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Review

Potential Application of Genomic Technologies in Breeding for Fungal and Oomycete Disease Resistance in Pea

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Abstract: Growth and yield of pea crops are severely affected by various fungal diseases, including root rot, Ascochyta blight, powdery mildew, and rust, in different parts of the world. Conventional breeding methods have led to enhancement of host plant resistance against these diseases in adapted cultivars, which is the primary option to minimize the yield losses. To support the breeding programs for marker-assisted selection, several successful attempts have been made to detect the genetic loci associated with disease resistance, based on SSR and SNP markers. In recent years, advances in next-generation sequencing platforms, and resulting improvements in high-throughput and economical genotyping methods, have been used to make rapid progress in identification of these loci. The first reference genome sequence of pea was published in 2019 and provides insights on the distribution and architecture of gene families associated with disease resistance. Furthermore, the genome sequence is a resource for anchoring genetic linkage maps, markers identified in multiple studies, identification of candidate genes, and functional genomics studies. The available pea genomic resources and the potential application of genomic technologies for development of disease-resistant cultivars with improved agronomic profile will be discussed, along with the current status of the arising improved pea germplasm.

Keywords: Ascochyta blight; disease resistance; *Fusarium* spp.; powdery mildew; root rot

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1. Introduction

1.1. Importance of Pea and Global Pea Production Trends

Field pea (*Pisum sativum*) is a temperate-region crop that belongs to the Fabaceae family with other important pulse crops such as chickpea, faba bean, and lentil. Pulse crops have the capacity to fix atmospheric nitrogen and thus reduce global reliance on synthetic fertilizers. Among pulse crops, pea is second only to common bean in terms of area of growth and tons of production. Pea is an important source of protein; carbohydrate; fiber; and micronutrients such as folates, iron, zinc, selenium, and carotenoids that play a significant role in human nutrition [1–11].

In 2018, the global production of dry pea was 13.5 million tons from 7.9 million hectares harvested area (http://faostat.fao.org, accessed on 18 May 2020). Canada was the leading producer of dry pea with an annual production of 3.6 million tons, followed by the Russian Federation (2.3 million tons) and China (1.5 million tons). Out of the 7.9 million hectares, Canada alone accounted for 1.4 million hectares of harvested area (http://faostat.fao.org). In a span of ten years, from 2009 to 2018, the overall production of dry pea increased from 10.4 to 13.5 million tons with an increased harvested area from 6.4 to 7.9 million hectares (http://faostat.fao.org). In recent years, the utilization of pea to produce high value protein and starch fractions and isolates has expanded substantially. These fractions are being used

as ingredients in various food markets including plant-based burgers and beverages in the case of protein, and vermicelli noodles in the case of the starch.

1.2. Production of Pea Is Limited by Fungal Diseases

Diseases are the most important biotic constraints affecting global pea production [12], causing yield losses that range from a small percentage to complete crop loss depending on location and environmental conditions. Notable of these diseases are those caused by foliar and root infecting fungal pathogens, and the oomycete pathogens *Aphanomyces euteiches* and *Peronospora viciae*. The root rot complex caused by *A. euteiches*, *Fusarium solani* f. sp. *pisi* and *F. avenaceum* is the most devastating soil-borne disease(s) of pea. Occurrence of wilt caused by *F. oxysporum* f. sp. *pisi* [13] and white mold caused by *Sclerotinia sclerotiorum* are reported from a few pea growing areas. Several other *Fusarium* spp. that can colonize pea roots are non- to weakly aggressive [14].

Ascochyta blight caused by a complex of three pathogens, *Peyronellaea pinodes* (Berk. and A. Bloxam) Aveskamp, Gruyter, and Verkley (syn. *Didymella pinodes* (Berk. and A. Bloxam) Petr.; *Mycosphaerella pinodes* (Berk. and A. Bloxam) Vestergr.); *Ascochyta pisi* (syn. *Didymella pisi*); and *Peyronellaea pinodella* (syn. *Phoma medicaginis* var. *pinodella*, *Phoma pinodella*) is a major foliar disease of pea. Of these three pathogens, *P. pinodes* is the principal pathogen of Ascochyta blight complex [15]. Davidson et al. [16] identified *Phoma koolunga* sp. nov. as another causal agent of Ascochyta blight in South Australia. Subsequently, *Phoma herbarum* [17] and *Phoma glomerata* [18] have also been identified as associated with the Ascochyta blight complex in Australia. Other important foliar diseases of pea are powdery mildew (*Erysiphe pisi*), downy mildew (*Peronospora viciae* f. sp. *pisi*), rust (*Uromyces* spp.), and gray mold (*Botrytis cinerea*). Cultural practices such as tilling, crop rotation, and fungicide treatment are known to partially control the diseases. The cost benefit ratios and limitations associated with the requirement of repeated application of fungicides limit the use of fungicides in disease management. Host plant resistance (HPR) is the primary option for effective and economical management of fungal diseases of pea, but high levels of HPR are often scarce in cultivated pea germplasm. The development of resistant cultivars is based on the transfer of available levels of HPR from wild or distantly related pea accessions into adapted genetic backgrounds. In a few studies, HPR from multiple sources was transferred to a common genetic background to pyramid the disease resistance. In this context, identification of disease resistance loci accelerates both the identification of resistance sources in diverse germplasm and breeding efforts to introgress or pyramid the identified resistance using marker-assisted selection. This concept is the primary focus of this review article. The identification of disease-resistant quantitative trait loci (QTLs), and the marker-assisted introgression of these QTLs for the development of resistant cultivars, is reviewed. The scope of current genomic resources of pea and the possible application of recent advances in genomic technologies for the accelerated breeding of disease-resistant cultivars are discussed.

2. Challenges/Opportunities in the Development of Resistant Cultivars

Identification of sources of disease resistance is the first step in breeding for development of resistant cultivars. To identify potential sources of resistance, a large number of pea accessions both in the primary gene pool (*Pisum sativum*) and secondary gene pool (*P. sativum* subsp. *elatius* and *P. fulvum)* were screened for reaction to root rot complex, Ascochyta blight, powdery mildew, downy mildew, rust, and white mold.

2.1. Identification of Disease Resistance in Land Races

Partial resistance to root rot complex pathogens *A. euteiches*, *F. solani*, and *F. oxysporum* was identified in several studies. Evaluation of 2500 accessions identified 123 accessions with relatively greater resistance to *A. euteiches* [19]. Twenty of these accessions were identified for varying levels of resistance against five strains of *A. euteiches* [20]. Two accessions, PI 244162 and PI 393487, were ranked for low disease severity against all five

pathogen strains. Four other accessions, PI 210641, PI 269802, PI 411143, and PI 413696 were ranked for low disease severity against four pathogen strains. Partial resistance against *F. solani* f. sp. *pisi* was identified in 44 accessions in a *Pisum* core collection of 387 accessions from USDA, Pullman, WA [21]. The resistance was positively correlated with Aphanomyces root rot resistance. In a different study, pea accessions, PI 125673, 5003, Banner, Carneval, PS 05300234, and Whistler were identified as resistance sources of *F. solani* resistance [22]. In general, genotypes with pigmented-flowers, with exception of PI 180693, had lower disease scores than white-flowered genotypes [22].

Resistance sources of Fusarium wilt and white mold have been identified in pea core collection. McPhee et al. [23] identified that 62 of the 452 accessions representing a world collection of peas were resistant to Fusarium wilt race 2 when artificially inoculated with the pathogen. For white mold, the screening of pea core collection of 504 pea accessions, including 497 accessions from USDA-ARS and Western Regional Plant Introduction Station (WRPIS), Pullman, WA, identified partial resistance of 22 accessions and one woody-stem line [24].

