

(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 *Brassicaceae*, 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 F₁ 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-dichloropropanoic acid) that alter the epicuticular wax structure (Rawlinson *et al.* 1978a). 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 <i>et al.</i> 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 <i>et al.</i> 1999	Cotyledons scored for presence/absence of <i>P. brassicae</i> asexual sporulation and presence or absence of black flecking	Major gene-mediated	No sporulation Black flecking	<i>PBR1</i> , chromosome A1 <i>PBR2</i> , chromosome C6
Boys <i>et al.</i> 2012	9-point scale (1, most severe; 9, no symptoms) and % leaf area covered with <i>P. brassicae</i> asexual sporulation	Major gene-mediated	Black flecking ^A	Chromosome A1

^APrevents asexual reproduction; allows sexual reproduction.

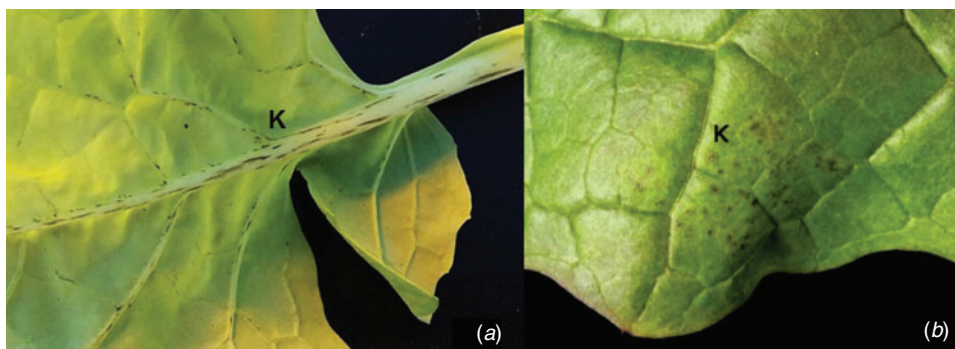


Fig. 2. Black necrotic flecking (K) on *Brassica napus* cv. Imola, which has a major gene for resistance against *Pyrenopeziza brassicae*: (a) along the leaf veins at 23 days post-inoculation (leaves were spray-inoculated with a mixture of *P. brassicae* populations collected from diseased oilseed rape 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
<i>Pyrenopeziza brassicae</i>	Light leaf spot on oilseed rape and vegetable brassicas (p)	Cuticular penetration	Subcuticular	Extracellular cutinases, extracellular proteases, cytokinins	No	Li <i>et al.</i> 2003; Batish <i>et al.</i> 2003; Ashby 1997
<i>Rhynchosporium commune</i>	Leaf blotch on barley (p)	Cuticular penetration	Subcuticular	Necrosis-inducing proteins (NIP), LysM	Yes (Penselin <i>et al.</i> 2016)	Kirsten <i>et al.</i> 2012; Zhan <i>et al.</i> 2008
<i>Oculimacula yallundae</i> and <i>O. accuformis</i>	Eyespot on wheat, barley, rye (m)	Cuticular penetration by formation of appressoria	After germination, the pathogen produces a mycelial network on plant surfaces and later colonises leaf sheath and stem cells		No	Crous <i>et al.</i> 2003; Blein <i>et al.</i> 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*.

Acknowledgements

We are grateful for funding provided by AHDB Cereals and Oilseeds, AHDB Horticulture, the Felix Thornley Cobbold Agricultural Trust, Biotechnology and Biological Sciences Research Council (BBSRC) (project no. BB/N005112/1), the Gen Foundation and the University of Hertfordshire. We also acknowledge in-kind contributions from Mark Nightingale (Elsoms Seeds UK Ltd), Dr Vasilis Gegas (Limagrain UK Ltd), Dr Faye Ritchie (ADAS UK Ltd) and Dr Neal Evans (Weather Innovations (WIN)). We thank Dr Emily Graham (Née Boys) for her kind permission to reproduce Figs 1b and 2b and Dr Andreas von Tiedemann (Georg-August Universität Göttingen) for facilitating plant growth experiments (CAK).

