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Review

Breeding for Biotic Stress Resistance in Pea

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Abstract: Pea (*Pisum sativum*) stands out as one of the most significant and productive cool-season pulse crops cultivated worldwide. Dealing with biotic stresses remains a critical challenge in fully harnessing pea's potential productivity. As such, dedicated research and developmental efforts are necessary to make use of omic resources and advanced breeding techniques. These approaches are crucial in facilitating the rapid and timely development of high-yielding varieties that can tolerate and resist multiple stresses. The availability of advanced genomic tools, such as comprehensive genetic maps and reliable DNA markers, holds immense promise for integrating resistance genes from diverse sources. This integration helps accelerate genetic gains in pea crops. This review provides an overview of recent accomplishments in the genetic and genomic resource development of peas. It also covers the inheritance of genes controlling various biotic stress responses, genes that control pathogenesis in disease-causing organisms, the mapping of genes/QTLs, as well as transcriptomic and proteomic advancements. By combining conventional and modern omics-enabled breeding strategies, genetic gains can be significantly enhanced.

Keywords: legumes; *Pisum* spp.; resistance; breeding

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1. Introduction

Pea (*Pisum sativum*) is a cool season annual legume crop cultivated throughout the world. Depending on their uses, three major types of peas are recognized, each with differing quality requirements. These types are dry or field peas, vegetable or green peas, and forage peas. Pea usage ranges from dry seeds used for animal feed, dehulled/split seeds and meal for the food industry, immature seeds or pods for food, to whole plants for silage or grazing [1]. Dry and forage peas are typically grown under low input conditions, unlike vegetable peas, which require more intensive irrigation and fertilization. As a result, although all pea types are prone to the same pests and diseases, variations in cropping practices might influence their severity. Resistances are equally useful in resistance breeding for all types of peas [2].

In 2021, dry peas were cultivated on 7.0 Mha worldwide. It was mainly grown as a low-input crop, with an average yield of 1837 kg/ha over the last 10 years, resulting in a production of 12.4 MT [3]. The main dry pea producer countries in 2021 were Russia (3.2 MT), followed by Canada (2.3 MT), China (1.5 MT), India (0.9 MT), Ukraine (0.6 MT), and France (0.6 MT). Historically, France was the largest worldwide producer from 1988 until 1998, when it was surpassed by Canada, and in 2008, it was also surpassed by Russia, China, and India (Figure 1).

A completely different trend for vegetable peas can be observed. Despite being grown on a smaller acreage (2.6 Mha in 2021), green pea achieves higher production (20.5 MT) thanks to a higher world average yield that reaches 7752 kg/ha. World production has increased markedly since 1990, mainly due to increased production in China. The main vegetable pea producers in 2021 were China (11.5 MT), India (5.8 MT), Pakistan (0.5 MT), and France (0.3 MT) (Figure 2).

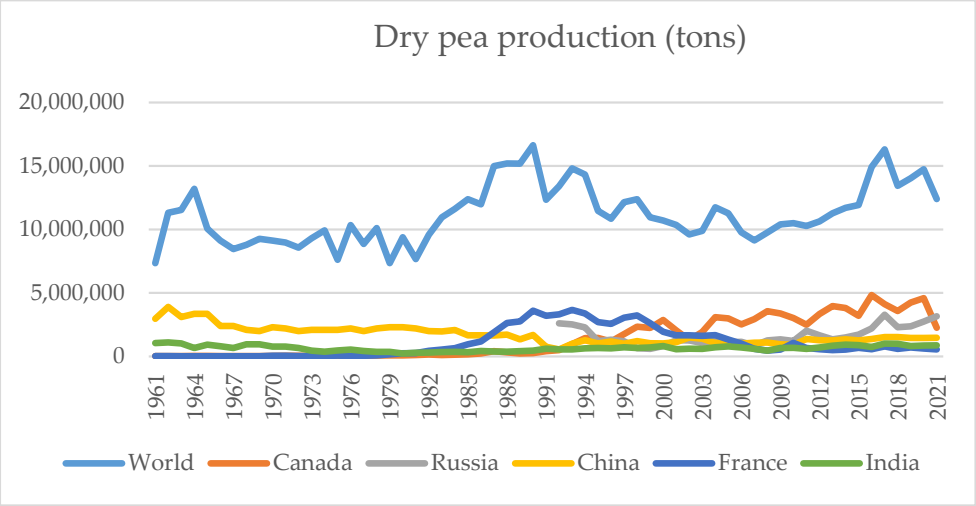

Figure 1. Trend (1961–2021) of dry pea production globally and in the five largest producing countries.

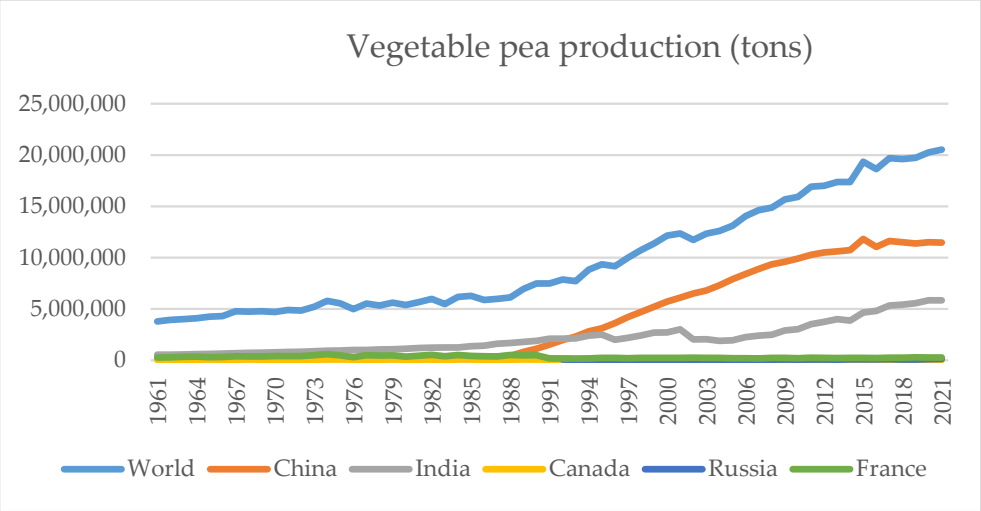

Figure 2. Trend (1961–2021) of vegetable pea production globally and in the five largest producing countries.

