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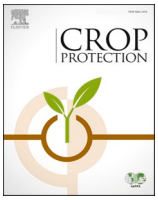

Global dimension of root rot complex in garden pea: Current status and breeding prospective

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ABSTRACT

Crop health is acknowledged as a key component to secure global food security. *Pisum sativum* L. (pea) cultivation is questioned by different biotic, and abiotic stresses. Among them, pea root rot complex (PRRC) is the most prevalent disease caused by soil-borne fungi, and it is usually considered the limiting factor in pea yield (30–57% reduction in yield). PRRC has a diverse spectrum of hosts and may be found all around the globe. The condition stands complex, and numerous approaches such as cultural, physical, biological, and pharmaceutical have failed miserably to control PRRC pathogens. In light of the many tactics and their success, host-plant resistance represents just one realistic choice for controlling the disease in pea agriculture for the long run. It's a strong, low-cost, long-lasting, and environmentally sustainable phenomenon. Plant breeding strategies along with advanced molecular approaches *viz.*, SNP genotyping, gene(s), QTLs mapping, marker assisted selection (MAS), have been led to rise of host-plant-resistance against PRRC. Partial resistance to *Fusarium* spp. is more common in pea genotypes with coloured flowers and seed coatings. MAS can speed up the breeding process by assisting in the genetic selection of targeted traits in early generations. QTLs for resistance to PRRC can be validated using NILs/RILs generated by MAS. In the present manuscript, we review the recent results and breeding strategies for PRRC.

1. Introduction

Garden pea (*Pisum sativum* L.) is an important nitrogen-fixing vegetable crop in the Leguminosae family that grown for its tender green pods, seeds, and leaves all over the world. The garden pea originates in the Central Asian, Mediterranean, and Ethiopian regions. Vavilov (1928) regarded Central Asia and North East are the primary and secondary centre of origin, respectively. Garden pea forms a significant element of sustainable cropping systems because of its atmospheric nitrogen-fixing characteristics, which help to preserve soil health and output (Sharma et al., 2020). Its seeds serve as a protein-rich (15.8–32.1%) food for humans (Al Bari et al., 2021) and also serve as a source of starch (18.6–54.1%), oil (0.6–5.5%), soluble sugars (5%), antioxidants, anti-inflammatory agents, vitamins A, B, E, K and C (Rana et al., 2021), omega-3 fatty acid and omega-6 fatty acid (Arnoldi et al., 2015). Peas are high in potassium, calcium, folate, and digestible fibres (5.9%–12.7%), all of which support gut health, provide cardiovascular aids and help prevent some malignancies (Mudryj et al., 2014).

On the global level, the area under garden pea is 2.18 million hectares with a production of 21.77 million tonnes and productivity of around 9.99 mt/ha (FAO 2019). In India, green peas are cultivated over an area of about 0.56 million ha with an annual production of 5.66 million tonnes and productivity of 10.11 mt/ha (FAO 2019). India exports excellent amount of peas to Nepal (197.16 MT) followed by UAE (74.36 MT), Bangladesh (62.00 MT), Qatar (36.20 MT) and UK (13.08 MT) (Anonymous 2019). Multiple biotic and abiotic stressors, most notably several soil-borne diseases, pose a threat to garden pea farming (Rubiales and Mikic 2014). Approximately 20 distinct pathogens including *Fusarium solani* f. sp. *pisi* (Fsp), *F. oxysporum* f. sp. *pisi* (Fop), *Aphanomyces euteiches*, *Phoma medicaginis* var. *pinodella* (Pmp), *Rhizoctonia solani*, *Thielaviopsis basicola*, *Hycosphaerella pinodes*, *Pythium ultimum*, *P. aphanidermatum* etc. believed to be connected with pea disease from various parts of the world (Thakur et al., 2016).

In the field, several pea pathogen species coexist and disease syndrome like seed rot, damping-off, seedling blight, yellowing of leaves, wilting, root rot and foot rot, and collectively represent a detrimental

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disease complex and hence, referred as wilt complex disease/pea root rot complex (PRRC) (Thakur et al., 2016; Williamson-Benavides and Dhingra 2021). PRRC has a diverse spectrum of hosts and may be found all around the globe. In India, pea wilt and root rot have been recorded to cause crop losses of up to 93 percent (Sharma et al., 2005). In Himachal Pradesh a province of India, fungal pathogens viz., *Fusarium solani, Sclerotinia sclerotiorum, Fusarium oxysporum, Rhizoctonia solani,* and *Phoma medicaginis* var. *pinodella* have been linked to pea root rot (Kapoor et al., 2006). These are strikes pea plants through soil, wind and water.

The disease is occurring in all pea growing areas at pre-flowering or flowering stage to affect the initial crop stand of pea which ultimately leads to enormous yield losses ranged from 30 to 57% (Bodah et al., 2016). Nevertheless, this varies according on variety, field history, as well as climatic factors. Being soil-borne pathogen, the commonly used management practices like soil treatments, adjustment of planting time, vigorous and high-quality seed, seed treatment with fungicides and crop rotation not much successful (Zitnick-Anderson et al., 2018). As a result, the concept of host plant resistance is efficient, economically sound and long-lasting option for management of fungal diseases of pea. Due to a dearth of consolidated knowledge on root rot of pea, this review paper was written to provide an overview of the various elements of this disease complex.

2. History and distribution

The primary contributors to the PRRC are fungi and oomycetes, while bacteria and viruses are also proven to cause root rot (Bodah 2017; Williamson-Benavides and Dhingra 2021). Interaction among several of these pathogens results in root rot complex, hence the role of a single pathogen remains ambiguous in disease development (Sisic et al., 2017). Depending on the soil microbial population, meteorological circumstances, crop rotation, and other agricultural management measures, the occurrence and development of PRRC varies by year and region (Feng et al., 2010). The root rot of peas has been linked to a complex of more than 20 distinct species (*Fusarium* spp., *Phoma medicaginis*, *Aphanomyces euteiches*, *Pythium* spp., *Rhizoctonia solani*, *Thielaviopsis basicola*, *Hycosphaerella pinodes* etc.) of soil-borne fungal pathogens (Arias-Diaz et al., 2013). Among the pea root rot-complexes, *Fusarium* spp. have been found to a prominent role in disease manifestation and are commonly identified from infected pea roots (Willsey et al., 2018).