Pea accessions with moderate levels of resistance for Ascochyta blight resistance were identified in studies that involved screening of 2936 [25] and 558 pea accessions [26].

High levels of resistance against powdery mildew were identified in multiple accessions in different studies. Nisar et al. [27] noted high resistance of three genotypes, PS0010128, PS99102238, and Fallon, upon assessment of 177 pea genotypes for reaction to *E. pisi*. Rana et al. [28] evaluated 701 pea accessions that originated from 60 countries for *E. pisi* resistance. Fifty-seven accessions were identified as resistant to powdery mildew under field conditions, whereas 14 accessions showed high resistance against four isolates in laboratory conditions. Based on their results, they recommended the use of resistant accessions IC218988, IC278261, EC598878, EC598655, and EC598704 for pea breeding. Wang et al. [29] identified 101 powdery mildew resistant accessions in a collection of 396 accessions.

Evaluation of 601 pea lines from the John Innes Pisum germplasm collection identified 47 lines with high resistance to *Peronospora pisi* (*P. viciae* f. sp. *pisi*) [30]. Stegmark [31] reported two lines, No. 21 and No. 119, as sources of high levels of resistance and another 'Dark Skin Perfection' cultivar as source of moderate levels of resistance against *P. pisi*. Line No. 119 was also identified for partial resistance against Scandinavian isolates and race 8' from The Netherlands, an isolate that is virulent on many pea genotypes [32]. This study also established the downy mildew resistance of Starcovert, Gastro, Puget, Starnain, and Cobri cultivars. Davidson et al. [33] identified PS998, a promising pea accession from Afghanistan as resistant to 'Kaspa' and 'Parafield' strains, and this accession was used as an important source of downy mildew resistance in pea breeding in Australia.

A panel of 93 pea varieties tested against three isolates of *U. fabae* (syn. *U. viciae-fabae*) Uf-1, Uf-2, and Uf-3 identified high levels of resistance to rust in pea accessions, Century, Tara, Titan, and Yellowhead [34]. These accessions are genetic resources for resistance breeding. Similarly, the evaluation of 345 pea accessions identified slow rusting genotypes, HUP 14, HUDP 16, JPBB 3, FC 1, and Pant P 11, which delay the susceptibility to near maturity of pea crops [35]. Barilli et al. [36] evaluated a panel of 2759 pea accessions of diverse origins and identified IFPI3260, PI347310, PI347321, PI347336, PI347388, and PI347343 as the most resistant accessions. Recently, Das et al. [37] identified KPMR-936, IPF-2014-13, and IPF-2014-16 as genotypes resistant to rust in multiple locations and environmental conditions.

The use of molecular markers facilitates marker-assisted selection in resistant breeding to develop cultivars with improved disease resistance. For example, the USDA-ARS, along with the French National Institute for Agricultural Research (INRA) and North Dakota State University, released eight green pea germplasm lines developed from an F_8 RIL population ('Dark Skin Perfection' × 90-2131) [38]. These lines, when evaluated for *A. euteiches* resistance with isolates, Ae109, and RB84 under controlled conditions and in 12 environments for 4 years at different locations in the USA and France, showed improved

partial resistance to root rot disease. All these lines had good agronomic traits such as white flowers with flowering at the 14th–16th node in 57–61 days, semidwarf plant architecture, straight blunt green pods, clear seed coats, and green cotyledons. These lines are being used by pea breeders in developing either smooth- or wrinkled-seeded cultivars with increased Aphanomyces root rot resistance.

2.2. Identification of Disease Resistance in Wild Pea

The wild peas, including *P. fulvum* and *P. sativum* subsp. *elatius,* were explored as sources of fungal disease resistance, and complete to high levels of resistance against Fusarium root rot, Ascochyta blight, powdery mildew, rust, and white mold were identified (reviewed by Kosterin [39]). A high level of partial resistance to *F. solani* f. sp. *pisi* was identified in eight wild pea accessions [40]. A high level of Ascochyta blight resistance was identified in multiple wild pea accessions [41–44]. Jha et al. [44] identified *P. sativum* subsp. *elatius* accession PI344538 and four *P. fulvum* accessions (PI560061, PI595937, W615017, and P651) as promising sources of resistance. Fondevilla et al. [45] identified six *P. fulvum* accessions as completely resistant to powdery mildew in greenhouse conditions. Barilli et al. [36] identified a high level of resistance to rust in *P. fulvum* accession IFPI3260 both in controlled environmental and field conditions. Five wild pea accessions were identified for high levels of partial resistance to white mold [24]. PI 169603 and PI 240515 were identified for slow disease progression and are recommended as promising accessions for use in resistant breeding [46,47].

The level of disease resistance identified in *P. sativum* accessions often is not sufficient for disease management in natural conditions and against different strains of the pathogens. Wild peas were identified as potential sources of disease resistance that can be used to enhance disease resistance in cultivated pea. However, the use of wild peas is limited by their undesirable agronomic traits and introduced linkage drag when crossed with cultivated pea.

Additionally, wild peas are also known to have reproductive barriers that can result in sterility of the hybrids, thus limiting crossing between diverse species [48]. Based on cytological and genetic analyses, translocation events were observed in crosses that involve *P. fulvum* that can interfere with meiosis and cause sterility of the hybrids [49,50].

In a few studies, interspecific crosses were successfully used to transfer disease resistance to the adapted cultivars [51–55]. Jha et al. [55] observed Ascochyta resistance, a high lodging score (7 to 9 on the 0–9 scale), and low grain yield (100 to 600 kg/ha) in P651 (*P. fulvum*) and used a recombinant inbred line (RIL) population (PR-19) derived from the cross Alfetta × P651 to transfer Ascochyta resistance into the cultivated genetic background. In several other studies, the interspecific segregating populations were used to transfer disease resistance to an adapted genetic background, often using the molecular markers associated with disease resistance. These studies include introgression of resistance to Ascochyta blight [52,53,55], powdery mildew [56], and rust [57,58]. Overall, as wild species are poor in several agronomic traits, emphasis should be made to introgress resistance genes without compromising agronomic performance in which process marker-assisted selection plays a key role.

3. Molecular Markers Associated with Fungal Disease Resistance for Accelerated Breeding

Conventional breeding methods have led to the enhancement of HPR against fungal diseases, root rot, Ascochyta blight, powdery mildew, and rust. The steps involved in conventional breeding, including the requirement of multiple crosses, large progeny population, careful phenotyping at multiple stages to select for disease resistance, and avoidance of undesirable characters, are often challenged by the dynamic production and marketing requirements. Marker-assisted selection (MAS) can assist in the genetic selection of some or most of the traits in early generations and accelerate the breeding process. The early generation of attempts for identification of trait-linked markers are based on several types of molecular markers, including random amplified polymorphic DNA (RAPD), am-

plified fragment length polymorphism (AFLP), simple sequence repeat (SSR), and cleaved amplified polymorphic sequence (CAPS) [52,57,59–68]. Over the last decade, technological advancements in sequencing and genotyping platforms have resulted in development of high throughput and cost-effective methods for single nucleotide polymorphism (SNP) genotyping. SNPs, because of their abundance and distribution across different regions of chromosomes, provided a dense coverage of the genome for high-resolution mapping of disease resistance in pea [55,66,67,69–76]. Since the development of genotyping-by-sequencing (GBS) protocol by Elshire et al. [77], GBS has been used widely for genotyping of pea mapping populations for GWAS and QTL analysis of various diseases [8,9,75]. SNP arrays have been developed based on the identification of genome-wide SNPs from genome and/or transcriptome sequencing of a set of diverse pea lines. Sindhu et al. [54] developed a 1536 Illumina GoldenGate assay and used the technology for genotyping of five RIL populations and developing a consensus pea map. Tayeh et al. [78] developed a 13.2K SNP array, Genopea, and used the array for genotyping of multiple mapping populations. Recently, a 90K Axiom SNP array was developed and used for genotyping of a diversity set of peas [79].