References

- AHDB Cereals & Oilseeds (2016) AHDB Recommended Lists for Cereals and Oilseeds (2016/17). *Agriculture and Horticulture Development Board*. Available at: <https://cereals.ahdb.org.uk/varieties/ahdb-recommended-lists/rl-archive-2015-16.aspx> (accessed 30 September 2016)
- Ashby AM (1997) A molecular view through the looking glass: the *Pyrenopeziza brassicae*–*Brassica* interaction. *Advances in Botanical Research* **24**, 31–70. doi:10.1016/S0065-2296(08)60070-1
- Barrett LG, Thrall PH, Burdon JJ, Linde CC (2008) Life history determines genetic structure and evolutionary potential of host–parasite interactions. *Trends in Ecology & Evolution* **23**, 678–685. doi:10.1016/j.tree.2008.06.017
- Batish S, Hunter A, Ashby AM, Johnstone K (2003) Purification and biochemical characterisation of Psp1, an extracellular protease produced by the oilseed rape pathogen *Pyrenopeziza brassicae*. *Physiological and Molecular Plant Pathology* **62**, 13–20. doi:10.1016/S0885-5765(03)00022-5
- Blein M, Levrel A, Lemoine J, Gautier V, Chevalier M, Barloy D (2009) *Oculimacula yallundae* lifestyle revisited: relationships between the timing of eyespot symptom appearance, the development of the pathogen and the responses of infected partially resistant wheat plants. *Plant Pathology* **58**, 1–11. doi:10.1111/j.1365-3059.2008.01940.x

- Boys EF (2009) Resistance to *Pyrenopeziza brassicae* (light leaf spot) in *Brassica napus* (oilseed rape). PhD Thesis, University of Nottingham, UK.
- Boys EF, Roques SE, Ashby AM, Evans N, Latunde-Dada AO, Thomas JE, West JS, Fitt BDL (2007) Resistance to infection by stealth: *Brassica napus* (winter oilseed rape) and *Pyrenopeziza brassicae* (light leaf spot). *European Journal of Plant Pathology* **118**, 307–321. doi:10.1007/s10658-007-9141-9
- Boys EF, Roques SE, West JS, Werner CP, King GJ, Dyer PS, Fitt BDL (2012) Effects of *R* gene-mediated resistance in *Brassica napus* (oilseed rape) on asexual and sexual sporulation of *Pyrenopeziza brassicae* (light leaf spot). *Plant Pathology* **61**, 543–554. doi:10.1111/j.1365-3059.2011.02529.x
- Bradburne R, Majer D, Magreth R, Werner C, Lewis B, Mithen R (1999) Winter oilseed rape with high levels of resistance to *Pyrenopeziza brassicae* derived from wild *Brassica* species. *Plant Pathology* **48**, 550–558. doi:10.1046/j.1365-3059.1999.00373.x
- Brun H, Chèvre AM, Fitt BDL, Powers S, Besnard AL, Ermel M, Huteau V, Marquer B, Eber F, Renard M (2010) Quantitative resistance increases the durability of qualitative resistance to *Leptosphaeria maculans* in *Brassica napus*. *New Phytologist* **185**, 285–299. doi:10.1111/j.1469-8137.2009.03049.x
- Brunner PC, Torriani SFF, Croll D, Stukenbrock EH, McDonald BA (2013) Coevolution and life cycle specialization of plant cell wall degrading enzymes in a hemibiotrophic pathogen. *Molecular Biology and Evolution* **30**, 1337–1347. doi:10.1093/molbev/mst041
- Burdon JJ, Zhan J, Barrett LG, Papaix J, Thrall PH (2016) Addressing the challenges of pathogen evolution on the world's arable crops. *Phytopathology* **106**, 1117–1127. doi:10.1094/PHYTO-01-16-0036-FI
- Carter HE, Cools HJ, West JS, Shaw MW, Fraaije BA (2013) Detection and molecular characterisation of *Pyrenopeziza brassicae* isolates resistant to methyl benzimidazole carbamates. *Pest Management Science* **69**, 1040–1048. doi:10.1002/ps.3585
- Carter HE, Fraaije BA, West JS, Kelly SL, Mehl A, Shaw MW, Cools HJ (2014) Alterations in the predicted regulatory and coding regions of the sterol 14 α -demethylase gene (*CYP51*) confer decreased azole sensitivity in the oilseed rape pathogen *Pyrenopeziza brassicae*. *Molecular Plant Pathology* **15**, 513–522. doi:10.1111/mpp.12106
