(McDonald and Linde 2002). There is a need for a better understanding of the mechanisms of resistance against *P. brassicae* to inform the search for novel sources of resistance in oilseed rape and other *Brassicas*, where cultivar resistance against *P. brassicae* has been poorly documented.

Analysis of infection and colonisation stages of pathogen life cycles is useful to identify possible resistance mechanisms operating in the host against that particular pathogen. The potential mechanisms of *B. napus* resistance in relation to the *P. brassicae* life cycle have been reviewed by Boys *et al*. (2007). Subcuticular colonisation by the pathogen during its asymptomatic growth phase can be a key trigger for host resistance, which may operate to delay the accumulation of pathogen biomass and prevent production of asexual spores (Boys *et al*. 2007, 2012). There have been several studies on the operation of both major-gene-mediated and quantitative resistance against *P. brassicae* in *B. napus* (Table 1). A resistant phenotype associated with the formation of black necrotic flecking on leaves of infected plants (Fig. 2) has been described and the locus for resistance has subsequently been mapped (Bradburne *et al*. 1999; Boys *et al*. 2012). However, host resistance against *P. brassicae* may not always be associated with this phenotype (Bradburne *et al*. 1999). Major-gene-mediated resistance described in these studies appears to limit subcuticular colonisation and/or the asexual sporulation of *P. brassicae*, but with no effect on subsequent sexual sporulation of the pathogen (Boys *et al*. 2007, 2012). Further characterisation of the genetic basis of the resistance loci identified can provide useful information to search for new sources of resistance.

Involvement of gene-for-gene interactions between *P. brassicae* and *B. oleracea* was reported by Simons and Skidmore (1988). Their experiment on F_1 hybrid lines of cabbage and Brussels sprouts showed differential interactions with *P. brassicae* isolates tested. In addition to cultivar-specific resistance, pre-existing structural host defence mechanisms such as cuticle thickness and composition may provide resistance. Increased susceptibility to *P. brassicae* has been reported after application of herbicides such as dalapon (2,2-dichloropropionoc acid) that alter the epicuticular wax structure (Rawlinson *et al*. 1978*a*). Plant tolerance and disease escape can also play an important part in minimising yield loss by restricting pathogen penetration and the amount of inoculum. For example, delayed senescence in oilseed rape leaves can reduce the ascospore inoculum for new infections later in the cropping season (Boys *et al*. 2007).

Table 1. Research on identification and mapping of resistance against *Pyrenopeziza brassicae* in doubled-haploid populations of oilseed rape

Study	Method of assessment	Type of resistance	Resistant phenotype	QTLs and corresponding chromosomes identified
Pilet *et al*. 1998	Plots assessed for disease severity on leaves and stems using 11-point scale (1, healthy appearance of plots; 11, severely damaged plants)	Quantitative resistance		Ten (six environmentally stable) QTLs identified
Bradburne *et al*. 1999	Cotyledons scored for presence/absence of *P. brassicae* asexual sporulation and presence or absence of black flecking	Major gene-mediated	No sporulation Black flecking	*PBR1*, chromosome A1 *PBR2*, chromosome C6
Boys *et al*. 2012	9-point scale (1, most severe; 9, no symptoms) and % leaf area covered with *P. brassicae* asexual sporulation	Major gene-mediated	Black flecking[A]	Chromosome A1

[A]Prevents asexual reproduction; allows sexual reproduction.

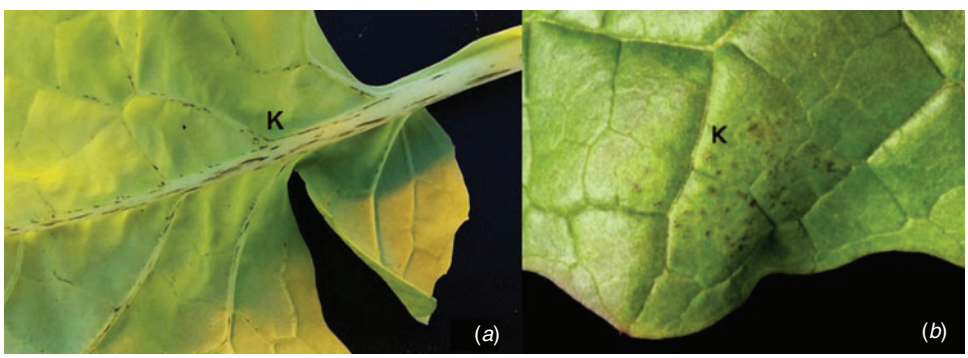

Fig. 2. Black necrotic flecking (K) on *Brassica napus* cv. Imola, which has a major gene for resistance against *Pyrenopeziza brassicae*: (*a*) along the leaf veins at 23 days post-inoculation (leaves were spray-inoculated with a mixture of *P. brassicae* populations collected from diseased oilseed rape leaves from winter oilseed rape crops); (*b*) on the leaf lamina at 28 days after point-inoculation with a suspension of *P. brassicae* conidia (Boys 2009).