In addition to SNPs, a few other types of markers based on next generation sequencing or array hybridization were also used for high throughput genotyping of pea. For example, Diversity Arrays Technology (DArT) combined with NGS (DArTseq™) was identified as a high-throughput genotyping method that can generate a greater number of markers at relatively low cost [80]. Identification of disease resistance markers based on genotyping of various mapping populations, both bi-parental and diverse collections, is summarized below.

3.1. Root Rot

Inheritance studies suggested the quantitative nature of genetic resistance to root rot in pea caused by *A. euteiches* and *F. solani* with the importance of additive gene action [81,82]. Various studies have been conducted to identify QTLs associated with resistance to root rot complex in pea (Table 1). Using a RIL population derived from a cross between Puget (susceptible) and 90-2079 (partially resistant), Pilet-Nayel et al. [59] identified seven QTLs for root rot resistance. The RILs were evaluated for *A. euteiches* resistance under field conditions at Pullman, Washington and LeSueur, Minnesota in the United States and genotyped based on RAPDs, AFLPs, SSRs, ISSRs, STSs, isozymes, and genes for morphological traits. One major QTL from this study, *Aph1* on LG IVb was stable across the locations and years, and it contributed up to 47% of the phenotypic variance. QTL, *Aph2* on LG V mapped near the *r* (wrinkled/round seeds) gene and explained up to 32% of the variation, whereas QTL *Aph3* on LG Ia mapped close to the *af* (normal/afila leaves) gene and explained 11% of the variation. In a subsequent study, Pilet-Nayel et al. [60] validated these QTLs in the same mapping population against two other pathogen isolates, SP7 (United States) and Ae106 (France), in a controlled greenhouse environment.

Table 1. Genomic regions and/or markers associated with resistance to root rot, Ascochyta blight complex *, powdery mildew, rust, and white mold in pea.

Disease and Pathogen	Plant Material	Genomic Region/Marker	Major QTL	Flanking Markers of Major QTL	Reference
Root rot					
Aphanomyces euteiches	Puget × 90-2079	7 QTLs	LG IVb (*Aph1*)	E7M4.251, N14.950, U326.190	[59]
Aphanomyces euteiches	Puget × 90-2079	10 QTLs	LG IVb (*Aph1*)	U326.190, E7M4.251	[60]
Aphanomyces euteiches	Baccara × PI180693	75 additive-effect QTLs	LG III (*Ae-Ps3.1*)	X03_1000	[83]
	Baccara × 552	60 additive-effect QTLs	LG VII (*Ae-Ps7.6b*)	AA176	
Aphanomyces euteiches	Puget × 90-2079	27 meta-QTLs,	LG VII (*MQTL-Ae25*)	IJB174	[84]
	Baccara × PI180693	318 candidate genes	LG VII (*MQTL-Ae26*)	AB122b	
	Baccara × 552				
	Dark Skin Perfection × 90-2131				
Aphanomyces euteiches	175 *Pisum sativum* lines	52 QTLs	LG IV (*Ae-Ps4.4-4.5*)	AA122	[74]
	(referred as the "pea-Aphanomyces		LG VII (*Ae-Ps7.6*)	AA387, AB101	
	collection")				
Aphanomyces euteiches	266 pea collection	11 genomic intervals	LG VII (*Ae-Ps7.6*)	Ps115429	[85]
Fusarium solani	JI 1794 × Slow	4 QTLs	LG IV (1)	Not reported	[86]
Fusarium solani	CMG × PI220174	3 QTLs	LG II (1)	Not reported	[87]
Fusarium solani	Carman × Reward	1 QTL	LG VII (1)	AA416, AB60	[88]
Fusarium solani	Dark Skin Perfection × 90-2131	5 QTLs	LG IIa (*Fsp-Ps2.1*)	Thiol (gene-based marker)	[71]
Fusarium solani	Baccara × PI180693	3 QTLs	LG IIa (*Fsp-Ps2.1*)	Ps900203	[72]
Fusarium avenaceum	Carman × Reward	1 QTL	Chromosome VII	AA160, AD53	[89]
Ascochyta blight complex					
Peyronellaea pinodes	A88 × Rovar	13 QTLs	LG 1 (*Asc1.1*)	c206	[90]
(formerly *Mycosphaerella*			LG I1 (*Asc2.1*)	sP2P5	
pinodes)			LG II1 (*Asc3.1*)	PI39	
Peyronellaea pinodes	A26 × Rovar	11 QTLs	LG I1 (*Asc2.1*)	sM2P5	[91]
	A88 × Rovar	14 QTLs	LG 1 (*Asc1.1*)	c206	
			LG II1 (*Asc3.1*)	PI39	
Peyronellaea pinodes	Carneval × MP1401	3 QTLs	LG IV (1)	cccc1 (Locus with the max. LOD)	[61]
Peyronellaea pinodes	JI296 × DP	10 QTLs	LG III (mpIII-1)	E08-980	[62]
			LG VI (mpVI-1)	G04-950	
Peyronellaea pinodes	P665 × Messire	6 QTLs	LG III (MpIII.3)	$OPAI14_{1353}/OPW2_{1157}$	[52]
			LG III (MpIII.1)	$OPM6_{598}/OPW5_{387}$	

Table 1. *Cont.*

Disease and Pathogen	Plant Material	Genomic Region/Marker	Major QTL	Flanking Markers of Major QTL	Reference
Peyronellaea pinodes	P665 × Messire	14 QTLs	LG III (MpIII.3_DRl_06) LG III (MpIII.3_DS_06) LG III (MpIII.3_DRst_06)	OPAI14_1353/AA175 OPAI14_1353/AA175 OPAI14_1273/OPAI14_1353	[53]
Peyronellaea pinodes	54 pea accessions (a subset of 169 accessions)	2 SNPs	LG III LG VII	PsDof1p308 RGA-G3Ap103	[92]
Peyronellaea pinodes	Alfetta × P651	9 QTLs	LG III (abIII-1) LG I-IV (abI-IV-2)	PsC8780p118, PsC22609p103, PsC8031p219	[55]
Peyronellaea pinodes	HIF-224 from PR-19-224 (Alfetta × P651)	2 QTLs	LG I-IV (abI-IV-2.1)	Sc1762_271077, PsC943p541	[75]
Powdery mildew					
Erysiphe pisi	Almota × 88V1.11 JI1794 × Slow	1 RADP marker linked to *er1*, 1 SCAR marker	LG VI	$PD10_{650}$, $PD10_{650}$ SCAR	[93]
Erysiphe pisi	Highlight × Radley	3 RAPD markers linked to *er1*	LG VI	OPO-181200, OPE-161600, OPL-61900	[94]
Erysiphe pisi	Seven pairs of near-isogenic lines (NILs)	1 SCAR linked to *er1*	LG VI	ScOPX 04880	[95]
Erysiphe pisi	Lincoln × JI2480	3 SSR and 2 RAPD markers linked to *er2*, 1 SCAR	LG III	RAPD: OPX17_1400, SCAR: ScX17_1400	[63]
Erysiphe pisi	C2 × Messire	6 RAPD markers linked to *Er3*, 2 SCAR markers	LG IV	OPW04_637, OPC04_640, OPF14_1103, OPAH06_539, OPAB01_874, OPAG05_1240, SCW4637, SCAB1874	[56]
Erysiphe pisi	ROI3/02 × Progress9	CAPS marker for *er1-5*	LG VI	GIM-300/SmlI	[96]
Erysiphe pisi	ROI3/02 × Sprinter ROI3/02 × Progress9	CAPS makers for *er1-1* and *er1-4* STS marker for *er1-2* dCAPS marker for *er1-3* HRM marker for *er1-5*	LG VI	er1-1/AsuHPI-B er1-4/AgsI er1-2/MGB er1-3/XbaI er1-5/HRM54	[97]
Erysiphe pisi	G0001778 × Bawan 6	SNP marker for *er1-6* allele	LG VI	SNP1121	[67]
Erysiphe pisi	Bawan 6 × DDR-11	InDel marker for *er1-7* allele	LG VI	InDel111–120	[68]