The average yield of dry pea has doubled globally, from approximately 1000 kg/ha in 1961 to the current approximately 2000 kg/ha, resulting in an annual yield gain of 16.4 kg/ha. However, this yield gain is lower than the one achieved for soybean (27.8 kg/ha)or wheat (40 kg/ha), indicating lower attention paid to pea research compared to those crops.

One of the main reasons for this relatively low yield is the susceptibility of peas to biotic stresses. Pea is highly susceptible to many root and aerial diseases (such as powdery mildews, rusts, mildews, wilts, and root rots) and pests, which constantly reduce its yield (from about 20% to 100% locally in case of acute infection) and product quality [1] (Table 1). In all cases, introducing durable resistance is recognized as the most efficient and environmentally friendly control measure. Some level of resistance has been identified against most pea diseases and pests [1]. Histological and biochemical studies showed that resistance was due to a wide range of defense mechanisms, including cell wall strengthening, papilla formation, hypersensitive response, and accumulation of phenolic compounds such as pisatin, PR proteins, and reactive oxygen species, among others [1]. This review provides a concise overview of recent accomplishments in the genetic and genomic resource development of peas. It also covers the inheritance of genes controlling the most important biotic stress responses in peas.

Table 1. Characteristics of the most important biotic stresses of pea crop.

Biotic Stress	Pathogen Species	Source of Infection	Organ	Distribution
Aerial fungi				
Ascochyta blight complex	*Ascochyta pisi* Lib.	Infected crop debris, seedborne, ascospores, and conidia	Leaves, stems, pods, and seeds	Europe and North America
	A. pinodes Berk. and Blox.	Infected crop debris, seedborne, ascospores, and conidia	Leaves, stems, pods, and seeds	Worldwide
	Phoma medicaginis var. *pinodella* (L.K. Jones) Boerema	Infected crop debris, seedborne, ascospores, and conidia	Leaves, stems, pods, and seeds	Worldwide
	P. koolunga Davidson	Infected crop debris, seedborne, ascospores, and conidia	Leaves, stems, pods, and seeds	Australia
	P. glomerata [(Corda) (Wollenw. and Hochapfel)]	Infected crop debris, seedborne, ascospores, and conidia	Leaves, stems, pods, and seeds	Australia
Powdery mildew	*Erysiphe pisi* (DC.)	Infected crop debris and conidia	Leaves, stems, and pods	Worldwide climates with warm, dry days and cool nights
	E. trifolii (Grev.)	Infected crop debris and conidia	Leaves, stems, and pods	USA, India, Spain, and Tunisia
Downy mildew	*Peronospora viciae* (Berk.) Caspary f.sp. *pisi* Sidow.	Infected crop debris, oospores, and conidia	Leaves, stems, pods, and seeds	Cool and wet weather conditions
Rust	*Uromyces pisi* (Pers.) Wint.	Infected debris of *Euphorbia cyparissias* L. and urediospores	Leaves, stems, and occasionally pods	Temperate regions
	U. viciae-fabae (Pers.) de Bary	Infected crop debris, aeciospores, and urediospores	Leaves, stems, and occasionally pods	Tropical and sub-tropical regions, e.g., India, China
Soilborne diseases				
Fusarium wilt	*Fusarium oxysporum* f.sp. *pisi* (W.C. Snyder and H.N. Hansen)	Infected crop debris, chlamydospores, and micro- and macroconidia	Roots, xylem vessels, and seeds	Worldwide, in both dry and wet field conditions
Fusarium root rot complex	*Fusarium solani* f. sp. *pisi* (W.C. Snyder and H.N. Hansen)	Infected crop debris, chlamydospores, and micro- and macroconidia	Roots and seeds	Worldwide (mainly in the Pacific North-West regions)
	F. graminearum Schw.	Infected crop and cereal debris, ascospores, and micro- and macroconidia	Roots and seeds	Canada, USA, and Europe
	F. avenaceum (Fries) Saccardo	Infected crop debris and ascospores	Roots and seeds	Canada, USA, and Europe
Common root rot	*Aphanomyces euteiches* (Drechsler)	Infected soil and crop debris, oospores, and zoospores	Roots, stems, and leaves	Worldwide, temperate, and wet areas
Rhizoctonia root rot	*Rhizoctonia solani* Kühn	Infected soil and sclerotia	Roots, stems, and leaves	Temperate and subarctic areas

Table 1. *Cont.*

Biotic Stress	Pathogen Species	Source of Infection	Organ	Distribution
Bacteria				
Pea blight	*Pseudomonas syringae* pv. *pisi* Sackett	Infected seeds and crop debris	Leaves, stems, pods, and seeds	Areas with cool and wet weather
Viruses				
Pea Seed-borne Mosaic Virus	PSbMV	Infected seeds and aphids	Leaves, stems, pods, and seeds	Worldwide
Pea Enation Mosaic Virus	PEMV	Infected aphids (infected seeds in a small proportion)	Leaves, stems, pods, and seeds	USA, Europe, Africa, and India
Nematodes				
Cyst nematodes	*Heterodera goettingiana* Liebscher	Infected soil and roots with eggs	Roots	Worldwide
Root-knot nematodes	*Meloidogyne incognita* (Kofoid and White) Chitwood	Infected soil and roots with eggs	Roots	Europe
Root lesion nematodes	*Pratylenchus neglectus* Rensch	Infected soil and roots with eggs	Roots	Worldwide
	P. thornei Sher and Allen	Infected soil and roots with eggs	Roots	Worldwide
Parasitic plants				
Broomrapes	*Orobanche crenata* Forskal	Infested soil with seeds	Roots	Mediterranean basin
Insect pests				
Pea weevil	*Bruchus pisorum* L.	Infested seeds	Pods and seeds	Worldwide
Pea aphid	*Acyrthosiphon pisum* H.	Infested soil and crop debris with eggs, parthenogenetic individuals	Leaves, stems, pods, and seeds	Temperate areas