Different components of resistance may contribute differently in minimising yield losses (Boys *et al*. 2007). *R*-gene-mediated resistance is favoured by most plant breeders, because it can completely prevent the disease. Moreover, the selection of such resistance is much more straightforward than selection for quantitative resistance, because of its Mendelian inheritance. Nevertheless, the durability of *R*-gene-mediated resistance can be short, because the selection exerted on the pathogen population selects for virulent pathogen races. Therefore, it is important to consider genetic variation in *P. brassicae* populations to detect the presence of effector genes. Pyramiding several *R* genes in elite cultivars can provide better resistance because it requires several mutations in the pathogen genome to overcome host resistance (McDonald and Linde 2002). Nevertheless, this does not eliminate the risk of selection for virulent pathogen races over time. Rotation of cultivars that contain different *R* genes or growing them together as multilines decreases the rate of selection for virulent alleles (McDonald and Linde 2002). Addition of *R*-gene-mediated resistance into a quantitative resistance background could enable cultivar resistance to last longer (Brun *et al*. 2010).

Novel genomic approaches for rapid identification of *R* genes and pathogenicity determinants

Successful disease management strategies require a thorough understanding of the underpinning molecular mechanisms and the genetic basis of host–pathogen interactions (Burdon *et al*. 2016). Rapid expansion of genomic approaches has enabled significant improvements in control of crop diseases. Improved efficiency and cost-effectiveness of next generation sequencing (NGS) technologies have allowed whole-genome sequences of numerous crop and pathogen species to be generated. Increasing availability of *Brassica* genomic information offers new possibilities for the identification of host resistance and new opportunities to provide molecular tools to assist in breeding for disease resistance.

The genomes of five *Brassica* species have been sequenced. The first *Brassica* genome sequence was obtained from *B. rapa* (Wang *et al*. 2011). Genome sequences of *B. oleracea* (Liu *et al*. 2014) and *B. napus* (Chalhoub *et al*. 2014) followed; *B. napus* is an allotetraploid species that contains A and C sub-genomes from its ancestors, *B. rapa* and *B. oleracea*, respectively. Recently, the genomes of allotetraploid *B. juncea* and its B genome progenitor *B. nigra* were sequenced (Yang *et al*. 2016). The implications of genome-enabled technologies for the breeding of crops have been reviewed (Snowdon and Iniguez Luy 2012). Single-nucleotide polymorphism (SNP) markers and transcriptome sequencing (mRNA-Seq) have been used for genome-wide association studies (GWAS) to identify individual genes that contribute to important agronomic traits (Harper *et al*. 2012). Since then, a *Brassica* 60k SNP array has been used in combination with large association panels by several research teams to analyse the genetic basis of traits, including resistance against pathogens (Li *et al*. 2014; Hatzig *et al*. 2015; Wu *et al*. 2016). Resequencing of 52 diverse natural and synthetic *B. napus* accessions has resulted in identification of >4 million SNPs, which are being exploited for breeding using primary and secondary gene pools (Schmutzer *et al*. 2015).

Transcriptome sequencing has been used to analyse the interaction between *B. napus* and *L. maculans* (Lowe *et al*. 2014; Haddadi *et al*. 2016); both studies have used susceptible cultivars to determine pathogen and host gene expression. Such studies are useful to determine potential pathogenicity (e.g. effector) and resistance genes. Transcriptome analysis can provide valuable information related to quantitative resistance of the host against particular pathogens (Joshi *et al*. 2016; Wu *et al*. 2016).

Pyrenopeziza brassicae is an apoplastic fungal pathogen; *R*-gene-mediated resistance against it is likely to involve receptor-like proteins (RLP), which contribute to recognition of pathogen effectors that are secreted into the extracellular environment of the host (effector-triggered defence, ETD) (Stotz *et al*. 2014). This resistance is different from that involving *R* genes operating against appressorium-forming, cell-penetrating fungal pathogens that cause diseases such as rusts and mildews, which recognise pathogen effectors that are delivered into the cytoplasm of the host cell (effector-triggered immunity, ETI) (Jones and Dangl 2006). The different categories of *R* genes have recently been reviewed (Sekhwal *et al*. 2015). *R*-gene-specific sequence information has recently been exploited for resistance gene enrichment and sequencing (RenSeq) to identify previously unknown *R* genes (Jupe *et al*. 2013). Such approaches, in combination with advanced genome information, hold the promise of rapidly identifying the genetic basis of several resistance traits, including major resistance quantitative trait loci operating against *P. brassicae*.