Table 1. *Cont.*

Disease and Pathogen	Plant Material	Genomic Region/Marker	Major QTL	Flanking Markers of Major QTL	Reference
Erysiphe pisi	256 pea accessions panel	8 KASPar markers for *er1* alleles, *er1-1* *er1-3* *er1-4* *er1-5* *er1-6* *er1-7* *er1-10* *er1-11*	LG VI	KASPar-*er1-1* KASPar-*er1-3* KASPar-*er1-4* KASPar-*er1-5* KASPar-*er1-6* KASPar-*er1-7* KASPar-*er1-10* KASPar-*er1-11*	[98]
Erysiphe pisi	WSU 28 × G0004389 Bawan 6 × G0004400	InDel marker for *er1-8* KASPar markers for alleles, *er1-8* and *er1-9*	LG VI	InDel-er1-8 KASPar-er1-8, KASPar-er1-9	[76]
Rust					
Uromyces fabae	HUVP 1 (HUVP 1 × FC 1)	2 RAPD markers flanking *Ruf* gene	-	*SC10-82360* *SCRI-711000*	[99]
Uromyces fabae	HUVP 1 × FC 1	2 QTLs	LG VII (*Qruf*)	AA505, AA446	[64]
Uromyces fabae	HUVP 1 × FC 1	4 QTLs	LG VII (*Qruf*) LG I (*Qruf2*)	AA505, AA446 AA121, AD147	[100]
Uromyces pisi	IFPI3260 × IFPI3251	1 QTL	LG III (*Up1*)	OPY111316 OPV171078	[57]
Uromyces pisi	IFPI3260 × IFPI3251	3 QTLs	LG II (*UpDSII*) LG IV (*UpDSIV*)	3567800, AD280 3563695, 3569323	[58]
White mold					
Sclerotinia sclerotiorum	282 pea accessions 266 pea accessions	206 SNPs (lesion resistance) 118 SNPs (nodal resistance)	-	-	[101]
Sclerotinia sclerotiorum	Lifter × PI240515 PI169603 × Medora	7 QTLs 6 QTLs	LG III LG III	Chr5LG3_562563492 Chr5LG3_568430003 Chr5LG3_568430003 Chr5LG3_569648908	[102]

* Markers associated with the Ascochyta blight complex were either identified based on resistance to *Peyronellaea pinodes* or field evaluation of Ascochyta blight resistance. In the latter case, other pathogens of the disease complex were likely also present.

Hamon et al. [83] evaluated two mapping populations developed from crosses between Baccara (susceptible) and two new partially resistant accessions (PI 180693 and 552) for resistance to *A. euteiches* in controlled conditions, at three locations each in France and USA under field conditions. Overall, 135 additive-effect QTLs, corresponding to 23 genomic regions, and 13 significant epistatic interactions for *A. euteiches* resistance were observed in the two RIL populations. Five out of 23 additive-effect genomic regions were consistent in both RIL populations when tested with two *A. euteiches* strains, RB84 and Ae109, and in locations of both USA and France and contrasting growth conditions (controlled and field). Of these five consistent QTLs (*Ae-Ps1.2*, *Ae-Ps2.2*, *Ae-Ps3.1*, *Ae-Ps4.1*, and *Ae-Ps7.6*), *Ae-Ps1.2* on LG I was co-located with QTL *Aph3* from Pilet-Nayel et al. [59].

Hamon et al. [84] conducted a QTL meta-analysis to identify QTLs for Aphanomyces root rot resistance and associated morphological or phenological traits. The meta-analysis is based on 244 QTLs identified in four populations, Puget × 90-2079, Baccara × PI180693, Baccara × 552, and DSP × 90-2131. Twenty-seven meta-QTLs for resistance to *A. euteiches* that include 11 consistent meta-QTLs and seven highly consistent genomic regions were identified. Seven meta-QTLs, which include six consistent regions, *MQTL-Ae1* (LG I), *MQTL-Ae3* (LG I), *MQTL-Ae6* (LG II), *MQTL-Ae8* (LG III), *MQTL-Ae15* (LG IV), and *MQTL-Ae27* (LG VII), were co-located with six meta-QTLs on respective linkage groups: *MQTL-Morpho1* (LG I), *MQTL-Morpho2* (LG I), *MQTL-Morpho3* (LG II), *MQTL-Morpho4* (LG III), *MQTL-Morpho5* (LG IV), and *MQTL-Morpho7* (LG VII), associated with plant height, earliness, and/or three morphological genes, *Af* (normal leaves), *A* (colored flowers), and *R* (round seeds). However, alleles for resistance were linked with undesirable alleles for breeding such as normal leaves (*Af*), colored flowers (*A*), long vines, and late flowering. Due to the co-linearity between *Medicago truncatula* and pea genome sequences, inspection of the six main meta-QTL regions in the *M. truncatula* genome identified 318 candidate genes with 14 to 91 candidates per meta-QTL. These genes had known function involved in important cellular processes and disease resistance in plants, including protein kinases, pathogenesis-related-proteins, heat-shock proteins, transcription factors, and resistance gene analogs. This study identified several consistent genomic regions that can play an important role in MAS in pea breeding.

QTLs for partial resistance to Aphanomyces root rot were validated in near-isogenic lines (NILs) developed by marker-assisted backcrossing [103]. For NILs development, resistance alleles at seven QTLs associated with resistance to *A. euteiches* [59,60,83,84] were transferred to three genetic backgrounds, including spring (Eden) and winter (Isard) pea cultivars. Under controlled conditions, these NILs were evaluated against two strains of *A. euteiches*, RB84 (pathotype I) and Ae109 (pathotype III). Resistance alleles at the major-effect QTLs (*Ae-Ps4.5* and *Ae-Ps7.6*) and minor QTLs (*Ae-Ps2.2* and *Ae-Ps5.1*) were validated in NILs for either individual or combined effects on resistance. The severity of disease was less in NILs with resistance alleles at major QTLs (*Ae-Ps4.5* and *Ae-Ps7.6*) compared to lines without these alleles. Further, *Ae-Ps7.6* showed significant QTL × genetic background interactions with increased effect in Isard. In this study, previously identified QTLs for *A. euteiches* resistance [59,60,83,84] were validated. In a subsequent study, NILs carrying resistance alleles at the seven QTLs were evaluated for early steps of the pathogen life cycle, including root colonization and appearance of first disease symptoms [104]. Resistance alleles at minor-effect QTLs (*Ae-Ps2.2*, *Ae-Ps3.1*, and *Ae-Ps5.1*) significantly reduced root colonization, whereas combining resistance alleles at QTLs that include major-effect QTL *Ae-Ps7.6* (*Ae-Ps5.1*/*Ae-Ps7.6* or *Ae-Ps2.2*/*Ae-Ps3.1*/*Ae-Ps7.6*) had decreased root colonization by *A. euteiches* and delayed appearance of disease symptoms. NILs carrying QTL *Ae-Ps7.6* were also identified for varying levels of resistance against 43 *A. euteiches* isolates from different French pea growing areas [105].