2. State of the Art by Groups of Diseases and Pests

2.1. Ascochyta Blight

Ascochyta blight is a complex disease that causes necrotic spots on leaves and stems. It can be caused by several fungi, such as *Ascochyta pisi*, *Peyronellaea pinodes*, and different species of *Phoma*, including *Ph. medicaginis* var. *pinodella*, *Ph. koolunga*, and *Ph. glomerata* [4]. Out of these species, *P. pinodes* appears to be the most widespread and damaging. Only moderate levels of quantitative resistance are available [5–11]. Several quantitative trait loci (QTLs) associated with partial resistance to ascochyta blight have been reported (Table 2) [7–11]. However, these QTLs explain only a limited percentage of the phenotypic variation. In addition, the associated markers are still too far away, impeding their implementation for marker-assisted selection (MAS) and identification of the underlying genes. As a result, progress in resistance breeding is slow [12]. Defense responses against *P. pinodes* include the accumulation of pisatin [13], activation of defense genes such as phenylalanine ammonia-lyase, chalcone synthase, pathogenesis related (PR) proteins, and polyphosphoinositide metabolism [14]. Resistance is associated with reduced colony establishment and smaller lesion sizes as a consequence of protein cross-linking, hydrogen peroxide accumulation, and a greater frequency of epidermal cell death [15]. Early synthesis of pisatin was also identified as a key factor in resistance against *P. koolunga* [16]. Several transcriptomic and proteomic studies have been performed to identify candidate genes and proteins to be used as markers. A number of transcriptomic studies, such as the *Medicago truncatula* microarray [17], expressed sequence tag (EST)-based microarray analysis [18], DeepSuperSAGE genome-wide transcriptional profiling [19], or Massive Analysis of cDNA Ends (MACE) [20], identified a large number of up- or down-regulated genes that could be used as expression markers for resistance. Similarly, shotgun proteomics allowed the

identification of protein markers that could be used to select for resistance in peas [21]. A recent screening of a large collection of peas against multiple isolates of *P. pinodes* and *Ph. koolunga* identified novel resistance sources to both pathogenic species [10]. It also identified more closely linked markers and novel candidate resistance genes, showing promise for future resistance breeding of pea against ascochyta blight [10].

2.2. Mildews

Powdery mildew is a foliar disease mainly incited by the biotrophic fungus *Erysiphe pisi*, although other species such as *E. trifolii* can also infect peas. Three monogenic resistance genes are available so far for pea breeding, along with accessions showing varying levels of resistance. Two of these genes are recessive (*er1* and *er2*), while the third one is dominant (*Er3*) [22]. These three genes have been mapped using different types of markers and are located on chr1LGVI, chr5LGIII, and chr4LGIV, respectively [22,23]. From these three genes, *er1* is the most widely deployed gene in breeding programs. Despite being monogenic, the resistance provided by *er1* is considered durable [24]. This gene confers a pre-penetration non-hypersensitive response [25], not associated with callose papillae deposition but with protein cross-linking [26]. Histochemical and biochemical analyses suggest that *er1* resistance possibly utilizes antioxidant machinery to maintain a low level of ROS [27]. The *er1* phenotype is conferred by loss-of-function mutations in the susceptible gene *PsMLO1* [28,29]. To date, 12 *er1* alleles have been identified, including two artificial chemical mutations (*er1-5* and *er1-10*) and ten natural mutations [30]. However, *E. trifolii* is known to defeat *er1* resistance, requiring breeding attention [31,32]. Therefore, pyramiding more than one gene into a single background is desirable. The *er2* expression is affected by temperature and plant age, being effective in mature leaves and at temperatures higher than 25 °C [25]. The *er2* resistance is likely conferred by maintaining ROS balance coupled with rapid pathogenesis-related gene 1 (*PR-1*) accumulation [33]. Transcriptome analysis identified 2755 transcripts involved in resistance to *E. pisi* [34]. Proteomic analyses have identified proteins involved in virulence and pathogenesis, including signal transduction, secondary metabolites, and stress response [35,36]. *Er3* was initially identified in a wild *P. fulvum* accession. It confers complete resistance to *E. pisi* through hypersensitive cell death initiated rapidly after penetration [37]. *Er3* has been mapped onto chr4LGIV, but no candidate genes have been identified yet. It was nonetheless successfully transferred to some elite pea cultivars [37,38].

Pea downy mildew, caused by the oomycete *Peronospora viciae* f.sp. *pisi*, can be important in cooler areas. Monogenic resistance has been reported with at least one dominant gene (*Rpv*) and two complementary recessive ones (*rpv-1* and *rpv-2*) [39]. Differential expression of host proteins has been identified [40]. More recently, markers associated with adult plant resistance have been identified by genome-wide association study (GWAS) approaches on chr1LGVI, chr3LGV, and chr6LGII [41], offering some potential for future breeding although no candidate genes could be identified.

2.3. Rusts

Pea rust is a widespread disease that affects both leaves and stems. In temperate climates, it is caused by the pathogen *Uromyces pisi*, whereas *U. viciae-fabae* is prevalent in tropical areas. Despite a scarcity of hypersensitive responses, levels of partial resistance are available in both cases [42,43], possibly attributed to a single gene/major QTL (named *Ruf* for *U. viciae-fabae* and *UpDS* for *U. pisi*) [44–46]. In addition, a recent study has identified for the first time a late-acting hypersensitive response in a pea-*U. pisi* pathosystem [47]. Further studies targeting the establishment of its genetic base are ongoing.

Slow rusting resistance was described in peas as a type of resistance independent of the pathogenic race. It is characterized by retarded disease progression, resulting in moderate disease levels despite a compatible host-pathogen interaction [42,43,47,48]. This resistance is pre-haustorial in nature and influenced by the crop growth stage and environment. In addition, slow-rusting, is often associated with the formation of lignin and callose as part

of the plant's defense mechanisms. The phenyl ammonia lyase (PAL) enzyme might play a role in the expression of slow rusting, although additional genes might also participate. Total phenolic accumulation, induction of actin, and several pathogenesis-related proteins such as *PR-1* and *PR-2* have also been linked to partial resistance [48–50].