In many parts of the world, it is caused mostly by the pathogen *Fusarium solani* f. sp. *pisi* (Bodah et al., 2016). At harvest period, *F. solani* f. sp. *pisi* was found in 85 percent of pea fields and 42 percent of pea plants in a Canadian survey (Tonnberg 2016). In India, Maheshwari et al. (1981a) and Sharma et al. (1989) found *F. oxysporium* f.sp. *p151* and *F. solani* f. sp. *p151* on pea crops, with disease prevalence varying from 19.57 to 37.39 percent. Oomycete pathogen, *A. euteiches* was reported as a vastly pathogenic on pea vegetable by Kotova (1979); Abbo and Irwin (1990) in Russia and Australia, respectively. From the Southern part of Pakistan, *R. solani* as well as *Fusarium* species have been stated as the infecting organism of pea root rot by Ehteshamul-Haque and Ghaffar (1994). In Japan, *Fusarium* wilt (*Fusarium oxysporum* f. sp. *pisi*) of pea crop was 1st encountered by Matsuzaki et al. (2003) in Aichi Prefecture 2002 year and Shizuoka Prefecture 2003 year. Afterward, the fungus was as well located in Hokkaido Prefecture in 2015 and in Wakayama Prefecture in 2009, 2015 and 2016 (Sakoda et al., 2019). Fifty specimens of *Fusarium oxysporum* f. sp. *pisi* were collected from various locations of Algeria, and four races *viz.,* 1, 2, 5 and 6 have been distinguished so far (Merzoug and Belabid 2017). All the four races viz., 1, 2, 5 and 6 have been found in Europe (Merzoug et al., 2014).

3. Structure and classification of root rot pathogens

Fusarium spp. is primarily involved in causing root rot such as *F. oxysporum*, *F. solani* complexes (Nelson, 1999). *F. solani* is an important agricultural filamentous fungus. Because it comprises 60 phylogenetically different species, this species is classed as *F. solani* species complex (FSSC) (Coleman 2016). *Fusarium oxysporum* f. sp. *pisi* belonging to the family Tubercularіaceae, order Hyphomycetales, Deuteromycotina is a fungal pathogen which causes a devastating disease of peas (Sharma et al., 2018).

Rhizoctonia solani is a soil-born necrotroph from the kingdom Fungi's domain Eukarya (Ajayi-Oyetunde and Bradley 2018). Septate hyphae, multi-nucleate cells, and brown pigmentation are all features of *R. solani*. Based on hyphal incompatibility and host specificity, isolates are divided into 12 anastomosis groups (Anderson 1982). Over 200 *Pythium* spp. is designated, and at least 10 *Pythium* spp. cause damping-off and root rot in several legumes' crops (Beckerman 2011). Pythium belongs to the Chromista or Straminipila (= stramenopiles) kingdom (Kawamura et al., 2005). There are about 100 species of *Phytophthora* spp., the majority of which have been categorised as aggressive plant diseases. They have the potential to inflict significant losses in agricultural as well as horticultural crops (Hardham and Blackman 2010). The hyphae with a small constriction at the base of its branches may easily identify a species. *Aphanomyces euteiches* belongs to the Chromista/Straminipila kingdom of eukaryotic protists, class: oomycetes, order Saprolegniales, family Saprolegniaceae or Leptolegniaceae (Gaulin et al., 2007). *Aphanomyces euteiches* stands a diploid and homothallic pathogen that generates both ooospores and zoospores, and may finish its sexual cycle without the presence of another individual (Zitnick-Anderson et al., 2021).

4. Symptoms and disease incidence

This devastating disease of pea spread via infected soil and seed (Tonnberg 2016). It infects the developing pea plant and damaged seed, root, seedling etc. (Kraft 2001; Gunawardena et al., 2005). Stunting growth, chlorosis, and necrosis are among of the disease's visible signs above ground. Even when badly affected, plants growing in soils with good structure and adequate water supply may not always show above ground signs of root rot diseases (Bodker et al., 1993). Fsp generates chlamydospores as resting structures. These propagules can be found in soils that are naturally infected (Tonnberg 2016). Poor seed vigour and any condition that decreases root growth can increase root damage. High soil moisture favour development of disease symptoms (Tonnberg 2016). Table 1 shows the favourable environmental conditions for various PRRC species. Root damage can also be exacerbated by inadequate seed vigour and any situation that inhibits root development, such as soil compaction (Kraft and Boge 2001), severe temperatures, pH levels, and biotic and/or chemical harm (Bodah et al., 2016). A thorough survey and surveillance on the pea root rot/wilt complex was done in 2014–2015 (Thakur et al., 2016) in Himachal Pradesh, India wherein pea root rot/wilt complex is observed to be a rising problem in pea-growing districts. The percent disease incidence varied greatly among the state's pea-growing regions e.g., in agroclimatic Zone IV (high hill dry-temperate), the disease incidence ranged from 32 to 57.7% while it was 17.7–197% in Zone III (high hill humid temperate), and 22–35.3% in Zone II (mid hill temperate).

5. Detection of the pathogen

Since there are no commercially feasible methods for treating plants that have been infected by root rot pathogens in the field. The management focuses on prevention and diagnosis. Early detection of the causative organism (pathogen) will aid in the planning of future management options. *Fusarium* root rot of field pea causes above-ground leaf yellowing and stunting, which may not be noticeable until the plants start to blossom. Root rots caused by other soilborne pathogens can readily be mistaken for expanding black to brown root lesions (Porter et al., 2015). Placing pathogens on solid growth medium, classical PCR, and real-time quantitative PCR (qPCR) are all methods for detecting and

Table 1
Favourable conditions for different PRRC species.