- Chalhoub B, Denoeud F, Liu S, Parkin IAP, Tang H, Wang X, Chiquet J, Belcram H, Tong C, Samans B, Corréa M, Da Silva C, Just J, Falentin C, Koh CS, Le Clainche I, Bernard M, Bento P, Noel B, Labadie K, Alberti A, Charles M, Arnaud D, Guo H, Daviaud C, Alameiry S, Jabbari K, Zhao M, Edger PP, Chelaifa H, Tack D, Lassalle G, Mestiri I, Schnel N, Le Paslier MC, Fan G, Renault V, Bayer PE, Golicz AA, Manoli S, Lee TH, Thi VND, Chalabi S, Hu Q, Fan C, Tollenaere R, Lu Y, Battail C, Shen J, Sidebottom CHD, Wang X, Canaguier A, Chauveau A, Bérard A, Deniot G, Guan M, Liu Z, Sun F, Lim YP, Lyons E, Town CD, Bancroft I, Wang X, Meng J, Ma J, Pires JC, King GJ, Brunel R, Delourme R, Renard M, Aury JM, Adams KL, Batley J, Snowdon RJ, Tost J, Edwards D, Zhou Y, Hua W, Sharpe AG, Paterson AH, Guan C, Wincker P (2014) Early allopolyploid evolution in the post-Neolithic *Brassica napus* oilseed genome. *Science* **345**, 950–953. doi:10.1126/science.1253435
- Cheah LH, Hartill WFT, Corbin JB (1980) First report of the natural occurrence of *Pyrenopeziza brassicae* Sutton et Rawlinson, the apothecial state of *Cylindrosporium concentricum* Greville, in brassica crops in New Zealand. *New Zealand Journal of Botany* **18**, 197–202. doi:10.1080/0028825X.1980.10426917
- Collard BCY, Jahufer MZZ, Brouwer JB, Pang ECK (2005) An introduction to markers, quantitative trait loci (QTL) mapping and marker-assisted selection for crop improvement: the basic concepts. *Euphytica* **142**, 169–196. doi:10.1007/s10681-005-1681-5
- Courtice GRM, Ingram DS (1987) Isolation of auxotrophic mutants of the hemibiotrophic ascomycete pathogen of brassicas, *Pyrenopeziza brassicae*. *Transactions of the British Mycological Society* **89**, 301–306. doi:10.1016/S0007-1536(87)80110-9
- CropMonitor (2016) Survey of commercially grown winter oilseed rape. Department for Environment, Food and Rural Affairs. Available at: www.cropmonitor.co.uk/wosr/surveys/wosr.cfm (accessed 1 August 2016)
- Crous PW, Groenewald JZ, Gams W (2003) Eyespot of cereals revisited: ITS phylogeny reveals new species relationships. *European Journal of Plant Pathology* **109**, 841–850. doi:10.1023/A:102611030426
- Davies KA, De Loro I, Foster SJ, Li D, Johnstone K, Ashby AM (2000) Evidence for a role of cutinase in pathogenicity of *Pyrenopeziza brassicae* on brassicas. *Physiological and Molecular Plant Pathology* **57**, 63–75. doi:10.1006/pmpp.2000.0282
- Defra (2016) Horticulture statistics. Department for Environment, Food and Rural Affairs. Available at: www.gov.uk/government/collections/horticultural-statistics (accessed 22 July 2016)
- Evans N, Baierl A, Brain P, Welham SJ, Fitt BDL (2003) Spatial aspects of light leaf spot (*Pyrenopeziza brassicae*) epidemic development on winter oilseed rape (*Brassica napus*) in the United Kingdom. *Phytopathology* **93**, 657–665. doi:10.1094/PHYTO.2003.93.6.657
- Evans N, Butterworth MH, Baierl A, Semenov MA, West JS, Barnes A, Moran D, Fitt BDL (2010) The impact of climate change on disease constraints on production of oilseed rape. *Food Security* **2**, 143–156. doi:10.1007/s12571-010-0058-3
- Figuerola L, Shaw MW, Fitt BDL, McCartney HA, Welham SJ (1994) Effects of previous cropping and fungicide timing on the development of light leaf spot (*Pyrenopeziza brassicae*), seed yield and quality of winter oilseed rape (*Brassica napus*). *Annals of Applied Biology* **124**, 221–239. doi:10.1111/j.1744-7348.1994.tb04130.x
- Figuerola L, Fitt BDL, Shaw MW, McCartney HA, Welham SJ (1995) Effects of temperature on the development of light leaf spot (*Pyrenopeziza brassicae*) on oilseed rape (*Brassica napus*). *Plant Pathology* **44**, 51–62. doi:10.1111/j.1365-3059.1995.tb02715.x
- Fitt BDL, Doughty KJ, Gilles T, Gladders P, Steed JM, Su H, Sutherland KG (1998a) Methods for assessment of light leaf spot (*Pyrenopeziza brassicae*) on winter oilseed rape (*Brassica napus*) in the UK. *Annals of Applied Biology* **133**, 329–341. doi:10.1111/j.1744-7348.1998.tb05834.x