2.4. Wilts and Root Rots

Wilts and root rots are major soilborne diseases of peas that are difficult to manage. Fusarium wilt is incited by several races of *Fusarium oxysporum* f.sp. *pisi*. Single race-specific resistance has been detected for races 1, 5, and 6 [51] while resistance to race 2 is quantitative [52]. Resistance to races 1 and 5 has been successfully incorporated into pea cultivars through classical breeding [53,54]. Genetic mapping efforts located resistance to races 1 and 5 in pea chr5LGIII and chr6LGII, respectively [55]. Different studies have identified DNA markers linked to race 1 resistance genes [56–58]. Resistance to race 2 has also been identified [52,59], but it is more complex, with at least two minor loci (*Fnw3.1* and *Fnw3.2*) and a major one (*Fnw4.1*) [60]. Further studies showed the role of physical and chemical barriers within pea root tissues in resistance to race 2, leading to cell wall and xylem reinforcement to block pathogen growth [61]. A pre-penetration resistance mechanism reducing *Fop* race 2 germination mediated by the constitutive exudation of pisatin was also detected in some pea accessions [62]. In addition, a proteomic analysis identified 53 proteins responsible for various functions in pea, confirming the involvement of phenolics in the resistance to race 2 [63].

Fusarium root rot in pea is mainly incited by *Fusarium solani* f. sp. *pisi* (*Fsp*), although *F. avenaceum* is gradually gaining prominence [64]. Some levels of incomplete resistance have been reported. Interestingly, this resistance is more frequently detected in genotypes with pigmented flowers and seed coats [65,66]. QTL associated with *Fsp* resistance have been determined, explaining up to 53% of the phenotypic variance [65,67–69]. More recently, SNPs have been identified in *Fsp*-responsive differentially expressed genes. They were used to refine the location of QTLs associated with partial *Fsp* resistance using composite interval mapping (CIM) in two recombinant inbred line (RIL) populations [70]. This approach identified five QTLs explaining from 5.3% to 14.8% of the variance. The evaluation of another RIL population also allowed the identification of five QTLs for resistance to *F. graminearum*, another species of the Fusarium root rot complex. The most stable QTL was localized in linkage group IV [71].

Aphanomyces root rot is caused by the soilborne oomycete *Aphanomyces euteiches*. The general threat of this rot complex on peas in most growing regions drove research to improve its management and resistance. These studies have aided in pathogen characterization and identified alleles linked to established partial resistance [72]. Genetic studies, using either biparental populations [73,74] or GWAS [75–77], show the complex inheritance underlying resistance, complicating resistance breeding. A transcriptome analysis revealed the involvement of genes associated with phenylpropanoid metabolism, strengthening of the cell wall, and hormonal signaling (jasmonic acid, auxin, and ethylene) in response to *A. euteiches* [78]. These efforts have guided the improvement of root rots resistance, specifically toward precision and maker-assisted breeding [79]. This allows the transfer of several of the main QTLs to advanced pea lines showing increased levels of resistance, although no cultivar with full resistance has been developed so far [80].

Rhizoctonia root rot, caused by *Rhizoctonia* spp., is another soilborne disease that can reduce pea yield in some regions. Little resistance is available so far, with only reports of reduced infection linked to seedling epicotyl thickness and plants becoming less susceptible with age [81]. On the other hand, the *Pythium* spp. complex is responsible for dumping off as well as seed/seedling and shoot rot. Few sources of resistance are available, calling for the need to intensify resistance screenings [82,83].

2.5. Root Parasitic Nematodes

Parasitic nematodes can cause significant damage to peas. They are challenging to manage due to their broad host range and the scarcity of available resistance sources. The most damaging nematodes include cyst, root knot, and root lesion nematodes. The most widespread cyst nematode is *Heterodera goettingiana*, which can survive in the soil for long periods [84]. No resistance has been reported against this pathogen so far. However, studies have shown that lipoxygenase enzymes can inhibit *H. goettingiana* growth in pea roots [85], offering potential for resistance breeding.

The most damaging knot nematode is *Meloidogyne incognita*, whose management is also difficult due to its broad host range and the lack of identified resistance. A negative correlation between pea biomass and root knot infection has been found [86]. The most damaging root lesion nematodes are *Pratylenchus neglectus* and *P. thornei*. No resistance is available so far in pea, while some resistance has been identified in other legume crops [87]. By contrast, some resistance has been identified in pea against *P. nanus* [88].

2.6. Broomrapes

Broomrapes are soil-borne root parasitic plants belonging to the family Orobancheae. Among the most damaging and widely distributed species infecting peas is *Orobanche crenata* [89]. Pea breeding for broomrape resistance has been slow but successful [90,91]. Phenotypic evaluations in the field and under controlled conditions in pots and rhizotrons have revealed some sources of partial resistance. Resistance was mediated by a range of mechanisms, including avoidance, low induction of seed germination, and inhibition mechanisms against the pathogen [92–95]. Partial resistance has been identified in wild pea and landraces [90] and successfully bred into pea cultivars [96–98]. As an alternative to resistance, broomrape can be managed by breeding for early maturity lines, which have the advantage of escaping to outcompete the parasite [99]. Preliminary evaluations on a pea core collection panel have also presented several potential resistance lines against *O. crenata* under field conditions [100].

A first mapping study detected two QTLs for broomrape resistance using DNA markers in an $F_{2:3}$ bi-parental population [101]. Later, four QTLs were identified as associated using RIL populations derived from the same cross [102]. These were associated with broomrape emergence and development under field conditions and/or with specific resistance mechanisms in vitro. More recently, the study of a different RIL population [103] allowed the identification of three QTLs associated with the field response to *O. crenata* infection and the development of three KASP markers linked to these QTLs.

Gene expression approaches have been used to profile *Medicago truncatula* against *O. crenata*, revealing a potential comprehensive source of *O. crenata* resistance and gene patterns associated with plant pathological resistance [104]. Proteomics has been employed to decipher protease inhibition pathways to improve the molecular basis for early broomrape infection, first in *M. truncatula* [105] and then in pea [106]. This helped our understanding of the biochemical processes involved in resistance and the selection of potential candidates for improvement through gene silencing (RNAs, siRNA) or gene editing (CRISPR/Cas9), which could contribute to delivering *O. crenata* resistance in the future.