In contrast to *Brassica* genomic information, little information is available about the *P. brassicae* genome. Research on *P. brassicae–B. napus* interactions provides a framework to understand its pathogenicity; however, the number of factors so far known to be involved in defence signalling pathways is limited. There are substantial improvements in efficiency of DNA-sequencing technologies. Whole-genome sequencing of pathogens allows for genome-wide analysis of pathogenicity-related genes (Klosterman *et al*. 2016). Comparative genomics approaches can be applied between related pathogen species to improve understanding of the pathogenicity in poorly understood pathosystems. Several phytopathogenic fungi are evolutionarily related to *P. brassicae* (Table 2). Sequence information for the internal transcribed spacer (ITS) region provided evidence for a close phylogenetic relationship between *Rhynchosporium commune* (formerly known as *R. secalis*) and two Leotiomycete genera, *Pyrenopeziza* and *Oculimacula* (formerly *Tapesia*) (Goodwin 2002).

The genome of *R. commune* has been sequenced (Penselin *et al*. 2016). Seven proteins with at least one LysM domain, which are mostly found in secreted LysM effectors of fungi, have been identified in the *Rhynchosporium* genome. LysM-domain-containing effector proteins prevent the activation of pathogen associated molecular pattern (PAMP)-triggered immunity by sequestering chitin oligosaccharides. The close phylogenetic relationship between *R. commune* and *P. brassicae* can be exploited to identify whether pathogenicity-related genes of *P. brassicae* and *R. commune* LysM-domain-containing proteins are good candidate effectors for *P. brassicae* infection. Moreover, ~330 cell-wall-degrading enzymes (CWDEs) have been identified in the *R. commune*

Table 2. Phytopathogenic fungi evolutionarily related to *Pyrenopeziza brassicae*

Leotiomycete pathogen genera *Pyrenopeziza*, *Rhynchosporium* and *Oculimacula* were considered to have a close phylogenetic relationship based on sequence information for the internal transcribed spacer (ITS) region (Goodwin 2002). Main host species are listed; diseases are categorised as polycyclic (p) or monocyclic (m)

Pathogen	Disease and the host	Mode of infection	Niche	Pathogenicity factors identified	Genome sequenced	References
Pyrenopeziza brassicae	Light leaf spot on oilseed rape and vegetable brassicas (p)	Cuticular penetration	Subcuticular	Extracellular cutinases, extracellular proteases, cytokinins	No	Li *et al*. 2003; Batish *et al*. 2003; Ashby 1997
Rhynchosporium commune	Leaf blotch on barley (p)	Cuticular penetration	Subcuticular	Necrosis-inducing proteins (NIP), LysM	Yes (Penselin *et al*. 2016)	Kirsten *et al*. 2012; Zhan *et al*. 2008
Oculimacula yallundae and *O. accuformis*	Eyespot on wheat, barley, rye (m)	Cuticular penetration by formation of appresoria	After germination, the pathogen produces a mycelial network on plant surfaces and later colonises leaf sheath and stem cells		No	Crous *et al*. 2003; Blein *et al*. 2009

genome, and considering their putative substrates, ~64% of these were identified to target host cell walls. Gene expression data have been analysed for the necrosis-inducing protein (NIP) and small, secreted effector proteins (Penselin *et al*. 2016). This information can be incorporated into gene expression analysis to identify candidate effector genes. Whole-genome sequencing and re-sequencing of allelic variants can be used as an effective tool for studying pathogen population variation by identifying molecular markers such as microsatellites and SNPs.

Concluding remarks

Understanding of the molecular genetic mechanisms underpinning the *B. napus–P. brassicae* interactions is essential for developing effective, durable disease-management strategies. Although light leaf epidemiology is well understood, substantial gaps remain in understanding of the operation of *Brassica* resistance and *P. brassicae* pathogenicity. With recent advances in *Brassica* genomics and understanding of the genetic basis of resistance against extracellular pathogens (i.e. *B. napus* resistance against *Leptosphaeria maculans*) (Larkan *et al*. 2013, 2015), rapid improvement in identifying novel sources of resistance against *P. brassicae* can be expected. Resistance genes mapped in previous studies can be further examined to characterise the genetic basis of resistance and they can be cloned. This information can be utilised to search for similar genes and to produce molecular markers to facilitate marker-assisted selection (MAS) in oilseed rape breeding programs (Collard *et al*. 2005). However, to achieve effective disease control through deployment of cultivar resistance, considerable improvements in understanding of *P. brassicae* genomics are also needed. Differences in cultivar resistance between different regions in the UK indicate the presence of pathogen population variation, and this can also put pressure on breeding programs. It is important to study this variation by using molecular markers related to pathogenicity. This information will then need to be considered when recommending cultivars for different regions to sustain the available sources of resistance against *P. brassicae*.

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