- Fitt BDL, Doughty KJ, Gladders P, Steed JM, Sutherland KG (1998b) Diagnosis of light leaf spot (*Pyrenopeziza brassicae*) on winter oilseed rape (*Brassica napus*) in the UK. *Annals of Applied Biology* **133**, 155–166. doi:10.1111/j.1744-7348.1998.tb05816.x
- Fitt BDL, Fraaije BA, Chandramohan P, Shaw MW (2011) Impacts of changing air composition on severity of arable crop disease epidemics. *Plant Pathology* **60**, 44–53. doi:10.1111/j.1365-3059.2010.02413.x
- Gilles T, Evans N, Fitt BDL, Jeger MJ (2000) Epidemiology in relation to methods for forecasting light leaf spot (*Pyrenopeziza brassicae*) severity on winter oilseed rape (*Brassica napus*) in the UK. *European Journal of Plant Pathology* **106**, 593–605. doi:10.1023/A:1008701302853
- Gilles T, Ashby AM, Fitt BDL, Cole T (2001a) Development of *Pyrenopeziza brassicae* apothecia on agar and oilseed rape debris. *Mycological Research* **105**, 705–714. doi:10.1017/S0953756201003902
- Gilles T, Fitt BDL, Jeger MJ (2001b) Effects of environmental factors on development of *Pyrenopeziza brassicae* (light leaf spot) apothecia on oilseed rape debris. *Phytopathology* **91**, 392–398. doi:10.1094/PHYTO.2001.91.4.392
- Gilles T, Fitt BDL, McCartney HA, Papastamati K, Steed JM (2001c) The roles of ascospores and conidia of *Pyrenopeziza brassicae* in light leaf spot epidemics on winter oilseed rape (*Brassica napus*) in the UK. *Annals of Applied Biology* **138**, 141–152. doi:10.1111/j.1744-7348.2001.tb00096.x
- Goodwin SB (2002) The barley scald pathogen *Rhynchosporium secalis* is closely related to the discomycetes *Tapesia* and *Pyrenopeziza*. *Mycological Research* **106**, 645–654. doi:10.1017/S0953756202006007

- Haddadi P, Ma L, Wang H, Borhan MH (2016) Genome-wide transcriptomic analyses provide insights into the lifestyle transition and effector repertoire of *Leptosphaeria maculans* during the colonization of *Brassica napus* seedlings. *Molecular Plant Pathology* **17**, 1196–1210. doi:10.1111/mpp.12356
- Harper AL, Trick M, Higgins J, Fraser F, Clissold L, Wells R, Hattori C, Werner P, Bancroft I (2012) Associative transcriptomics of traits in the polyploid crop species *Brassica napus*. *Nature Biotechnology* **30**, 798–802. doi:10.1038/nbt.2302
- Hatzig SV, Frisch M, Breuer F, Nesi N, Ducourmau S, Wagner M-H, Leckband G, Abbadi A, Snowdon RJ (2015) Genome-wide association mapping unravels the genetic control of seed germination and vigor in *Brassica napus*. *Frontiers in Plant Science* **6**, 221–233. doi:10.3389/fpls.2015.00221
- Hickman CJ, Schofield ER, Taylor RE (1955) Light leaf spot of Brassicaceae. *Plant Pathology* **4**, 129–131. doi:10.1111/j.1365-3059.1955.tb00060.x
- Ilott TW, Ingram DS, Rawlinson CJ (1984) Heterothallism in *Pyrenopeziza brassicae*, cause of light leaf spot of brassicas. *Transactions of the British Mycological Society* **82**, 477–483. doi:10.1016/S0007-1536(84)80012-1
- Jones JGD, Dangl JL (2006) The plant immune system. *Nature* **444**, 323–329. doi:10.1038/nature05286
- Joshi RK, Megha S, Rahman MH, Basu U, Kav NN (2016) A global study of transcriptome dynamics in canola (*Brassica napus* L.) responsive to *Sclerotinia sclerotiorum* infection using RNA-Seq. *Gene* **590**, 57–67. doi:10.1016/j.gene.2016.06.003
- Jupe F, Witek K, Verweij W, Śliwka J, Pritchard L, Etherington GJ, Maclean D, Cock PJ, Leggett RM, Bryan GJ (2013) Resistance gene enrichment sequencing (RenSeq) enables reannotation of the NB-LRR gene family from sequenced plant genomes and rapid mapping of resistance loci in segregating populations. *The Plant Journal* **76**, 530–544. doi:10.1111/tpl.12307
- Karolewski Z (1999) The occurrence of light leaf spot on winter oilseed rape in Western Poland in 1991–1996 and the characteristics of *Pyrenopeziza brassicae* isolates. *Phytopathologia Polonica* **18**, 113–121.
