severity and decreased plant death in both seasons of field experiments. Analysis of progeny derived from a 'Darmor'×'Samourai' cross, consisting of one DH population and a number of $F_{2:3}$ families, identified six QTL in the DH population and four QTL in the $F_{2:3}$ families (Pilet et al., 2001). Out of a total of sixteen loci detected in the four cultivars, only four QTL were common to the 'Darmor-*bzh*'×'Yudal' and 'Darmor'×'Samourai' crosses. Pilet et al. (2001) concluded that the genetic background contributes greatly to the observed QTL and that the concentration of *L. maculans* inoculum at each location is probably important in revealing QTL with small contributions to overall field resistance to *L. maculans*.

The genomic regions carrying the most consistent resistance QTL in 'Darmor' do not correspond to the two regions on LG10 and LG16 identified as carrying race specific resistance genes to *L. maculans* (Delourme et al., 2004). The position of *Rlm2* on LG16 corresponds to a QTL identified for adult plant resistance in the 'Darmor'×'Samourai' DH population (Pilet et al., 2001). The cultivar Samourai carries both the resistance allele at this QTL and *Rlm2*. Since no French isolates of *L. maculans* carry *AvrLm2* (Rouxel et al., 2003), two hypotheses can be proposed to explain this co-location; either the *Rlm2* gene has a residual effect at the adult plant stage, similar to that suggested in other pathosystems, or genes linked to *Rlm2* are responsible for part of the variation for resistance at this QTL.

Towards identification of the function of resistance genes

Although resistance genes have been cloned from many plant species, including the model species *A. thaliana*, none has yet been characterised in *Brassica* species. Expressed Sequence Tags (ESTs) were derived from *B. napus* 'Glacier' leaves inoculated with a *L. maculans* PG2 isolate (Fristensky et al., 1999). Resistance gene analogues (RGA), either derived from ESTs that have sequence homology to cloned resistance genes or from PCR products amplified with primers based on the conserved nucleotide binding site and leucine-rich repeat regions of cloned genes, have been mapped in *B. napus* (Joyeux et al., 1999; Sillito et al., 2000, Fourmann et al., 2001). Some of these RGA mapped to LG N7 carrying race specific genes for resistance to *L. maculans* (Sillito et al., 2000). A *B. nigra* cDNA sequence, denoted *Lm1*, improved resistance to *L. maculans* in both cotyledons and leaves when it was expressed in transgenic oilseed rape (Wretblad et al., 2003). Identification of differential gene expression using microarray

technology has been used to understand the interactions between *L. maculans* and resistant (*LepR3* gene)/susceptible host plants (Kaur et al., 2004). Work is in progress to clone the 'Crésor' resistance gene (I Parkin, unpubl.) and the *LepR3* gene introgressed from *B. rapa* var. *sylvestris* (Larkan et al., 2004). Isolation of *Rlm* genes that correspond to the avirulence genes that are being cloned in *L. maculans* (Kuhn et al., 2006) will make the *B. napus*/*L. maculans* pathosystem an excellent model system for studies of the molecular interactions between a host and its pathogen.

Conclusions

Our understanding of *L. maculans*/*Brassica* interactions has increased greatly in recent years with developments in genetic studies on both the pathogen and the host plant, and with increased knowledge of the distribution of avirulence alleles in *L. maculans* populations (Balesdent et al., 2005; Stachowiak et al., 2006). To understand the interactions, it is necessary to distinguish between two types of resistance; a qualitative resistance effective from the seedling to the adult plant stage (race specific resistance) and a quantitative adult-stage resistance that is controlled by many genes with small individual effects. Field resistance can be conferred by race specific major genes and/or by polygenes. Partial resistance in the field can be due either to a major gene on which the *L. maculans* population is partly virulent or to quantitative resistance. It is only through investigating the presence of race specific resistance gene(s) in the *B. napus* genotype tested and avirulence alleles in the *L. maculans* isolates used in controlled environment or field experiments that the type of resistance can be determined. Currently, it seems that the genes involved in race specific resistance and polygenic race non-specific resistance are distinct. However, mechanisms leading to quantitative resistance can be effective at different stages of epidemic development and may differ depending on the resistance source. A better understanding of the mechanisms underlying quantitative resistance would help our understanding of the relationships between quantitative and major resistance genes.

Leptosphaeria maculans populations have a very great potential to evolve to virulence under selection pressure exerted by race specific resistance genes and single resistance genes do not provide a durable resistance. This has been shown both in a field experiment using the *Jlm1*/*Rlm6* gene introgressed into *B. napus* from *B. juncea* (Brun et al., 2000) and in commercial crops for the *Rlm1* cultivars and the *LepR3* gene introgressed from *B. rapa* var. *sylvestris* (Li et al., 2003, 2005; Rouxel et al., 2003; Sprague et al., 2006). Polygenic resistance has generally been considered durable. This is supported by evidence for the commercial cultivar Jet Neuf. However, the polygenic resistance in some *B. napus* cultivars has become less effective with time. Sprague et al. (2006) reported that, after its release in 1993, 'Rainbow' maintained its Australia Blackleg Rating (ABR) for resistance at 6.5 until 2000 but it decreased to 5.5 in 2004 while the ABR of 'Ripper' decreased more rapidly, from 7.5 in 2000 to 5.0 in 2004. This is presumed to be the result of changes in virulence and aggressiveness (the ability to cause more severe disease) of the *L. maculans* population. It is difficult to test experimentally the hypothesis that aggressiveness has changed, since this requires multi-season, multi-location field testing of *B. napus* cultivars and continual monitoring of the pathogen population for aggressiveness and frequency of different pathotypes.

To maximise durability of resistance, it is necessary to identify as many different resistance genes as possible to diversify their use and establish strategies to manage them through genotype construction and deployment. To achieve this objective, there is a need to improve characterisation of the race specific resistance genes and QTL for non specific resistance to *L. maculans*.

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