Disease name	Species	Temperature	Moisture	Remark
Rhizoctonia root rot	*Rhizoctonia solani*	25 °C to30 °C	Moderate to high soil moisture conditions	Survives in the soil or crop debris and may persist for several years
Aphanomyces root rot	*Aphanomyces euteiches* Drechs.	22 °C to 27 °C	Excessive soil moisture	When a chilly, moist spring is followed by an early, hot, dry summer, the situation becomes much more critical
Root rot	*Pythium ultimum*	17 °C to 23 °C	High soil moisture	At every soil temperature or moisture level, peas planted in soil infected with both *Fusarium* and *Pythium ultimum* are more severely attacked than plants infected with only one pathogen
Fusarium root rot	*Fusarium* spp.	26 °C to 27 °C	Moderate soil moistures	

identifying plant pathogens in damaged plant tissues (Zitnick-Anderson et al., 2018). For the development of fungal structures, direct plating of damaged root tissues requires ideal incubation conditions and the proper growth media. It takes many weeks to identify *Fusarium* spp. morphologically, and reliable identification of these pathogens is challenging because numerous *Fusarium* spp. can invade field pea roots, and hence, accurately representing the pathogen complex can be difficult (Zitnick-Anderson et al., 2018). Standard PCR can be used to identify species, it cannot be employed to accurately quantify each species present in a sample. Direct plating and classical PCR procedures are less efficient, sensitive, and specific than qPCR tests, which can be utilised to swiftly quantify multiple targets within a sample. The creation of viable multiplex qPCR tests using DNA isolates from field pea root tissues would significantly minimise the time and skill necessary to detect and quantify root rot causing pathogen (Zitnick-Anderson et al., 2018).

6. Host range and transmission

Root rot complexes cause economically important diseases of monocots and dicots, cereals, oilseed and pulse crops, vegetables such as tubers, dianthus, tomatoes, asparagus etc, fruit trees, ornamental and some weed species. Surprisingly, legumes appear to be the pathogens' most prevalent host (Williamson-Benavides and Dhingra, 2021). The hosts range along with distribution of PRRC are depicted in Table 2. All root-rotting fungi are "soil inhabitants" and once introduced will persist in the soil for more than 6 years in the absence of a pea crop/host crop. The resting stage (oospores/chlamydospores/macroconidia) are highly resistant to adverse environmental conditions. This disease's spores/inoculum (PRRC) are easily spread over long distances by surface-drainage and splashing water, soil movement from one place to another, and contaminated seed.

7. Management of PRRC in garden pea

7.1. Integrated disease management strategy

Since the disease is soil-borne and involved more than one pathogen, it has been difficult to manage via single strategy. Effective management

Table 2
Stated hosts range along with distribution of pea root rot complexes (PRRC).

Pathogen species	Host range	Geographic distribution	References	Remark
F. solani f. sp. *pisi*	Alfalfa, chickpea, soybean common bean, cottonwood, ginseng, mulberry, lentil, red clover, sainfoin, ryegrass (*Lolium* spp.), potato, mulberry tree and tulip tree	Worldwide	Miller et al. (1980); Smith et al. (2009); Porter et al. (2014); Porter et al. (2015); Sisic et al. (2018); Safarieskandari et al. (2021).	Broad host range, moderate levels of resistance
Phytophthora sansomeana Hansen et Reeser	Douglas fir (*Pseudotsuga. menziesii*), soybean (*Glycine max* L.), white clover (*Trifolium repens* L.), and weeds like wild carrot (*Daucus carota* L.) and white cockle (*Silene latifolia* Poir. ssp. alba) Greuter and Burdet	America, Alberta, Canada	Hacker et al. (2005); Chang et al. (2017)	–
Aphanomyces euteiches Drechs.	*Medicago truncatula*, lentil (*Lens culinaris*), Faba bean (*Vicia faba*), red clover (*Trifolium pratense*), white clover (*Trifolium repens*),	In Asia, Europe, Oceania, and North America	Hagedorn (1984); Persson et al. (1997); Malvick and Percich (1998); Wicker et al. (2003)	High partial resistance
Fusarium avenaceum	With almost 200 species, it has a wide range, consist of pulses, alfalfa (*Medicago truncatula*), cereals, canola, and flax (*Linum* spp.).	Worldwide	Feng et al. (2010); Chittem et al. (2010); Li et al. (2012); Leach and Hobbs (2013); Lysoe et al. (2014); Eranthodi et al. (2020); Safarieskandari et al. (2021)	Moderate resistance
Rhizoctonia solani	Infecting large number of species such as legumes, cereals, pulses, vegetables like potato, cabbage, carrot, lettuce, onion, potato, tomato.	Worldwide	Keinath and Farnham (1997); Yanar et al. (2005); Hwang et al. (2007); Yanga et al. (2007); Taheri et al. (2012)	Moderate resistance

of PRRC is possible by utilizing the principal and strategies of Integrated Disease Management through cultural, chemical (Table 3a) and biological (Table 3b) methods. The first step in the management of root diseases is the use of high-quality and pathogen free seeds. Selection of suitable cropping sequences *viz*; crop rotation with non-leguminous crops like rice-pea and maize-pea and cover crops (Brassicaceae plants) affect the soil populations of root pathogens directly or indirectly (Kapoor et al., 2006; Bodah, 2017). The development of fungicide-resistant pathogenic strains and increase use of pesticides have an adverse effect on soil, plant health, and crop production (Hamid et al., 2012). Biological control of plant pathogens involving microorganisms and natural antagonists have been considered as more natural and environmentally acceptable. Strains of *Trichoderma* spp., *Pseudomonas* spp., and *Bacillus* spp. have been reported in controlling plant infections and also supplement plants in acquiring nutrients (Urkude, 2021). *Trichoderma* spp. also promotes plant defence mechanisms and improves plant and root growth.