- Karolewski Z (2010) Development of light leaf spot (*Pyrenopeziza brassicae*) on brassicas. *Phytopathologia* **55**, 13–20.
- Karolewski Z, Fitt BDL, Latunde-Dada AO, Foster SJ, Todd AD, Downes K, Evans N (2006) Visual and PCR assessment of light leaf spot (*Pyrenopeziza brassicae*) on winter oilseed rape (*Brassica napus*) cultivars. *Plant Pathology* **55**, 387–400. doi:10.1111/j.1365-3059.2006.01383.x
- Karolewski Z, Kaczmarek J, Jedryczka M, Cools HJ, Fraaije BA, Lucas JA, Latunde-Dada AO (2012) Detection and quantification of airborne inoculum of *Pyrenopeziza brassicae* in Polish and UK winter oilseed rape crops by real-time PCR assays. *Grana* **51**, 270–279. doi:10.1080/0017134.2011.653401
- Kirsten S, Navarro-Quezada A, Penselin D, Wenzel C, Matern A, Leitner A, Baum T, Seiffert U, Knogge W (2012) Necrosis-inducing proteins of *Rhynchosporium commune*, effectors in quantitative disease resistance. *Molecular Plant-Microbe Interactions* **25**, 1314–1325. doi:10.1094/MPMI-03-12-0065-R
- Klosterman S, Rollins J, Sudarshana M, Vinatzer B (2016) Disease management in the genomics era—summaries of focus issue papers. *Phytopathology* **106**, 1068–1070. doi:10.1094/PHYTO-07-16-0276-FI
- Koike S, Gladders P, Paulus A (2007) 'Vegetable diseases: a color handbook.' (Manson Publishing Limited: London)
- Lacey ME, Rawlinson CJ, McCartney HA (1987) First record of the natural occurrence in England of the teleomorph of *Pyrenopeziza brassicae* on oilseed rape. *Transactions of the British Mycological Society* **89**, 135–140. doi:10.1016/S0007-1536(87)80074-8
- Larkan NJ, Lydiate DJ, Parkin IAP, Nelson MN, Epp DJ, Cowling WA, Rimmer SR, Borhan MH (2013) The *Brassica napus* blackleg resistance gene *LepR3* encodes a receptor-like protein triggered by the *Leptosphaeria maculans* effector *AVRLm1*. *New Phytologist* **197**, 595–605. doi:10.1111/nph.12043
- Larkan NJ, Ma L, Borhan MH (2015) The *Brassica napus* receptor-like protein *RLM2* is encoded by a second allele of the *LepR3/Rlm2* blackleg resistance locus. *Plant Biotechnology Journal* **13**, 983–992. doi:10.1111/pbi.12341
- Laugé R, De Wit PJGM (1998) Fungal avirulence genes: structure and possible functions. *Fungal Genetics and Biology* **24**, 285–297. doi:10.1006/fgbi.1998.1076
- Li D, Ashby AM, Johnstone K (2003) Molecular evidence that the extracellular cutinase *Pbc1* is required for pathogenicity of *Pyrenopeziza brassicae* on oilseed rape. *Molecular Plant-Microbe Interactions* **16**, 545–552. doi:10.1094/MPMI.2003.16.6.545
- Li F, Chen B, Xu K, Wu J, Song W, Bancroft I, Harper AL, Trick M, Liu S, Gao G (2014) Genome-wide association study dissects the genetic architecture of seed weight and seed quality in rapeseed (*Brassica napus* L.). *DNA Research* **21**, 355–367. doi:10.1093/dnares/dsu002