2.7. Bacterial Blight

Up to eight races of the seedborne bacteria *Pseudomonas syringae* pv. *pisi* have been reported to affect peas [107]. Race-specific monogenic resistances have been identified and mapped [108–111]. Additional QTLs have also been reported [112]. Interestingly, *P. abyssinicum* accessions exhibit resistance (total or partial) to all races, including race 6. This valuable resistance in *P. abyssinicum* is controlled by a major recessive gene along with several modifiers [113]. In an effort to gain deeper insights into the molecular mechanisms underlying bacterial blight resistance in peas, a deepSuperSAGE transcriptomic approach was employed. This led to the identification of UniTags differentially expressed between

resistant and susceptible accessions [110]. These UniTags represent potential candidate genes that may play crucial roles in conferring resistance against this pathogen.

2.8. Viruses

Pea Seed-borne Mosaic Virus (PSbMV) can be transmitted through both infected seeds and aphids. Up to four different races or pathotypes of the virus have been detected. Race-specific recessive resistance genes are available (*sbm1* to *4*) [114,115]. It is worth noting that all these genes, except *sbm2*, are clustered in the same chr1LGVI region [116]. KASP markers have been developed, identifying two PSbMV alleles, and used to identify novel sources of resistance in pea germplasm [117,118]. Apart from PSbMV, other viruses affecting peas have been studied for resistance. A recessive monogenic resistance has been identified against the Bean Yellow Mosaic Virus (BYMV) [119]. Similarly, resistance to Bean Leaf Roll Virus (BLRV) has been found to be conferred by a recessive gene [120]. By contrast, Pea Enation Mosaic Virus (PEMV) resistance is controlled by a dominant gene (*En*), located on chr5LGIII. The identification of closely linked markers has allowed the prediction of *En* presence with 99.4% accuracy, making it highly suitable for MAS strategies [121].

2.9. Insect Pests

Pea weevils (*Bruchus pisorum*) cause significant damage to stored pea seeds, leading to increasing concerns in organic production. Adults feed on pollen, causing no damage, but larvae emerging from eggs laid on young pods penetrate through the pod and seeds, feeding on the cotyledon and molting inside the seeds. Moderate levels of resistance have been reported in cultivated and wild pea relatives [122,123]. Resistance involves a combination of antixenosis and antibiosis mechanisms, resulting in reduced seed infestation and retarded larval development [124,125]. Genetic studies in interspecific crosses of *P. fulvum* suggested three recessive alleles [126]. Additionally, neoplasm formation is suggested to contribute to bruchus resistance. Neoplasm formation is controlled by a single dominant gene, and its expression is highly influenced by environmental factors [127]. Accordingly, three QTLs associated with reduced seed infestation and one QTL for reduced larval development were identified from a RIL population, along with seven potential candidate genes located in close proximity to these QTLs [128]. This offers breeders opportunities to develop effective and sustainable strategies for weevil control in peas.

Pea aphids (*Acyrthosiphon pisum*) can be very constraining to peas. Incomplete resistance is available. It results from a combination of antixenosis and antibiosis resistance mechanisms [129–132]. QTLs associated with tolerance to aphid damage have been reported in a RIL population derived from two *P. fulvum* accessions [133]. Further genetic studies have enabled the identification of a major-effect quantitative trait locus, *ApRVII*, on Chr7LGVII, associated with resistance against different adapted and non-adapted biotypes of pea aphids [131]. A subsequent GWAS [132] on a different pea panel identified additional SNPs associated with resistance. Earlier proteomic analysis identified proteins related to various processes, including amino acid and carbohydrate metabolism, photosynthesis, folding/degradation, stress response, signal transduction, and transcription/translation [134].

Table 2. List of genes and QTL available for pea resistance breeding against the most important pea diseases and pests.

Biotic Stress	Pathogen	Gene/QTL	Effect	Linkage Group	Resistance Type	Reference
Aerial fungi or oomycete						
Ascochyta blight	*Peyronellaea pinodes*	*Dp1.1*, *Dp1.2*, *Dp1.3*, *MpII.1*, *Dp3.1*, *Dp3.2*, *Dp3.3*, *Dp3.4*, *Dp3.5*, *Dp3.6*, *Dp3.7*, *Dp3.8*, *Dp3.9*, *MpIV.1* *Dp5.1*, *Dp5.2*, *Dp5.3*, *Dp6.1*, *Dp6.2*, *Dp6.3*, *Dp6.4*, *Dp7.1*, *Dp7.2*, *Dp7.3*	Minor to moderate	Chr2LGI Chr6LGII Chr5LGIII Chr4LGIV Chr3LGV Chr1LGVI Chr7LGVII	Incomplete	[6,7,11]
Powdery mildew	*Erysiphe pisi*	*er1*, *er2*, *Er3*	Major Major Major	Chr1LGVI Chr5LGIII Chr4LGIV	Incomplete	[22,23]
Downy mildew	*Peronospora viciae* f. sp. *pisi*	*3552605*, *3559062*, *5943381* *Rpv* *rpv-1* *rpv-2*	Major Minor Minor	Chr1LGVI Chr3LGV Chr6LGII Chr2LGI	Complete	[39,41]
Rust	*Uromyces pisi*	*UpDSII*, *UpDSIV*, *UpDSIV.2*	Major Major Minor		Incomplete	[46]
	Uromyces viciae-fabae	*Ruf*	Major		Incomplete	[44]
Soilborne fungi or oomycete						
Fusarium root rot	*Fusarium solani* f. sp. *pisi*	*Fsp-Ps2.1*, *Fsp-Ps6.1*, *Fsp-Ps3.1*, *Fsp-Ps3.2*, *Fsp-Ps3.3*, *Fsp-4.1*, *Fsp-Ps7.1*		Chr6LGII Chr1LGVI Chr5LGIII Chr7LGVII	Incomplete	[68,69]
	F. graminearum	*Fg-Ps3.1*, *Fg-s3.2*, *Fg-Ps4.1*, *Fg-s4.2*, *Fg-Ps5.1*	Minor Moderate Minor	Chr5LGIII Chr4LGIV Chr3LGV	Incomplete	[71]
Fusarium wilt	*F. oxysporum*. f. sp. *pisi* race 1	*Fw*	Major	Chr5LGIII	Complete	[56–58]
	F. oxysporum. f. sp. *pisi* race 2	*Fnw 3.1*, *Fnw 3.2*, *Fnw 4.1*	Minor Major	Chr5LGIII Chr4LGIV	Complete	[60]
	F. oxysporum. f. sp. *pisi* race 5	*Fwf*	Major	Chr6LGII	Complete	[55]
Common root rot	*Aphanomyces euteiches*	*Ae-Ps1.1*, *Ae-Ps1.2* *Ae-Ps2.1*, *Ae-Ps2.2* *Ae-Ps3.1*, *Ae-Ps3.2* *Ae-Ps4-4*, *Ae-Ps4.5* *Ae-Ps5.1*, *Ae-Ps6.1*, *Ae-Ps7.6*	Minor Minor Minor Minor Minor Minor Major	Chr2LGI Chr6LGII Chr5LGIII Chr4LGIV Chr3LGV Chr1LGVI Chr7LG7	Incomplete	[74–76]