7.2. Resistant sources in garden pea

The adoption of disease-resistant cultivars is universally acknowledged as the safest, most cost-efficient, and most successful form of disease protection (Feng et al., 2010). The Western Regional Plant Introduction Station, Washington State University in Pullman, has assessed the *Pisum* germplasm collection for resistance to numerous soilborne plant diseases, including *Fusarium* wilt races 1 and 2 (*F. oxysporum f. sp. pisi*) and Aphanomyces root rot (*Aphanomyces euteiches Drechs.*) (Grunwald et al., 2003). The primary gene pool (*Pisum sativum*) and secondary gene pool (*Pisum sativum* subsp. *elatius* and *P. fulvum*) accessions having the potential sources of root rot complex resistance (Kosterin 2016; Jha et al., 2021). Wild peas are serve as potential sources of disease resistance, however, the use of wild peas is limited by their undesirable agronomic traits, linkage drag and reproductive barriers that can result in sterility of the hybrids (Kosterin and Bogdanova 2015). Porter (2010) identified a high level of partial resistance to *F. solani* f. sp. *pisi* in eight wild pea accessions. Partial resistance to root rot complex pathogens like *A. euteiches*, *F. solani*, and *F. oxysporum* were identified in several studies (Jha et al., 2021) and the extent varies depending on the cultivar (Porter 2010). Bodah et al. (2016) reported *F. solani* resistant pea accessions namely PI 125673, 5003, Banner, Carneval, PS 05300234, and Whistler. In general, genotypes with pigmented-flowers, with exception of PI 180693, had lower disease scores than white-flowered genotypes (Jha et al., 2021).

Several sources of resistance have been found in different pea species (Ondrej et al., 2008). Grunwald et al. (2003) evaluated introduced (PI) accessions and found 387 accessions partially resistant to *Fusarium* root rot as well as *Aphanomyces* root rot, which include five taxon namely *Pisum sativum* (361), *P. sativum* subsp. *sativum* (15), *P. sativum* var. *arvense* (2), *P. sativum* subsp. *elatius* (5) and *P. sativum* subsp. *Abyssinicum* (4). The USDA-ARS Pisum core collection accessions were tested for Fusarium root rot resistance, and the findings indicated that 44 PI lines were somewhat resistant, with just a handful remaining with high levels of resistance, particularly PI180693, PI197990, PI505122, PI196877, and PI197450 (Infantino et al., 2006). The National Plant Germplasm System website, USDA-ARS (http://www.ars-grin.gov/npgs/orders), has a comprehensive listing of data on partial resistance of all accessions examined. The sources of resistant germplasm, variety and commercial cultivars of PRRC are presented in Table 4.

Six pea pathogenicity (PEP) genes (Table 5) are grouped on a 1.6 Mb chromosome that is either supernumerary or conditionally dispensable (CD) (Etebu and Osborn 2011). Estimating the disease potential in a

Table 3b
Biological methods to Control Pea root Rot Complex (PRRC).

PRRC Spp.	Biological agents	Remarks	Reference (s)
Fusarium solani f. sp. *Pisi*	*Trichoderma harzianum, T. viride, Gliocladium virens, Pseudomonas fluorescence*	Low disease incidence and severity reported in *T. harzianum* (21.30 & 10.94%) followed by *T. viride* (25.30 & 12.02%), *P. fluorescence* (29.28 & 14.98%) and *G. virens* (38.64 & 17.58%)	Hamid et al. (2012)
Rhizoctonia solani, *Fusarium solani*	*T. harzianum, T. virens, Pseudomonas fluorescence*	Seed bio priming in combination *T. harzianum* + P. *fluorescens,* followed by *T. harzianum* + *P. fluorescens* + *T. virens*	Negi et al. (2014)
Fusarium oxysporum, Rhizoctonia solani, Sclerotinia sclerotiorum	*Trichoderma harzianum, Rhizobium* sp, *Bacillus subtilis, Thielaviopsis basicola*	Significantly decreased the disease severity also decreased enzyme activity involved in pathogenesis	Muhanna et al. (2018)
Rhizoctonia solani, *Fusarium solani*	*Rhizobium leguminosarum, Trichoderma lignorum, T. longibrachiatum, T. koningii*	Combination of *R. leguminosarum* + *T. longibrachiatum* reduced the root rot by 9.58% whereas combination of *R. leguminosarum* + *T. koningii* against *F. solani* reduced up-to root rot 13.06%	Ketta and Hewedy, (2021)

Table 3a
Integrated disease management techniques to control pea root rot complex.

Crop	Root rot complex Spp.	Operation/treatment	Reference(s)
Cultural methods	*Aphanomyces euteiches*	Soil amendments of cabbage leaves and stems	Papavizas, et al. (1966)
	Fusarium oxysporium, Fusarium solani	Proper sowing method, sowing time, irrigation and balanced fertilizer application essentially phosphorous	Maheshwari et al. (1981b)
	Aphanomyces euteiches	Crop rotation with white mustard (Sinapis alba) and crucifer green manures	Muehlchen et al. (1990)
	Aphanomyces euteiches	Green manures of oat, rape and sweet corn	Williams-Woodward et al. (1997)
Chemical methods	*R. solani, F. solani, Aphanomyces euteiches*	Benomyl	Harper (1968)
	R. solani	Treatment of pea seed and soil with benomyl.	Powell (1988)
	Thielaviopsis basicola	Benomyl-Dexon combination	Blume and Harman (1979)
	F. oxysporium and *F. solani*	Beomyl and derosal	Wahid et al. (1995)
	Aphanomyces euteiches, *Fusarium oxysporium* f.sp. *pisi*, *F. solani* f.sp. *pisi* and *Rhizoctonia solani*	Drenching with benlate, ridomil and captan	Khan et al. (1998)
	Fusarium oxysporum f. sp. *pisi*, *F. solani* f. sp. *pisi* and *Rhizoctonia solani*	Seed treatment with carbendazim (@ 2.5 g/kg) followed by spray of triadimefon (@ 0.4 g/L)	Kumar (2013)
	Aphanomyces euteiches	Seed treatments with Apron Advance (thiabendazole + fludioxonil + metalaxyl) + Vibrance (difenoconazole + metalaxyl- M + sedaxane)	Wu et al. (2018)

Table 4
Sources of resistant germplasm, variety and commercial cultivars of PRRC.