- Liu S, Liu Y, Yang X, Tong C, Edwards D, Parkin IAP, Zhao M, Ma J, Yu J, Huang S, Wang X, Wang J, Lu K, Fang Z, Bancroft I, Yang TJ, Hu Q, Wang X, Yue Z, Li H, Yang L, Wu J, Zhou Q, Wang W, King GJ, Pires JC, Lu C, Wu Z, Sampath P, Wang Z, Guo H, Pan S, Yang L, Min J, Zhang D, Jin D, Li W, Belcram H, Tu J, Guan M, Qi C, Du D, Li J, Jiang L, Batley J, Sharpe AG, Park BS, Ruperao P, Cheng F, Waminal NE, Huang Y, Dong C, Wang L, Li J, Hu Z, Zhuang M, Huang Y, Huang J, Shi J, Mei D, Liu J, Lee TH, Wang J, Jin H, Li Z, Li X, Zhang J, Xiao L, Zhou Y, Liu Z, Liu X, Qin R, Tang X, Liu W, Wang Y, Zhang Y, Lee J, Kim HH, Denoeuf F, Xu X, Liang X, Hua W, Wang X, Wang J, Chalhoub B, Paterson AH (2014) The *Brassica oleracea* genome reveals the asymmetrical evolution of polyploid genomes. *Nature Communications* **5**, 3930. doi:10.1038/ncomms4930
- Lowe RGT, Cassin A, Grandaubert J, Clark BL, Van de Wouw AP, Rouxel T, Howlett BJ (2014) Genomes and transcriptomes of partners in plant-fungal-interactions between canola (*Brassica napus*) and two *Leptosphaeria* species. *PLoS One* **9**, e103098. doi:10.1371/journal.pone.0103098
- Maddock SE, Ingram DS (1981) Studies of survival and longevity of the light leaf spot pathogen of brassicas, *Pyrenopeziza brassicae*. *Transactions of the British Mycological Society* **77**, 153–159. doi:10.1016/S0007-1536(81)80189-1
- Maddock SE, Ingram DS, Gilligan CA (1981) Resistance of cultivated brassicas to *Pyrenopeziza brassicae*. *Transactions of the British Mycological Society* **76**, 371–382. doi:10.1016/S0007-1536(81)80063-0
- Majer D, Lewis BG, Mithen R (1998) Genetic variation among field isolates of *Pyrenopeziza brassicae*. *Plant Pathology* **47**, 22–28. doi:10.1046/j.1365-3059.1998.00204.x
- McCartney HA, Lacey ME (1990) The production and release of ascospores of *Pyrenopeziza brassicae* on oilseed rape. *Plant Pathology* **39**, 17–32. doi:10.1111/j.1365-3059.1990.tb02471.x
- McDonald BA (2015) How can research on pathogen population biology suggest disease management strategies? The example of barley scald (*Rhynchosporium commune*). *Plant Pathology* **64**, 1005–1013. doi:10.1111/ppa.12415
- McDonald BA, Linde C (2002) Pathogen population genetics, evolutionary potential, and durable resistance. *Annual Review of Phytopathology* **40**, 349–379. doi:10.1146/annurev.phyto.40.120501.101443
- Mycobank (undated) *Pyrenopeziza brassicae*. International Mycological Association. Available at: www.mycobank.org/name/Pyrenopeziza%20brassicace&Lang=Eng (accessed 6 February 2017).