Table 2. *Cont.*

Biotic Stress	Pathogen	Gene/QTL	Effect	Linkage Group	Resistance Type	Reference
Bacteria						
Pea blight	*Pseudomonas syringae* pv. *pisi*	*Ppi1*, *Ppi2*, *Ppi3*, *Ppi4*, *Ppi8*		Chr1LGVI Chr7LGVII Chr6LGII, Chr5LGIII	complete	[108–110]
	Pseudomonas syringae pv. *syringae*	*PsBB1-Psy*	Minor	Chr6LGII	complete	[111,112]
		Psy1, *PsBB3-Psy*, *PsBB4-Psy*	Major	Chr5LGIII		
		Psy2, *PsBB5-Psy*, *PsBB6-Psy*	Minor Minor	Chr1LGVI Chr7LG7		
Viruses						
Pea Seed-borne Mosaic Virus	*PSbMV*	*sbm-1*, *sbm-3*, *sbm-4* *sbm-2*	Major	Chr1LGVI Chr6LGII	Complete	[114]
Pea Enation Mosaic Virus	*PEMV*	*En*	Major	Chr5LGIII	Complete	[121]
Pea common Mosaic virus	*PMV*	*mo*	Major	Chr6LGII	Complete	[119]
Parasitic Plant						
Broomrape	*Orobanche crenata*	*N°br03-1*, *N°br03-2*, *PsOcr3* *N°br03-3*, *N°br04*, *PsOcr2*, *PsOcr1*	Chr2LGI Chr5LGIII Chr3LGV Chr1LGVI Chr4LGIV	Moderate Minor Moderate Moderate Major	Partial	[102,103]
Insect Pest						
Pea weevil	*Bruchus pisorum*	*BpSI.I*, *BpSI.II*, *BpSI.III*, *BpLD.I*	Chr2LGI Chr6LGII Chr4LGIV	Moderate	Partial	[128]
Pea aphid	*Acyrthosiphon pisum*	*ApI*, *ApII*, *ApIII*, *ApIV.1*, *ApIV.2* *ApV.1*, *ApV.2*, *ApV.3*, *ApRVII*	Chr7LG7 Chr3LGV Chr5LGIII Chr6LGII Chr1LGVI Chr7LG7	Minor Minor Minor Minor Major Major	Partial	[131,133]

3. Germplasm Resources for Tolerance Traits

Pea was primarily domesticated in the Near East about 10,000 years ago, with secondary expansion and diversification in the Mediterranean, East Africa with the Abyssinian types, and central Asia with the long-vined Afghan types. Similar to other grain legumes, peas were a key diet component of early civilizations, complementing cereals. It is widely grown in temperate regions as a winter crop across Europe, Asia, and North America. The most commonly accepted taxonomic classification assigns peas to the *Pisum* genus and distinguishes three species: *P. sativum*, *P. fulvum*, and *P. abyssinicum* [135]. However, the classification of *P. abyssinicum* as an independent species or a subspecies within *P. sativum* is still under debate [136,137]. *P. sativum* is the major species of the genus. It contains both wild and cultivated peas. A recent study separated this species into at least five subspecies: *P.s. elatius* (wild), *P.s. humile* (wild), *P.s. jomardii* (domesticated), *P.s. arvense* (domesticated), and *P.s. sativum* (domesticated) [137]. Additional wild subspecies have also been described, although their taxonomic status remains unclear [137]. All *Pisum* species and subspecies

are crossable and produce viable hybrids, albeit at a low rate [138,139]. This facilitates the exploitation of the wide genetic variation of peas during pre-breeding.

Breeding requires the availability of germplasm with sufficient diversity for the desired traits and affordable screening methods. Large germplasm collections, amounting to over 60,000 accessions and encompassing wild, landrace, breeding lines, and mutants, are maintained in a number of gene banks, constituting a valuable pre-breeding resource [140–144]. Pea diversity held in gene banks has been characterized using morphological descriptors and agronomic traits. Subsets of pea germplasm have also been searched for resistance to specific stresses. However, it represents only 1% of the collections. The rest remains largely uncharacterized against most stresses, leaving room to identify needed resistances. Wild relatives are excellent candidates for sources of resistance to biotic stresses. Fortunately, the various *Pisum* species and subspecies cross readily, making the genetic diversity available in the secondary gene pool accessible for pea breeding. As a result, resistance to pea weevil, ascochyta blight, broomrape, and powdery mildew has already been transferred to pea from wild *Pisum* by sexual hybridization [37,98,126]. To access the tertiary gene pool, attempts have been made to cross *P. sativum* with more distant species, such as *Lathyrus sativus*, through protoplast fusion. This allows the formation of somatic hybrids [145], but no fertile plants have been generated so far.