A genome-wide association study (GWAS) was conducted using 175 pea lines evaluated in field infested nurseries in nine environments and for response to inoculation by two isolates of *A. euteiches* in controlled conditions [74]. This study identified 52 QTLs for *A. euteiches* resistance and validated six out of seven previously identified QTLs [83,84].

This study also identified marker haplotypes at 14 consistent QTLs for disease resistance. In a later study, in 266 pea lines using the GWAS approach, Desgroux et al. [85] reported an association of 75 and 11 genomic intervals with plant architecture and *A. euteiches* resistance, respectively. In this study, several QTLs identified in previous studies [74,83,84] were confirmed, and these QTLs were associated with plant architecture. Further, one SNP marker, Ps115429 mapped at *Ae-Ps7.6* (major QTL), showed significant association with root system architecture (total root projected area) as well as disease resistance. For the Ps115429 marker, the allele for increased *A. euteiches* resistance was correlated with an increased total root projected area.

For *F. solani* f. sp. *pisi* (Fsp) resistance, four QTLs were identified on LGs III, IV, and VI in an interspecific RIL population developed from JI 1794 (*P. sativum* ssp. *elatius* var. *pumilo*) and Slow (*P. sativum* ssp. *sativum*) [86]. Two QTLs on LG III were located close to *M* (brown mottle on testa) and *Le* (tall stature), and one QTL each on LG IV (proximal to the ribosomal array) and LG VI (distal to Gty, gritty seed coat). Weeden and Porter [87] detected three QTLs for Fsp resistance in a RIL population derived from CMG × PI 220174 on LGs II, IV, and VII. The QTL on LG II was associated with the greatest resistance to Fsp compared to the other two QTLs, and it overlapped with *A* (pigmented flower). Similarly, one QTL was identified for Fsp resistance on linkage group VII in 71 lines of RIL population developed from Carman (resistant cultivar) and Reward (susceptible cultivar) [88]. This QTL explained 39% of the phenotypic variance. Microsatellite markers AA416 and AB60 flanking this QTL can be utilized for MAS.

Five QTLs were reported for Fsp resistance in a pea RIL population of 111 lines developed from a cross between susceptible 'Dark Skin Perfection' (DSP) (W6 17516) and the resistant line 90-2131 (PI 557501) [71]. The major QTL *Fsp-Ps2.1* was identified in all three years of testing, and it explained 22.1–72.2% of the phenotypic variance. In this study, *Fsp-Ps7.1* was detected in only one year; however, this QTL was in the same region on LG VII where Feng et al. [88] observed a QTL for Fsp in two years field trial in the same RIL population. Similarly, QTLs *Fsp-Ps3.1* and *Fsp-Ps4.1*, detected from single growing season, were present on LG III and LG IV, respectively, where Hance et al. [86] also detected QTLs for Fsp resistance in *P. sativum* ssp. *elatius*. Among them, *Fsp-Ps3.1* located near *Le* was the same QTL detected previously by Hance et al. [86]. Near to this QTL, three genes of defensins gene family, pI39 and DRR230-A, and DRR230-B were mapped in this study. Among these genes, *pI39* was mapped on LG III in the region where QTL (Asc3.1) for Ascochyta blight resistance [91] was mapped, and this locus was adjacent to QTL mpIII-4 for Ascochyta blight resistance [106]. Further, Fondevilla et al. [107] reported differential expression of *pI39* in contrasting pea genotypes infected with *M. pinodes*. These observations suggest the important role of *pI39* gene for conferring resistance against fungal pathogens. Four QTLs for resistance to Fsp—*Fsp-Ps2.1*, *Fsp-Ps4.1*, *Fsp-Ps6.1*, *Fsp-Ps7.1*—identified in this study were co-localized with QTLs for resistance to *A. euteiches*, *Ae-Ps2.1*, *Ae-Ps4.5*, *Ae-Ps6.1*, and *Ae-Ps7.6* detected by Hamon et al. [84], respectively. For both studies, the same mapping population (DSP × 90-2131) was used.

In a later study, three QTLs were identified for Fsp resistance in 178 lines of RIL population developed from 'Baccara' (susceptible) and PI 180693 (partially resistant) under greenhouse conditions [72]. PI 180693 contributed alleles for disease resistance. The major QTL (*Fsp-Ps2.1*) explained 44.4–53.4% of the phenotypic variance, whereas the other two QTLs (*Fsp-Ps3.2* and *Fsp-Ps3.3*) explained 3.6–4.6% of the variance. This QTL was previously identified in DSP × 90-2131 mapping population under field conditions [71]. Thus, *Fsp-Ps2.1* was confirmed in two different RIL populations and two separate types of growing conditions (field and greenhouse). Further, it was co-located with QTL (*Ae-Ps2.1*) identified by Hamon et al. [84] for *A. euteiches* resistance. These SNPs associated with QTLs could be utilized as markers for MAS in pea breeding for Fusarium as well as Aphanomyces root rot resistance.

For *F. avenaceum* root rot resistance, one QTL was identified on chromosome VII in the pea RIL population (Carman × Reward), and this QTL explained 21.7% of the

phenotypic variance [89]. Feng et al. [88] and Li et al. [89] used the same RIL population for QTL detection, and the markers flanking their QTLs were also the same for Fsp and *F. avenaceum*, respectively. Further, the flanking marker AA416 associated with the QTL for *F. avenaceum* resistance [89] was also associated with one QTL for *A. euteiches* reported by Hamon et al. [83]. These results suggest that one QTL is associated with resistance to three root rot pathogens: *F. avenaceum*, *F. solani* and *A. euteiches*.

3.2. Ascochyta Blight

Several studies have suggested the quantitative nature of disease resistance with dominance and additive effects (Table 1) [51,53,62,108,109]. Timmerman-Vaughan et al. [90,91] detected many QTLs for resistance to Ascochyta blight on LGs I, II, III, IV, V, VII, and Group A in two populations (A26 × Rovar and A88 × Rovar) under field conditions. Individually, these QTLs contributed 5.7% to 21.2% of the phenotypic variation. Tar'an et al. [61] found three QTLs on LGs II, IV, and VI in a pea RIL population (Carneval and MP1401), and these QTLs explained 5 to 17% of the phenotypic variation. In a population developed from JI296 and DP, Prioul et al. [62] detected 10 QTLs on LGs II, III, Va, and VII, and six QTLs on LGs III, Va, VI, and VII under field and controlled conditions, respectively. These QTLs explained 6 to 42% (field condition) and 5 to 20% (controlled condition) of the phenotypic variance. In an inter-specific population derived from P665 (*P. sativum* ssp. *syriacum*) and Messire (*P. sativum*), six QTLs were located on LGs II, III, IV, and V, and collectively these QTLs contributed from 31 to 75% of the phenotypic variation [52]. In this population, three additional QTLs were detected for Ascochyta blight on LGs III and VI after adding SSR markers to the previous linkage map [53], whereas four new QTLs were observed on LGs II, III, and V for cellular processes related to disease resistance. Based on common SSR markers, a comparative study indicated that QTLs MpIII.1, MpIII.3, and MpIII.2 reported by Fondevilla et al. [53] in *P. sativum* ssp. *syriacum* corresponded to mpIII-1, mpIII-3, and mpIII-5 detected by Prioul et al. [62] in *P. sativum*.