- Oerke EC (2006) Crop losses to pests. *The Journal of Agricultural Science* **144**, 31–43. doi:10.1017/S0021859605005708
- Oxley SJP, Walters DR (2012) Control of light leaf spot (*Pyrenopeziza brassicae*) on winter oilseed rape (*Brassica napus*) with resistance elicitors. *Crop Protection* **40**, 59–62. doi:10.1016/j.cropro.2012.04.028

- Penselin D, Münsterkötter M, Kirsten S, Felder M, Taudien S, Platzer M, Ashelford K, Paskiewicz KH, Harrison RJ, Hughes DJ, Wolf T, Shelest E, Graap J, Hoffmann J, Wenzel C, Wöltje N, King KM, Fitt BDL, Güldener U, Avrova A, Knogge W (2016) Comparative genomics to explore phylogenetic relationship, cryptic sexual potential and host specificity of *Rhynchosporium* species on grasses. *BMC Genomics* **17**, 953. doi:10.1186/s12864-016-3299-5
- Pilet ML, Delourme R, Foisset N, Renard M (1998) Identification of QTL involved in field resistance to light leaf spot (*Pyrenopeziza brassicae*) and blackleg resistance (*Leptosphaeria maculans*) in winter rapeseed (*Brassica napus* L.). *Theoretical and Applied Genetics* **97**, 398–406. doi:10.1007/s001220050909
- Pöggeler S (2001) Mating-type genes for classical strain improvements of ascomycetes. *Applied Microbiology and Biotechnology* **56**, 589–601. doi:10.1007/s002530100721
- Rawlinson CJ, Muthyalu G, Turner RH (1978a) Effect of herbicides on epicuticular wax of winter oilseed rape (*Brassica napus*) and infection by *Pyrenopeziza brassicae*. *Transactions of the British Mycological Society* **71**, 441–451. doi:10.1016/S0007-1536(78)80071-0
- Rawlinson CJ, Sutton BC, Muthyalu G (1978b) Taxonomy and biology of *Pyrenopeziza brassicae* sp. nov. (*Cylindrosporium concentricum*), a pathogen of winter oilseed rape (*Brassica napus* ssp. *oleifera*). *Transactions of the British Mycological Society* **71**, 425–439. doi:10.1016/S0007-1536(78)80070-9
- Rohe M, Gierlich A, Hermann H, Hahn M, Schmidt B, Rosahl S, Knogge W (1995) The race-specific elicitor, *NIP1*, from the barley pathogen, *Rhynchosporium secalis*, determines avirulence on host plants of the *Rrs1* resistance genotype. *The EMBO Journal* **14**, 4168–4177.
- Rothamsted Research (2016) Regional light leaf spot risk forecast 2016/17 season. Rothamsted Research, UK. Available at: www.rothamsted.ac.uk/light-leaf-spot-forecast/regional-light-leaf-spot-risk-forecast (accessed 8 September 2016).
- Savary S, Ficke A, Aubertot J-N, Hollier C (2012) Crop losses due to diseases and their implications for global food production losses and food security. *Food Security* **4**, 519–537. doi:10.1007/s12571-012-0200-5
- Schmutzer T, Samans B, Dyrszka E, Ulpinnis C, Weise S, Stengel D, Colmsee C, Lespinasse D, Micic Z, Abel S, Duchscherer P, Breuer F, Abbadi A, Leckband G, Snowdon R, Scholz U (2015) Species-wide genome sequence and nucleotide polymorphisms from the model allopolyploid plant *Brassica napus*. *Scientific Data* **2**, 150072. doi:10.1038/sdata.2015.72
- Sekhwil MK, Li P, Lam I, Wang X, Cloutier S, You FM (2015) Disease resistance gene analogs (RGAs) in plants. *International Journal of Molecular Sciences* **16**, 19248–19290. doi:10.3390/ijms160819248
- Siebold M, von Tiedemann A (2012) Potential effects of global warming on oilseed rape pathogens in Northern Germany. *Fungal Ecology* **5**, 62–72. doi:10.1016/j.funeco.2011.04.003
- Simons AJ, Skidmore DI (1988) Race-specific resistance to light leaf spot in *Brassica oleracea*. *Transactions of the British Mycological Society* **90**, 431–435. doi:10.1016/S0007-1536(88)80152-9
- Singh G, Ashby AM (1998) Cloning of the mating type loci from *Pyrenopeziza brassicae* reveals the presence of a novel mating type gene within a discomycete *MAT 1-2* locus encoding a putative metallothionein-like protein. *Molecular Microbiology* **30**, 799–806. doi:10.1046/j.1365-2958.1998.01112.x
- Singh G, Ashby AM (1999) Cloning of the mating type loci from *Pyrenopeziza brassicae* reveals the presence of a novel mating type gene within a discomycete *MAT 1-2* locus encoding a putative metallothionein-like protein. *Molecular Microbiology* **32**, 1115. doi:10.1046/j.1365-2958.1999.01115.x
- Snowdon RJ, Iniguez Luy FL (2012) Potential to improve oilseed rape and canola breeding in the genomics era. *Plant Breeding* **131**, 351–360. doi:10.1111/j.1439-0523.2012.01976.x
- Staunton W, Kavanagh T (1966) Natural occurrence of the perfect stage of *Gloeosporium concentricum* (Grev.) Berk. and Br. *Irish Journal of Agricultural Research* **5**, 140–141.