4. Generating Novel Variations for Pest and Disease Resistance

4.1. Induced Mutagenesis

Induced mutagenesis has been frequently used in legume breeding and remains a valuable breeding tool [146–148]. Large mutant collections can be easily produced through chemical or physical methods. However, identification of the desired mutants within these large collections required the availability of a strong selection method. Although tedious, this approach has allowed the identification of mutants with resistance against powdery mildew [148–150], fusarium wilt [151], and aphanomyces root rot [152]. More recently, the establishment of targeted-induced local lesion in the genome (TILLING) and deletion-TILLING platforms has facilitated high-throughput identification of mutated sites [153,154]. Although their application has been so far restricted to functional analysis of candidate genes, identified mutants can be applied directly or as a pre-breeding material for resistance breeding.

4.2. Transgenic Technology

Pea genetic transformation is feasible but arduous due to difficulties in plant regeneration [155]. Transgenic pea lines resistant to the tobacco budworm insect [156] or with increased resistance to viruses such as Alfalfa Mosaic Virus [157], Pea Seedborne Mosaic Virus [158], and Pea Enation Mosaic Virus [159] have been achieved. Despite these achievements, the level of resistance gained by the transgenic lines was sometimes lower than expected, or these lines were not accepted by the market for various reasons. For example, transgenic lines expressing four antifungal genes did not show consistent resistance to fusarium root rot [160]. By contrast, the transfer of α-amylase inhibitor from common beans provided protection against bruchus weevil in pea [161], but raised concerns due to their potential immunogenicity [162]. The main obstacle to adopting transgenic technology in pea breeding is the rigid genetically modified organism (GMO) legislation in some countries, coupled with low public acceptance. Accordingly, no transgenic pea lines have so far reached commercial application.

4.3. Gene Editing

New breeding techniques based on targeted gene editing offer new hope [163]. This approach is based on the targeted modification of endogenous genes. It could potentially remove some of the social concerns raised by GMOs since it does not involve the addition of foreign DNA. Targeted gene editing has been successfully established for several legumes but remains a challenge for peas due to their regeneration recalcitrance [164].

Two transformation methods have recently been tested for gene editing in pea [163,165]. These methods, based on mesophyll protoplast transformation and *Agrobacterium*-mediated explant transformation, respectively, showed promise for efficient gene editing of pea cells. However, regeneration of stable gene-edited plants, which has only been tested for the *Agrobacterium* transformation methods, was lower than 1%. This demonstrates the feasibility of the transformation methods, although additional efforts should be made to improve gene-editing efficiency and regeneration rate in this species [163,165].

5. Understanding the Genetic Makeup of Plant Traits Imparting Resistance

Pea has a long history as a model species since the studies of Mendel, which contributed to establish his laws of genetics and heredity. Despite that, pea research lagged behind many other crops for decades due to its large genome size, which delayed the development of genomic resources [137]. Fortunately, modern genomic tools, including next generation sequencing (NGS)-derived approaches that allow genome-wide association studies (GWAS), genomic selection (GS), and omic platforms (transcriptomic, proteomic, and metabolomic), are rapidly developing in pea and are readily adopted by breeders [166–168]. When lacking in elite pea cultivars, resistance to pests and diseases can be searched for in wild, mutant, unadapted germplasm, or other species and introgressed by crossing, mutagenesis, transgenic technology, or gene editing. The improvement and cost reduction of NGS-derived approaches and the release of the pea reference genome are facilitating the identification of new resistance loci or alleles. It also facilitates the development of diagnostic markers to be used in breeding, which should allow the more efficient implementation of marker-assisted breeding (MAB) [169,170]. This is expected to accelerate the generation of novel pea lines with higher resistance to pests and diseases. However, introducing durable and sustainable resistance requires complementing this molecular knowledge with a thorough understanding of plant and pathogen biology, their genetic variability, and host-pathogen associations. Implementation of advanced histological approaches [25,61] allows identification of the range of resistance mechanisms available against each biotic stress. In addition, integration of transcriptomic, proteomics, and metabolomics approaches [18–21,34,35,63,72,87] can contribute to identifying their underlying genes and proteins. However, implementation of these approaches requires detailed phenotyping and the establishment of affordable and reliable resistance screening methods, which is becoming the true bottleneck for resistance breeding.

5.1. Phenotyping and Phenomics

The decrease in sequencing cost and the constant development of novel genomic tools provide opportunities for the identification of new allelic variants effective against complex pea diseases. However, the exploitation of this wealth of resources requires accurate and affordable screening tools, which is today a major bottleneck. Detailed screening protocols have been established for most pests and diseases under field, greenhouse, or controlled conditions, but they remain highly time-consuming [23,37,39,43,47,52,66,81,86,91,107]. High-throughput phenomic platforms are becoming available and being used in pea research, shedding some light on how to solve the challenge of phenotyping [171–174]. Reports on the implementation of a semi-automated phenotyping platform are limited. Only one study reported the implementation of a greenhouse-based phenotyping platform to assess disease resistance in peas [171]. This study allowed screening of a set of 300 advanced breeding lines for aphanomyces root rot resistance and facilitated GWAS mapping [171]. In addition, automated phenotyping platforms under controlled conditions were also implemented in peas to assess cold tolerance [172] and early vigor [173] by digital color imaging technology. In open field conditions, aerial-based imaging platforms [174] and unmanned aerial systems [175,176] have also been used to phenotype pea biomass or yield. Implementation of such large-scale phenotyping approaches is expected to increase in the near future, providing detailed phenotypic information on pea responses to diseases.

In parallel to the implementation of semi-automated phenotyping platforms, image-based analysis systems are being developed to estimate disease severity [177–179]. They are expected to improve and increase the precision of disease ratings. Application of infrared thermography allows discrimination of susceptible and resistant pea plants against fusarium wilt before typical wilt symptoms can be visually detected [177]. An image-based analysis system implemented in R was also recently developed to assess rust disease progression parameters under controlled conditions, which could be implemented in automated phenotyping platforms [178]. Machine learning coupled with image analysis has also been attempted to improve pea screening for aphanomyces root rot resistance [179]. These initial attempts at image-based analysis of disease have demonstrated their effectiveness in improving accuracy in measurements and reducing processing time. Accordingly, the development and application of image-based systems will play a key role in the future development of resistance breeding in peas.