Name	Accession	Resistance	Status	Reference
Franklin	PI 628275	*Fw, Frr*	Cultivar	McPhee and Muehlbauer (2002b)
Novella II	PI 600875	*Fw, En*	Cultivar	Ondrej et al. (2008)
Freezer	PI 596702	-	Cultivar	Ondrej et al. (2008)
Almoto	W6 175 35	-	Cultivar	Ondrej et al. (2008)
MN 144	W6 26201	*Aphb, Fnwc*	Germplasm	Davis et al. (1995)
Wis 8903	PI 538357	*Aph, Fw*	Germplasm	Gritton (1990)
OSU 667	W6 175 23	-	Germplasm	Ondrej et al. (2008)
LPKE 36	-	-	Germplasm	Ondrej et al. (2008)
Herold	-	-	Cultivar	Ondrej et al. (2008)
Kamelot	-	-	Cultivar	Ondrej et al. (2008)
Gotik	-	*Fw*	Cultivar	Ondrej et al. (2008)
Sponzor	-	-	Cultivar	Ondrej et al. (2008)
Hardy	-	-	Cultivar	Ondrej et al. (2008)
Zekon	-	-	Cultivar	Ondrej et al. (2008)
Garde	-	-	Cultivar	Ondrej et al. (2008)
Sommerwood	-	-	Cultivar	Ondrej et al. (2008)
Dakota	-	-	Cultivar	Ondrej et al. (2008)
B 99/118	-	*Fw, Fnw*	Germplasm	Ondrej et al. (2008)
Midget	PI 600965	-	Cultivar	Ondrej et al. (2008)
G 9174	PI 257593	*Frr*	Germplasm	Ondrej et al. (2008)
Hohenheimer Pink Flowered	PI 180693	-	Germplasm	Ondrej et al. (2008)
Line 260	PI 600942	-	Germplasm	Ondrej et al. (2008)
Joel	PI 619080	*Fw, Frr,*	Cultivar	Ondrej et al. (2008)
WR-1167	PI 618635	*Fw*	Germplasm	Ondrej et al. (2008)
Roi des Conserves	PI 244222	-	Germplasm	Ondrej et al. (2008)
Lincoln	PI 250447	-	Germplasm	Ondrej et al. (2008)
WSU 23	CSR 200	*Fw, Fnw, Fwf*	Germplasm	Ondrej et al. (2008)
B 99/103	-	*Fw, Fnw*	Germplasm	Ondrej et al. (2008)
Gypsy	PI 595575	*Fw*	Cultivar	Ondrej et al. (2008)
Duke	PI 600765	*Fw*	Cultivar	Ondrej et al. (2008)
Capella (tolerant)	–	-	Variety	Ondrej et al. (2008)
Wis 8905	PI 538359	*Aph, Fw*	Germplasm	Gritton (1990)
MN 314	W6 26203	*Aph, Fnw*	Germplasm	Davis et al. (1995)
Wis 8904	PI 538358	*Aph, Fw*	Germplasm	Gritton (1990)
MN 313	W6 26202	*Aph, Fnw*	Germplasm	Davis et al. (1995)
Wis 8902	PI 538356	*Aph, Fw*	Germplasm	Gritton (1990)
Wis 8901	PI 538355	*Aph, Fw*[d]	Germplasm	Gritton (1990)
96–2052	PI 606694	*Aph, Frrf, Fw, Fnw*	Germplasm	Kraft and Coffman (2000a)
96–2058	PI 606695		Germplasm	
		Aph, Frrf, Fw, Fnw		Kraft and Coffman (2000a)
90–2068	PI 606696	*Aph, Frrf, Fw, Fnw*	Germplasm	Kraft and Coffman (2000a)
96–2198	PI 606697	*Aph, Frrf, Fw, Fnw*	Germplasm	Kraft and Coffman (2000a)
96–2222	PI 606698	*Aph, Frrf, Fw, Fnw*	Germplasm	Kraft and Coffman (2000a)
97–261	PI 606702	*Aph, Frr, Fw, Fnw, Fwfg*	Germplasm	Kraft and Coffman (2000b)
97–2154	PI 606703	*Aph, Frr, Fw, Fnw, Fwf*	Germplasm	Kraft and Coffman (2000b)
97–263	PI 606699	*Frr, Fw, Fnw, Fwf, Fws*	Germplasm	Kraft and Coffman (2000c)
97–2170	PI 606700	*Aph, Fw, Fnw, Fwf, Fws*	Germplasm	Kraft and Coffman (2000c)
97–2162	PI 606701	*Aph, Fw, Fnw*	Germplasm	Kraft and Coffman (2000c)
Stirling	PI 634571	*Fw*	Cultivar	McPhee and Muehlbauer (2004)
Lifter	PI 628276	*Fw, Frr*	Cultivar	McPhee and Muehlbauer (2002a)
RIL 846-39, RIL 847-28	W6 26742, W6 26746	*Frr, Fw*	Germplasm	Coyne et al. (2006a)
RIL 846-31, RIL 847-28	W6 27367, W6 27368	*Aph, Fw*	Germplasm	Coyne et al. (2006b)

USDA-ARS provides the lines online at http://www.ars-grin.gov/npgs/orders.html.
[b]*Aph*: enhanced Aphanomyces resistance.
[c]*Fnw*: Fusarium near wilt resistance, race 2.
[d]*Frr*: Fusarium root rot resistance.
[g]*Fwf*: Fusarium wilt race resistance.
[d] *Fw*: Fusarium wilt resistance, race 1.

Table 5
List of pea pathogenicity genes along with primers.