- Stotz HU, Mitrousis GK, de Wit PJGM, Fitt BDL (2014) Effector-triggered defence against apoplastic fungal pathogens. *Trends in Plant Science* **19**, 491–500. doi:10.1016/j.tplants.2014.04.009
- Strange RN, Scott PR (2005) Plant disease: a threat to global food security. *Annual Review of Phytopathology* **43**, 83–116. doi:10.1146/annurev.phyto.43.113004.133839
- Teng PS, Shane WW, MacKenzie DR (1984) Crop losses due to plant pathogens. *Critical Reviews in Plant Sciences* **2**, 21–47. doi:10.1080/07352688409382187
- Turgeon BG, Yoder O (2000) Proposed nomenclature for mating type genes of filamentous ascomycetes. *Fungal Genetics and Biology* **31**, 1–5. doi:10.1006/fgbi.2000.1227
- Vegetables New Zealand (2016) Vegetable Brassica IPM manual: Pests, natural enemies, diseases and disorders of vegetable brassicas in New Zealand. Horticulture New Zealand. Available at: www.vegetablesnz.co.nz/research-and-development/current-research-projects/ (accessed 26 February 2017).
- Wafford JD, Gladders P, McPherson GM (1986) The incidence and severity of Brussels sprout diseases and the influence of oilseed rape. *Aspects of Applied Biology* **12**, 1–12.
- Wang X, Wang H, Wang J, Sun R, Wu J, Liu S, Bai Y, Mun J-H, Bancroft I, Cheng F (2011) The genome of the mesopolyploid crop species *Brassica rapa*. *Nature Genetics* **43**, 1035–1039. doi:10.1038/ng.919
- Welham SJ, Turner JA, Gladders P, Fitt BDL, Evans N, Baierl A (2004) Predicting light leaf spot (*Pyrenopeziza brassicae*) risk on winter oilseed rape (*Brassica napus*) in England and Wales, using survey, weather and crop information. *Plant Pathology* **53**, 713–724. doi:10.1111/j.1365-3059.2004.01105.x
- West JS, Atkins SD, Emberlin J, Fitt BDL (2008) PCR to predict risk of airborne disease. *Trends in Microbiology* **16**, 380–387. doi:10.1016/j.tim.2008.05.004
- Woolhouse MEJ, Taylor LH, Haydon DT (2001) Population biology of multihost pathogens. *Science* **292**, 1109–1112. doi:10.1126/science.1059026
- Wu J, Zhao Q, Liu S, Shahid M, Lan L, Cai G, Zhang C, Fan C, Wang Y, Zhou Y (2016) Genome-wide association study identifies new loci for resistance to sclerotinia stem rot in *Brassica napus*. *Frontiers in Plant Science* **7**, 1418. doi:10.3389/fpls.2016.01418
- Yang J, Liu D, Wang X, Ji C, Cheng F, Liu B, Hu Z, Chen S, Pental D, Ju Y (2016) The genome sequence of allopolyploid *Brassica juncea* and analysis of differential homoeolog gene expression influencing selection. *Nature Genetics* **48**, 1225–1232. doi:10.1038/ng.3657
- Yoder OC, Valent B, Chumley F (1986) Genetic nomenclature and practice for plant pathogenic fungi. *Phytopathology* **76**, 383–385. doi:10.1094/Phyto-76-383
- Zhan J, Fitt BDL, Pinnschmidt HO, Oxley SJP, Newton AC (2008) Resistance, epidemiology and sustainable management of *Rhynchosporium secalis* populations on barley. *Plant Pathology* **57**, 1–14.