Using an inter-specific RIL population, PR-19 (*P. fulvum* × *P. sativum*), Jha et al. [55] identified nine QTLs for Ascochyta blight resistance on LGs I-IV (5), LG III (2), and VII (2), based on a linkage map generated by genotyping using a 1536 Illumina GoldenGate array [54]. Out of nine QTLs, six and three were detected under field and greenhouse conditions, respectively, and these QTLs explained 7.5% to 28% of the variation individually. QTLs, abI-IV-2 and abIII-1 were consistent across station-years and were used to develop a heterogeneous inbred family (HIF) population [55,75]. Two new QTLs—abI-IV-2.1 and abI-IV-2.2—were reported within the QTL region of abI-IV-2 by fine mapping in the HIF-224 population generated from PR-19-224 line [75]. For fine mapping, 51 PR-19 RILs were genotyped by GBS that resulted in the identification of an additional eight SNP markers within the abI-IV-2 QTL region. Based on common anchored markers used for a consensus pea map prepared by Sindhu et al. [54] from five RIL populations including PR-19, the locations of QTLs identified by Jha et al. [55,75] were compared with QTLs from previous studies [52,53,62,110]. Results showed that none of the QTLs reported by Jha et al. [55,75] was in the region where previous researchers located QTLs for Ascochyta blight resistance.

Several studies reported the co-localization of Ascochyta blight QTLs with candidate genes related to disease resistance, including a pea defensin (*DRR230-b*), resistance gene analogs (*RGAs*), and a transcription factor (*PsDof1*) [90,106,111]. On LG VII, Timmerman-Vaughan et al. [90] reported co-localization between *RGAs*, *RGA-G3A*, *RGA2.97*, and *RGA1.1*, and QTLs for Ascochyta blight resistance, whereas Prioul-Gervais et al. [106] observed co-localization of *RGAs* (*IJB174*, *IJB91*, *RGA-G3A*, *RGA2*, and *RGA3*) with mpVII-1 QTL for Ascochyta blight reported previously by Prioul et al. [62]. Similarly, on LG III, Prioul-Gervais et al. [106] reported co-localization between candidate genes *DRR230-b* and *PsDof1* with QTLs mpIII-1 and mpIII-4 reported by Prioul et al. [62]. Timmerman-Vaughan et al. [111] mapped 14 candidate genes in two populations (A88 × Rovar and A26 × Rovar). Eight of these genes were linked to Ascochyta blight resistance and showed resemblance to *Arabidopsis thaliana* genes associated with defense mechanisms, including jasmonic acid

and ethylene signalling, and a member of the defensin family. Seven candidate genes were mapped in proximity to Ascochyta blight QTLs: 2700574 and 2698120 (*Asc2.1*), 2702337 (*Asc3.2*), 2681734 (*Asc4.2*), *Asc4.3* (2694005), and 2676364 and 2680014 (*Asc7.1*). Further, two candidate genes—2676364 and 2693718—were linked with epistatic interactions.

In P665 (*P. sativum* ssp. *syriacum*), differentially expressed genes related to defense responses against the pea pathogen *P. pinodes* were identified using microarray [107] and deepSuperSAGE transcription profiling [112]. Expressed genes could play a significant role in disease resistance since these were associated with various transcription factors, metabolic and signaling pathways, and components of biotic as well as abiotic stresses [112]. In the same pea line (P665), Carrillo et al. [110] reported several candidate genes involved in cellular responses for resistance to Ascochyta blight. In a subsequent study in an interspecific population (P665 × Messire), Fondevilla et al. [113] mapped ten candidate genes that were expressed in P665 (resistant accession) and were present in regions that control disease resistance. They observed a significant association of five candidate genes, *RGA1.1* (putative NBS-LRR type disease resistance protein), *DRR49a* (*P. sativum* disease resistance response protein PR10), *JERF1* (putative ethylene responsive transcription factor), *Blec4* (lectin), and *EREBP* (putative ethylene responsive transcription factor), with disease resistance traits that included disease ratings on leaves and stems, and percentage of germinated spores that can cause cross-linking of protein and death of epidermal cells. In a recent study, Castillejo et al. [114] detected putative peptides for Ascochyta blight resistance using a targeted proteomics method, and these protein-based markers can be of potential use in pea breeding.

Jha et al. [92] reported association of SNPs, PsDof1p308, and RGA-G3Ap103 that is located within *PsDof1* and *RGA-G3A* candidate genes with Ascochyta blight scores. To validate previously identified markers for Ascochyta blight resistance, Jha et al. [115] tested 10 SNP markers in 36 pea cultivars in a Saskatchewan pea regional variety trial. Results indicated the usefulness of markers RGA-G3Ap103 and PsC8780p118 in future pea breeding. QTLs and/or SNP markers detected for Ascochyta blight resistance from various studies can be useful in MAS to develop cultivars with improved disease resistance.

3.3. Rust

Vijayalakshmi et al. [99] identified two RAPD markers—*SC10-82*360 and *SCRI-71*1000—in pea for resistance to *U. fabae* in the flanking region of a single partially dominant gene, designated as *Ruf*, which controls non-hypersensitive rust resistance (Table 1). Similarly, for *U. fabae* resistance, two QTLs—Qruf (major QTL) and Qruf1 (minor QTL)—were identified on LG VII in 136 RILs of pea mapping population (HUVP 1 × FC 1) under field and controlled conditions [64]. Qruf was detected in all the tested conditions and explained 23.5–58.8% of the phenotypic variation for area under the disease progress curve. Thus, flanking SSR markers (AA446 and AA505) at this QTL could be important in breeding. In a subsequent study, using the same population, Rai et al. [100] identified four QTLs, including two previously identified QTLs for slow rusting components (leaf area covered by sporulating pustules, number of aecial pustules per leaf, and number of aecial cups per leaf) and percent disease severity. Two new QTLs (Qruf2 and Qruf3) were present on LG I and LG VI, respectively. Qruf2 (major QTL) was key in determining the disease resistance, and it explained 21.3 to 29.6% of the total phenotypic variation. Further, comparisons were made between LG VII of Loridon et al. [116] with QTLs reported for rust resistance [64], Fsp [83,88], and *A. euteiches* resistance [83]. Results indicated that Qruf for rust resistance was co-localized with *Ae-Ps7.5* (QTL for *A. euteiches* resistance), whereas the other QTL (Qruf1) was co-located with resistance to *A. euteiches* (*Ae-Ps7.2*, *Ae-Ps7.3*, and *Ae-Ps7.4*) and *F. solani*. These genomic regions could be targeted in resistance breeding as these provide resistance to multiple pathogens. In a separate study, Singh et al. [117] reported the effectiveness of flanking SSR markers of QTLs Qruf (AA446 and AA505) and Qruf1 (AD416 and AA146) by validating these markers in 30 pea genotypes. Based on the results, they found the usefulness of SSR markers in MAS in improving resistance to pea rust.

For *U. pisi* resistance, Barilli et al. [57] detected one major QTL on LG 3 in the F_2 population of wild pea—IFPI3260 (*P. fulvum*) × IFPI3251 (*P. fulvum*)—and this QTL explained 63% of the phenotypic variance. They recommended the use of flanking RAPD markers OPY111316 and OPV171078 in MAS after converting them to sequence characterized amplified region (SCAR) markers. Further, in this population, three QTLs were detected for rust disease severity on LGs II (UpDSII) and IV (UpDSIV and UpDSIV.2) under controlled conditions using DArT-derived markers [58]. Individually, these QTLs contributed 14 to 29.2% of the total phenotypic variation. The comparisons of map used in this study with linkage maps of *P. sativum* from previous studies [110,116,118] indicated co-localization of UpDSII and UpDSIV with QTLs for Ascochyta blight resistance, MpV.2 and MpII.1 [110], and Aphanomyces root rot resistance, *Ae-Ps*5.2 and *Ae-Ps*2.2 [83], respectively. Thus, QTL regions UpDSII and UpDSIV could be utilized in MAS as these regions also contain regions for resistance to several diseases.