Pea pathogenicity genes	Root rot complex gene	Primer pair	Reference
PDA	PDA	PDAF2 PDAR2	Etebu and Osborn (2009); Tonnberg (2016)
PEP1		PEP1F1 PEP1R1	Etebu and Osborn (2009)
PEP2		PEP2F1 PEP2R1	Etebu and Osborn (2011)
PEP3	PEP3	PEP3F1 PEP3R1	Etebu and Osborn (2009); Tonnberg (2016)
PEP4		PEP4F1 PEP4R1	Etebu and Osborn (2011)
PEP5		PEP5F2 PEP5R2	Etebu and Osborn (2009)

field before planting might help farmers avoid badly infected crops and productivity losses. It was, however, unable to distinguish between the contributions of the most frequent diseases (*F. solani f. sp. pisi, A. euteiches,* and *T. basicola*). The quantity of virulence gene copies of PDA1 and PEP3 in soil samples has been used to try to establish a molecular prognostic approach. PDA1 and PEP3 appeared to be crucial for high virulence in root rot complexes when they were combined (Tonnberg 2016). Candidate gene approaches and comparative mapping of the

model legumes *Medicago truncatula* and pea (Choi et al., 2004a, b), *Medicago sativa* and pea, and *M. truncatula* and pea, chickpea, and faba bean can help us better understand host–pathogen relationships (Gutierrez et al., 2005). 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 14–91 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.

Once the candidate genes have been identified, primers can be built to provide polymorphic 'ideal markers' for the resistance genes, which can then be utilised in marker assisted breeding (Infantino et al., 2006). These are beginning to reveal details about how resistance is acquired at the molecular level (Colditz et al., 2004). The involvement of the candidate gene DDR206 (PI 206) in resistance is being investigated, and it is most likely a key regulator of quantitative resistance known to operate in pea resistance to *Fusarium* root rot and *Aphanomyces* root rot (Choi et al., 2004a, b). Another *F. solani* response gene DRR230 defensin family (PI230, PI39) was discovered (Lai et al., 2002). Understanding of pathogen genes implicated in pea infection by *F. solani*, such as an extracellular lipase (Eddine et al., 2001) and the ability to detoxify the phytoalexin pisatin, has also progressed (Temporini and VanEtten, 2004). Many candidate genes for resistance to soil dwelling fungal infections that harm pea have been identified and reported. Primers can be built to provide polymorphic 'ideal markers' for the resistance genes, which can then be utilised in marker assisted breeding.

7.3. Inheritance of root rot disease complex

The root rot disease complex has been linked to a number of pathogens. As a consequence, different inheritance pathways for root rot disease complex resistance in pea have been reported. Due to their complex and incomplete genome information, polygenic inheritance of resistance, and problems in field-based phenotyping, determining the genetic basis of resistance in pea species has proven difficult (Williamson-Benavides and Dhingra 2021). Muehlbauer and Kraft (1973) were the first to report on pea resistance to Fusarium root rot genetics, which was later confirmed as quantitatively inherited i.e., controlled by many genes (Mukankusi et al., 2011). Inheritance studies also revealed the quantitative character of genetic resistance to *A. euteiches* and *F. solani*, as well as the role of additive gene action (Shehata et al., 1983). The surrogate model used to investigate the molecular interactions and resistance mechanism against *A. euteiches* was *Medicago truncatula*, which has a much simpler genome (Williamson-Benavides and Dhingra 2021). Single dominant genes that are inherited separately provide resistance to races 1, 2, 5, and 6 of *F. oxysporum* f. sp. *pisi* (Kraft and Pfleger 2001) and are present in numerous releases of germplasm (Kraft et al., 1998).

7.4. Applications of genomic tools against root rot complex disease in pea

Host plant resistance (HPR) to root rot pathogens has been improved using traditional breeding approaches. The measures necessary in traditional breeding, such as several crosses, a large progeny population, rigorous phenotyping at multiple stages to select for disease resistance, and avoiding unwanted traits, are frequently hampered by the dynamic production and marketing requirements. Marker-assisted selection (MAS) can speed up the breeding process by assisting in the genetic selection of some or all features in early generations. MAS in resistant breeding to generate cultivars with better disease resistance is made easier and faster with the use of molecular markers (Table 6 and 7). Molecular markers were used to release eight green pea germplasm lines having partial resistance to *A. euteiches,* derived from a F_8 RIL population ('Dark Skin Perfection' 90–2131) by the USDA-ARS, the French National Institute for Agricultural Research (INRA), and North Dakota State University (McGee et al., 2012). Near-isogenic lines (NILs) generated by marker-assisted backcrossing were used to validate QTLs for partial resistance to *Aphanomyces* root rot (Lavaud et al., 2015). Likewise, using a RIL population derived from a cross between Puget (susceptible) and 90–2079 (partially resistant), Pilet-Nayel et al. (2002) identified seven QTLs for root rot resistance.

Table 6
Genomic regions and/or markers associated with root rot resistance.

Population	Mapping population	Population size	Total identified QTLs	Main QTLs	Linkage group	Flanking markers	Reference(s)
Aphanomyces euteiches							
Puget × 90-2079	RILs	127	7	Aph1	4b	E7M4.251, N14.950, U326.190	Pilet-Nayel et al. (2002)
Puget × 90-2079	RILs	127	10	Aph1	4b	U326.190, E7M4.25	Pilet-Nayel et al. (2005)
Baccara × PI180693	RILs	178	75 (additive-effect)	Ae-Ps3.1	3	X03_1000	Hamon et al. (2011)
Baccara × 552	RILs	178	60 (additive-effect)	Ae-Ps7. 6b	7	AA176	
Puget × 90-2079	RILs	127	27 meta-QTLs,	MQTL-Ae25, MQTL-Ae26	7	IJB174	Hamon et al. (2013)
Baccara × PI180693	RILs	178	318 candidate			AB122b	
Baccara × 552	RILs	178	genes				
Dark Skin Perfection × 90-2131	RILs	111					
Pea lines	RILs	175	52	Ae-Ps4.4–4.5, Ae-Ps7.6	4, 7	AA122 AA387, AB101	Desgroux et al. (2016)
Pea lines	RILs	266	11 genomic intervals	Ae-Ps7.6	7	Ps115429	Desgroux et al. (2018)
Fusarium solani							
JI 1794 × Slow	RILs	51	4		4(1)	Not reported	Hance et al. (2004)
CMG × PI220174	–	–	3		2(1)	Not reported	Weeden and Porter (2007)
Carman × Reward	RILs	71	1		7(1)	AA416, AB60	Feng et al. (2011)
Dark Skin Perfection × 90-2131	RILs	111	5	FspPs2.1	2a	Thiol (gene-based marker)	Coyne et al. (2015)
Baccara × PI180693	RILs	178	3	Fsp-Ps2.1	2a		
F. avenaceum							
Carman × Reward	RILs	161	1		7	AA160, AD53	Li et al. (2012)

Table 7
Fusarium root rot resistant microsatellite markers specific to single locus.