5.2. Genetic Mapping

Many pea linkage maps based on biparental populations have been generated over the years with the use of different DNA markers as they became available [168]. These maps are rapidly improved by the novel genome-wide sequencing approaches [46,137,166,180,181]. This, together with proper phenotyping, is allowing the identification of trait associations through QTL mapping or GWAS. As a result, markers associated with resistance genes/QTLs have been identified for resistance against ascochyta blight [7,8,119], powdery mildew [22,30,182,183], downy mildew [41], rust [44–46,184], fusarium root rot [67–70], fusarium wilt [60,185], aphanomyces root rot [73–79], broomrape [101–103], bacterial blight [110–112], several viruses [114,115,118,121], weevil [128], and aphid [131–133]. Earlier reported markers were often not close enough for precise utilization in MAS. However, this is being rapidly improved by the use of advanced sequencing technologies. This allows the saturation of genetic maps and the generation of gene-based markers, greatly reducing the distance between the linked markers and the trait. SNP markers are also being converted to competitive allele-specific PCR (KASP) markers for more flexible genotyping [11,118]. As an example, marker-assisted backcrossing (MABC) has been successfully used to introgress one to three of the seven main QTLs for aphanomyces root rot resistance into several recipient lines [74].

Both bi-parental and association mapping approaches have been utilized to identify closely associated markers with disease resistance genes in pea [8–10,46,60,69–72,75,102,128]. While these approaches are largely improving our understanding of the genetic control of resistance, they also have some limitations. To circumvent these limitations, multiparent populations such as nested association mapping (NAM) and multi-parent advanced generation inter-cross (MAGIC) populations that combine GWAS and QTL mapping approaches have been proposed [186]. Seminal works in different species, including *A. thaliana*, maize and barley, showed the usefulness of these approaches to unravel the genetic control of important traits and increase their precision [186]. Several NAM or MAGIC populations have been developed for several legumes, including peanuts, soybeans, cowpea, and fava beans [186]. Both approaches are also under development in pea in several programs involving crosses with different donors of resistance to its main pests and diseases. Exploitation of this pea multi-parental population is expected to allow an important step forward toward understanding the genetic makeup controlling disease resistance and identifying molecular markers readily applicable for MAS.

Reducing the gap between the responsible gene and the linked molecular marker and their characterization should allow their direct exploitation for resistance breeding. This requires the improvement of genome annotation, which could be gained by integrating transcriptome, proteome, and metabolome atlases [187,188]. Different approaches have been used in pea to identify candidate genes, including microarray, deepSuperSAGE, MACE, and RNASeq [17,19,20,34,78,110]. They could be used to develop functional markers for

MAB. Differently expressed proteins in response to pea pathogens could also be used as markers for resistance breeding [21].

6. Genomic Selection

GS is gaining attention in legume breeding and is supported by the constant decrease in genotyping costs, often below the cost of phenotyping [189]. GS combines genotypes and phenotypes from a training population to predict breeding values in genotyped but not phenotyped individuals by using appropriate statistical models [190]. Similarly, genomic predictions (GP) allow the efficient and quick assessment of the wealth of genetic diversity available in a germplasm collection to identify valuable germplasm accessions. GS has been initiated in pea for a number of agronomic and quality traits [189–193]. Implementation of GS approaches to improve biotic stresses in pea is only initiating, and very few reports are available so far on the development of GS models for disease resistance. Efforts have been made to produce the first GS models for resistance to ascochyta blight [194], bacterial blight [195], or rust [196]. Implementation of GS techniques is expected to steadily increase as genotyping costs decrease.

Implementation of MAS and GS approaches can potentially reduce the time required for selection. Notwithstanding, breeding still requires several generations of backcrosses to stabilize and homogenize the introduced trait(s) of interest. Breeding remains, therefore, a lengthy process that should benefit from a reduction in generation advancement time. In many crops, this has been efficiently reduced through double haploid techniques, but this has proven difficult in legumes. To overcome this limitation, speed breeding protocols allowing 4 to 5 breeding generations per year have been adjusted for pea [197,198] and are steadily implemented in pea breeding programs. Combining speed breeding approaches with other modern breeding and biotechnological techniques such as genome editing, GS, and high-throughput genotyping has great potential to boost the genetic gain toward the development of biotic stress-tolerant cultivars in the near future.

7. Conclusions and Perspectives

Some levels of resistance and associated molecular markers have been identified for many pea pests and diseases. However, in many instances, the identified resistance is incomplete and/or the markers are still too far from the responsible gene to allow precise MAS. In spite of these difficulties, resistant cultivars have been developed by breeding, even succeeding in introducing resistance from wild relatives through sexual hybridization and classical breeding. This process can today be highly facilitated by the adoption of modern genomic breeding tools and speed breeding approaches.

The large set of bi-parental and multi-parental populations segregating for diverse important agronomic traits, individual and consensus genetic maps, high-throughput genotyping tools, TILLING populations, and the whole-genome, transcriptome, and proteome sequences from diverse accessions have significantly enhanced our understanding of disease and pest resistance. More importantly, it will keep facilitating advances in gene discovery and the use of more diverse genetic resources for pea improvement. GWAS and GS, rapidly adopted in pea, coupled with the development of multi-parent populations, will certainly facilitate the identification of resistance gene(s)/QTLs with a small additive effect. All these approaches generated a wealth of data that is promising to improve resistance breeding. However, these data are currently scattered and disconnected. Implementation of advanced bioinformatic analytical tools should allow integration of the results obtained from the different omic platforms and from different studies to refine the list of candidate genes. While some attempts toward this have already been made [11], more efforts toward omic result integration would be needed to fill the gap between studies and refine candidate genes. Similarly, functional characterization of these genes to ascertain their involvement in resistance is generally missing. Functional characterization should be tackled urgently to validate these candidate genes before their transfer to elite pea cultivars. Then, speed

breeding (or Rapid Generation) techniques that have been refined for pea should speed up the generation of novel pea cultivars with enhanced resistance in the near future.

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