3.4. Powdery Mildew

Powdery mildew resistance in pea is controlled by two recessive genes, *er1* and *er2*, and one dominant gene *Er3*. Vaid and Tyagi [119] reported that a single recessive gene controls powdery mildew resistance and this gene provides resistance in eight cultivars of different origins against five *E. pisi* isolates. In a separate study, Tiwari et al. [120] showed the involvement of *er1*, a single recessive gene, for powdery mildew resistance in eight pea cultivars and lines, whereas a different recessive gene, *er2*, provided resistance in JI 2480. This study also indicated durable resistance of *er1* in comparison to *er2* (non-durable). Complete resistance was provided by *er1* with no visible sign of necrosis under controlled conditions and high resistance under field conditions, whereas the resistance of *er2* gene is dependent on temperature and leaf age [121]. These two genes also differ in their mode of action. Resistance of *er1* was due to avoidance of epidermal cell penetration, and *er2* was related to cell death after penetration accompanied by reduced penetration. Thus, *er1* has been widely used by breeders to provide powdery mildew resistance in pea [120,122]. The involvement of the dominant gene *Er3*, which segregated independently from *er1* and *er2*, was reported for the first time in wild pea, *P. fulvum* [45,122]. This gene provided complete resistance under controlled as well as field conditions, and therefore this gene could be a good source of powdery mildew resistance in pea breeding [45,122]. The resistance genes *er1*, *er2*, and *Er3* were located on LG VI [93], LG III [63], and LG IV [123], respectively.

Several markers linked to these genes were identified by linkage mapping analysis. Timmerman et al. [93] identified RADP, RFLP, and morphological marker Gty closely linked to *er1* by segregation analysis of two F_2 populations (Almota × 88V1.11 and JI1794 × Slow) (Table 1). A closely linked RAPD marker ($PD10_{650}$) was converted to a SCAR marker, $PD10_{650}$ SCAR, which can be used in MAS. Similarly, Tiwari et al. [94] reported three RAPD markers—OPO-181200, OPE-161600, and OPL-61900—linked to *er1*, whereas Srivastava et al. [95] identified a RAPD marker OPX 04880 linked to *er1* and developed a SCAR marker, ScOPX 04_{880} for MAS.

Katoch et al. [63] identified SSR and RAPD markers linked to *er2* in an F_2 population of Lincoln × JI2480. They also reported SCAR marker (ScX17_1400) generated from closely linked RAPD marker (OPX-17_1400 for MAS). Six RAPD markers tightly linked to the dominant gene *Er3* were identified in a F_2 segregating population generated by crossing breeding line (C2) containing *Er3* gene with cultivar Messire [56]. Further, SCAR markers SCAB1874 and SCW4637, generated from RAPD markers OPAB01_874 and OPW04_637, respectively, were validated in diverse genetic backgrounds.

Humphry et al. [124] reported that powdery mildew resistance by *er1* is due to loss-of-function mutations in *PsMLO1*, a member of *MLO* (M*ildew Resistance* L*ocus* O) gene family. So far, eleven *er1* alleles (*er1-1* to *er1-11*) associated with powdery mildew resistance have been identified and characterized in pea, and each of these alleles corresponds to different mutations in the *PsMLO1* coding sequence [65–68,76,96–98,124–127]. For *er1* alleles (*er1-1* to *er1-11*), functional markers, including sequence-tagged site (STS), high-

resolution melting (HRM), cleaved amplified polymorphic sequence (CAPS), and derived CAPS (dCAPS), were developed that can be used in MAS for developing powdery mildew resistant cultivars [65–68,76,96,97,126]. For example, Pavan et al. [96] identified the co-dominant CAPS marker GIM-300/SmlI for er1-5. In a subsequent study, Pavan et al. [97] reported CAPS makers for *er1-1* and *er1-4* (er1-1/AsuHPI-B and er1-4/AgsI), STS marker for *er1-2* (er1-2/MGB), dCAPS marker for *er1-3* (er1-3/XbaI), and HRM marker for *er1-5* (er1-5/HRM54). Sun et al. [67] developed a functional SNP marker—SNP1121—for novel allele er1-6 using HRM technique, whereas Sun et al. [68] reported a co-dominant functional insertion/deletion (InDel) marker—InDel111–120—for the novel *er1-7* allele. Similarly, co-dominant functional InDel and the Kompetitive allele-specific PCR (KASPar) markers were developed for novel alleles *er1-8* (InDel-er1-8 and KASPar-er1-8) and *er1-9* (KASPar-er1-9) [76]. The functional markers SNP1121, InDel111-120, InDel-er1-8, KASPar-er1-8, and KASPar-er1-9 were validated in diverse pea germplasms and therefore could be useful in MAS in pea breeding for developing powdery mildew resistance cultivars [67,68,76]. Ma et al. [98] developed eight KASPar markers for *er1* alleles, *er1-1*, *er1-3*, *er1-4*, *er1-5*, *er1-6*, *er1-7*, *er1-10*, and *er1-11* and validated these breeder-friendly markers in the USDA pea collection.

3.5. White Mold

GWAS and RNA-Seq studies were conducted to explore and compare lesion (stem lesion size restriction) and nodal (infection is restricted to an internode region) resistance in pea against white mold [101]. Using an association study, 206 SNPs were significantly associated with lesion resistance, whereas 118 SNPs were associated with nodal resistance (Table 1). Based on GWAS and RNA-Seq results, different redox-related transcripts were reported for nodal and lesion resistance, except for a transcript that encodes glutathione S-transferase (GST). GST plays an important role in balancing redox homoeostasis in a cell. SNP TP13557 was identified in the lesion resistance, as well as nodal resistance, using GWAS and was located within a transcript which encodes GST. Thus, GST could play a key role in nodal and lesion resistance to white mold in pea, and SNP TP13557 can be utilized in resistance breeding. In a recent study, Mahini et al. [102] identified 13 QTLs associated with white mold resistance in two RIL populations, PRIL17 (Lifter × PI240515) and PRIL19 (PI169603 × Medora). These QTLs explained a phenotypic variance of 5.1 to 64.3%.

4. Genomic Resources for Disease Resistance Breeding

4.1. Pea Genome Sequence

Until recently, pea had relatively few genomic resources compared to most other crop species. Kreplak et al. [128] recently reported the first annotated chromosome-level reference genome assembly for pea, which boosted the genomics-assisted breeding of pea. The reported genome assembly spans 3.92 Gb, representing ~88% of the estimated pea genome. Availability of this reference genome sequence has provided a uniform template for assembly of GBS (reads), a technique commonly used in most of the recent studies for genotyping of bi-parental mapping populations and genome wide association study (GWAS) panels. Alignment of sequence reads to a common reference genome sequence has facilitated the construction of combined linkage maps and comparison of markers across different studies. The reference pea genome sequence has also contributed to the advancement of other genotyping technologies. For example, an Affymetrix 90K SNP array was developed by INRA, France based on alignment of resequencing reads of a panel of diverse accessions with the reference genome sequence [79]. The annotated reference genome assembly also facilitates reverse genetic studies and candidate gene identification. The reference genome sequence was used as a template for identification of genetic loci associated with multiple traits of pea [8,9,102] and has a great potential for fine mapping disease resistance loci through identification of additional markers within the identified loci. The reference pea genome sequence has the potential to improve genotyping technologies (e.g., SNP arrays and resequencing), and identification of additional markers

(SNPs and SSR) across the seven chromosomes of pea. The pea genome sequence provides an excellent opportunity for the identification of SNPs across Hapmap populations to map the haplotype blocks [129].