S. No.	Markers	Chromosome (Location)	S. No.	Markers	Chromosome (Location)
1	AA160	VII	8	AD270	III
2	AA399	VII	9	AD53	VII
3	AA416	VII	10	AD57	III
4	AA467	VII	11	AD61	IV
5	AA5	III	12	B14	VII
6	AB33	VII	13	B16	VII
7	AB60	VII	14	B17	VII

Germplasm is a great resource for discovering new sources (QTLs) of root rot pathogen resistance (Muehlbauer 1992) (Table 6). To understand the inheritance of Aphanomyces root rot resistance, Pilet-Nayel et al. (2002) identified QTLs associated with field partial resistance. For that purpose they used RILs (127 lines) derived from a cross between Puget (susceptible) × 90–2079 (partially resistant). Seven QTLs were associated with *Aphanomyces euteiches* resistance, a major-effect locus located on LG IVb, *Aph1*, was highly and consistently detected over years, locations and field tolerance. Two other QTLs, namely *Aph2* (mapped near to *r*; wrinkled/round seed) on LG V and *Aph3* (*af*; afila/normal leaves) on LG Ia, were also consistently identified. In a subsequent study, Pilet-Nayel et al. (2005) validated above seven QTLs in the same mapping population (127 RILs) against two other pathogen isolates, SP7 (US) and Ae106 (France), under two screening conditions i.e. field and greenhouse, and suggested the adequacy of three consistent QTLs, *Aph1*, *Aph2*, and *Aph3*, for MAS.

Hance et al. (2004) used RIL derived from the cross 'JI 1794' (*Pisum sativum* ssp. *elatius* var. *pumilo*) × Slow (*Pisum sativum* ssp. *sativum*) to identify tolerance to *F. solani* f. sp. *pisi* in pea. Four QTLs on linkage map were identified, among these, two QTLs on LG III near to *M* (brown mottle on testa) and *Le* (tall height), one on LG IV adjoining to ribosomal array, and one on LG VI outlying to *Gty* (gritty seed coat) were located. Three QTLs on LG II, LG IV and LG VII were identified that effect tolerance to fusarium root rot (Weeden and Porter 2007). They were used RIL population of pea developed from a cross between CMG × PI 220174. The QTL on LG II was highly influenced the tolerance to *Fusarium* spp., and it overlapped with *A* locus (pigmented flower). QTL on chromosome IV only had an impact if the QTL on chromosome II was also present, suggesting that this QTL is engaged in a metabolic process farther down the line. Because the QTL region on chromosome II corresponded with the location of the *A* allele locus, the scientists speculated that the influence of the QTL was due to the *A* gene, which is involved in the anthocyanin/polyphenol/flavonoid pathway. Anthocyanin may have a role in Fusarium root rot resistance. Alike, one QTL on LG VII for resistance to Fusarium root rot was identified by Feng et al. (2011) in a RIL population (71 lines) derived from a cross of Carman (R) × Reward (S). A total of 213 SSRs were used to screen the parental lines. Among these 14 markers were polymorphic between the parents and were utilised for genotyping of RILs. The identified QTL was flanked by SSR markers namely 'AA416' and 'AB60'. Therefore, these markers could be used for MAS to develop varieties with exalted Fusarium root rot resistance.

A total of 111 RIL population of cross 'Dark Skin Perfection' (DSP) (W6 17516) × '90–2131' (PI 557501) were screened for *Fusarium solani* f. sp. *pisi* disease, Coyne et al. (2015) identified five QTLs accompanied one multiyear QTL *Fsp-Ps2.1*, with highest phenotypic variance i.e. R^2 = 22.1–72.2%, controlling genetic partial resistance to Fusarium disease. The QTL namely, *Fsp-Ps2.1* (LGII), *Fsp-Ps3.1* (LGIII), *Fsp-4.1* (LGIV) and *Fsp-Ps7.1* (LGVII) were flanked by co-dominant SSR markers namely, 'Thiol', 'PSAB68', 'AD53a′, 'PSAB122b′, respectively and can be used in marker assisted backcross breeding (MABB) of pea for great levels of partial resistance to disease. In another study, Coyne et al. (2019) used a RIL population (178 individuals) of cross Baccara × PI 180693 to determine QTL associated with good levels of partial-resistance to Fusarium root rot under controlled/greenhouse conditions. Three significant resistance QTLs were identified; among them one QTL i.e. '*Fsp-Ps 2.1*′ was strongest with high LOD value (25.3–32.4) and explained 44.4–53.4% of the variance for resistance. The next QTL named '*Fsp-Ps3.2*′ and '*Fsp-Ps3.3*′ were closely linked and explained 3.6–4.6% of the variance. All of the alleles are contributed by the resistant parent PI 180693. With the confirmation of important QTL '*Fsp-Ps 2.1*′ in two RIL populations, SNPs associated with this region/QTL make a good target for MAS in *Pisum sativum* breeding programs to obtain high levels of partial resistance to Fusarium root rot.