4.2. Identification of SSR Markers

The pea genome sequence has contributed to the identification of thousands of additional SNP markers in multiple resequencing/GBS-based studies, useful for fine mapping of genetic loci of breeding importance. The genome sequence acts as a template for identification of additional simple sequence repeat (SSR) markers that can be routinely used for fine mapping of identified loci, and for use in diverse genetic and breeding studies. Using the pea reference genome sequence [128], we have identified 428,545 SSR motifs comprising 86,871 compound repeats and 340,674 perfect repeats (Figure 1) [130]. These SSR motifs are distributed unevenly across the linkage groups and vary in SSR type, repeat number, and length, and can contribute to fine mapping of resistant loci. SSR markers were useful in fine mapping of a *Phytophthora sojae* resistance QTL (*RpsQ*) in soybean to a 118-kb region on chromosome 3 [131].

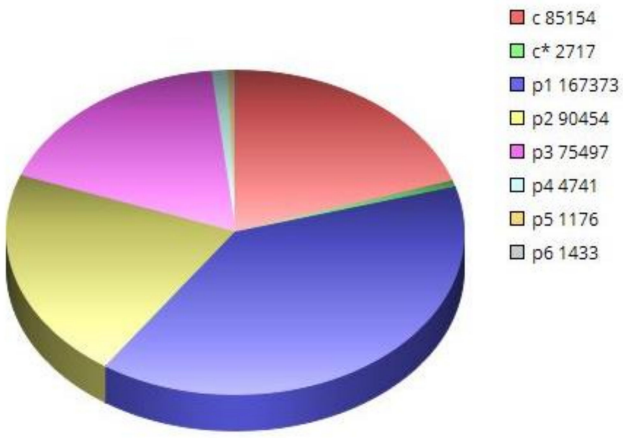

Figure 1. Distribution of SSR motif types in the reference pea genome sequence. **c**—compound motifs; **c***—compound interspersed motifs; **p**—perfect motifs. **P1** to **P6** represent the perfect motifs with 1 to 6 nucleotide repeats.

4.3. Functional Omics Studies

Functional omics analysis, including the messenger RNA and microRNA sequence analysis and DNA methylation patterns, facilitate a comprehensive genome-level analysis of complex diseases. These approaches represent an advancement over previous candidate gene or pathway analyses. The functional omics studies have enhanced the understanding of the genetic basis of plant resistance and pathogenicity in several crop species and contribute to improvement of plant resistance using genomics-assisted approaches. Based on a full length de novo assembly of RNA-seq data, Alves-Carvalho et al. [132] developed a gene expression atlas of pea to facilitate transcriptome and proteome approaches.

Modern functional genomic tools like RNA-seq have the potential to elucidate various aspects of plant–pathogen interactions at the transcriptome level to identify disease-responsive candidate genes [133,134]. Proteomics and metabolomics used in combination with RNA-seq are expected to unravel various signaling pathways and complex networks underlying the host-pathogen interaction. RNA-seq has been used for identification of differentially expressed genes of pea in response to cold [135] and flower development [136] and has a similar potential to identify the differentially expressed genes in response to pathogen infection.

A few differential gene expression studies have been conducted in pea based on the quantification of known genes. Tran et al. [137] studied the differential expression of 13 known defence-related genes during *Phoma koolunga* infection of stems and leaves

of susceptible and resistant pea accessions. Quantification of expression of these genes using qRT-PCR provided an understanding of up- and down-regulation of these genes to improve leaf and stem black spot disease resistance in pea. Barilli et al. [138] used three pea accessions, each carrying one of the three known powdery mildew resistance genes *er1*, *er2*, or *Er3*. By studying the differential expression of 20 known genes in these three accessions, it was determined that the accession with *er1* showed mainly Pschitin and Chi2 (encoding for endochitinases) accumulation after *E. pisi* inoculation, as well as genes encoding pea defensins. In comparison, the accession with *er2* gene showed accumulation of Pschitin and Chi2 and reduced activation of pea defensins. The accession with *Er3* showed the overall highest expression of pea defensins and an elicitor-inducible peroxidase. RNA-seq studies to determine the genome wide differential gene expression of pea in response to pathogen attack are scarce to date and have great potential to complement the loci identified in mapping studies. Williamson-Benavides et al. [139] used four each of tolerant and susceptible pea genotypes for time course RNA-seq after inoculation with *F. solani* f. sp. *pisi*. In this study, a set of genes overexpressed both in tolerant and susceptible genotypes were identified. Genes involved in exocytosis, anthocyanin synthesis pathway, and a pathogenesis-related gene DRR230 were among those overexpressed in tolerant genotypes.

5. Future Outlook

The increase in genomic resources of pea in recent years, particularly the reference genome sequence, facilitates understanding the allelic variation underlying the key phenotypes, which contributes to rapid cultivar development. Genomics-assisted breeding approaches such as MAS, haplotype-based breeding, allele modification through genome editing, and genomic selection (GS) in general are expected to be key components of designing future crops [140]. These approaches, importantly GS in combination with high-throughput SNP genotyping methods, are expected to contribute to breeding for enhanced productivity and quality of pea in the coming decade. Genomic selection allows for simultaneous selection of multiple traits compared to traditional marker-assisted selection.

Genomic selection, through prediction of genomic-estimated breeding values (GEBVs), contributes to selection of breeding lines for superior phenotypes of complex traits. A limited number of studies based on GS of pea have indicated its potential in future breeding of pea varieties. However, more reliable studies to establish the method for routine use are pending. Tayeh et al. [78] reported prediction accuracies of up to 0.83 for thousand-seed weight of pea. This trait was also reliably predicted in a parallel study using a collection of diverse pea accessions genotyped by SSR and retrotransposon-based insertion polymorphism (RBIP) markers [141]. Using GBS data, Annicchiarico et al. [142] predicted pea grain yield under drought conditions, and the prediction accuracy was 0.84. Carpenter et al. [143] predicted Ascochyta blight resistance with a prediction accuracy of 0.56. Using inter-population predictions, Annicchiarico et al. [144] predicted the inter-environment grain yield with a prediction accuracy of 0.19, onset of flowering of 0.40, seed weight of 0.28, and lodging susceptibility of 0.22. These studies provide proof of concept that a combination of better training and test population sets, and multiple prediction models, can increase the future efficiency of selection of pea breeding lines in multiple environments.

6. Conclusions

Breeding for disease resistance is an ongoing global challenge for pea breeders. Sourcing novel variations of disease resistance from unexploited land races and wild relatives is one way of strengthening the genetic base. Simultaneously, pyramiding of different resistance gene(s) by genomics-assisted breeding approaches could expedite breeding of resistant cultivars. Advances in genomics technologies along with availability of pea genome sequence information have the potential to deepen knowledge about the resistance candidate genes/haplotypes to accelerate breeding of disease resistant pea cultivars.

Functional genomics studies will expedite discovery of candidate loci and lead to better understanding of the molecular mechanisms underlying host-pathogen interactions. Targeted and rapid editing of disease resistance loci is possible with adoption of newer techniques like genomic selection and CRISPR/Cas9 genome editing. An efficient combination of these new approaches could accelerate the development of resistant pea cultivars in the genetic background of other desirable agronomic traits.

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