Partial resistances, often controlled by QTL, are believed to be more durable than the monogenic resistances. A consensus genetic map was developed by Hamon et al. (2011) using the two segregating populations Baccara × PI 180693 and Baccara × 552 (a total 178 RIL lines each) to identify QTL for *Aphanomyces euteiches* disease resistance. Five QTLs namely, *Ae-Ps1.2*, *Ae-Ps2.2*, *Ae-Ps3.1*, *Ae-Ps4.1* and *Ae-Ps7.6* were more stable, as they comprise resistance alleles covering a broad spectrum of environments and/or pathotypes and resistance criteria. Among them *Ae-Ps7.6* was the most consistent QTL. Simultaneously, to identify QTLs controlling resistance to *Fusarium avenaceaum*, 213 SSR were screened on 161 RILs developed by the crossing Carman (resistance) × Reward (susceptible) (Li et al., 2012). A QTL on distal end of LGVII was identified with the markers 'AA160' and 'AD53'.

A meta-analysis performed using four mapping populations, Puget × 90–2079, Baccara × PI180693, Baccara × 552 and DSP × 90–2131, and identified seven meta-QTLs i.e., *MQTL-Ae3* on LGI, *MQTL-Ae5* on LGII, *MQTL-Ae8/MQTL-Ae9* on LGIII, *MQTL-Ae12* and *MQTL-Ae15* on LGIV, *MQTL-Ae16/MQTL-Ae17* on LGV and *MQTL-Ae25/MQTL-Ae26* on LGVII, resistance to *Aphanomyces euteiches* (Hamon et al., 2013).

QTLs for partial resistance to *Aphanomyces* root rot were validated in different near-isogenic lines (NILs) developed by marker-assisted backcrossing (Lavaud et al., 2015). In controlled conditions, to check the disease resistance of NIL, two single spore strains of *Aphanomyces euteiches* (RB84 strain, pathotype I and Ae109 strain, pathotype III) were used. They observed that NILs which carried resistance alleles at major-effect QTL (*Ae-Ps4.5* and *Ae-Ps7.6*) and minor-effect QTL (*Ae-Ps2.2* and *Ae-Ps5.1*), either singly or combined have less disease incidence as compared to NILs without resistance alleles. For *Ae-Ps7.6*, QTL × genetic background interactions were also recorded. In the next research, Lavaud et al. (2016) studied the impact of single or multiple resistance QTL on delaying symptom appearance and/or slowing down root rot colonization by *Aphanomyces euteiches*. The combination of resistance alleles at two or three QTL including the major QTL *Ae-Ps7.6* (*Ae-Ps5.1/Ae-Ps7.6* or *Ae-Ps2.2/Ae-Ps3.1/Ae-Ps7.6*) had reduced root colonization by *Aphanomyces euteiches*, simultaneously delayed disease symptom appearance. NILs carrying QTL Ae-Ps7.6 were also identified for varying levels of resistance against 43 *Aphanomyces euteiches* isolates from different French pea growing areas (Quillevere-Hamard et al., 2021).

Genome-wide association study (GWAS) has recently arise as a most common significant approach for refining the genetic basis of polygenic resistance to major plant diseases, which are increasingly used in integrated strategies for durable crop protection. By using GWAS approach, Desgroux et al. (2016) evaluated 175 pea lines against *Aphanomyces euteiches* in field infested nurseries over nine environments and with two isolates in climatic chambers. They detected total 52 QTLs for disease resistance, and validated six of the seven previously detectedAphanomyces resistance QTLs (Hamon et al., 2011, 2013). The current study also provided marker haplotypes at 14 consistent QTLs associated with disease resistance. In the successive study, a collection of 266 lines were used for GWAS (Desgroux et al., 2018). They demonstrated a total of 75 genomic intervals associated to plant architecture and 11 genomic intervals with resistance to *A. euteiches*. Thereafter, one highly significant SNP marker, namely 'Ps115429', mapped to the major QTL *Ae-Ps7.6* (LG VII), was associated with both the disease resistance and the root system architecture. The SNP marker was located in the same genomic region as

the main QTL *Ae-Ps7.6* formerly identified by marker map (Hamon et al., 2013) and GWAS (Desgroux et al., 2016), the effect of which was validated in NILs (Lavaud et al., 2015). An Illumina GoldenGate test was utilised to genotype five RIL populations using 1536 polymorphic SNP loci picked through over 20,000 non-redundant SNPs discovered utilizing deep transcriptome sequencing of eight different Pisum accessions (Sindhu et al., 2014).

To identify the *Fusarium solani* f. sp. *pisi* responsive genes in pea, Williamson-Benavides et al. (2020) performed time-course transcriptome analysis on eight white flowered genotypes (four tolerant and four susceptible) after inoculation with *Fusarium solani* f. sp. *pisi*. In the tolerant pea genotypes, genes involved in exocytosis, secretion by cell, anthocyanin synthesis pathway, a pathogenesis-related (PR) gene 'DRR230', were overexpressed. In the next study, Williamson-Benavides et al. (2021) utilised SNPs mined from *Fusarium* spp. responsive differentially expressed genes (DEGs) for the identification of QTLs correlated with resistance using two RIL populations consisting of 190 lines each. They identified a total of 769 DEGs with SNPs. One QTL named '*WB. Fsp-Ps* 5.1′ on LG V explained 14.8% of the variance. The use of SNPs derived from *Fusarium solani* f. sp. *pisi* responsive DEGs for QTL mapping proved to be an efficient way to identify molecular markers associated with *Fusarium solani* f. sp. *pisi* resistance in *Pisum sativum*.

8. Conclusion and future prospects

The PRRC is a serious soil-borne disease that has spread all over the world's, causing massive crop losses. Because this disease is soil-borne and can survive in the soil for more than six years in the absence of a pea crop/host crop, control is extremely challenging. Various control measures have been tried, including physical, cultural, biological, and chemical means, but these are not much effective. In this case, the host plant resistance approach is the only sensible and long-term solution that has no negative environmental consequences. Wild peas are a possible source of disease resistance, but their usage is limited due to their unfavourable agronomic features, linkage drag, and reproductive hurdles, which can lead to hybrid sterility. By assisting in the genetic selection of some or all traits, marker-assisted selection (MAS) can speed up the breeding process. Marker-assisted backcrossing can be used to validate QTLs for partial resistance to root rot complexes. As a result, wild pea species resources must be screened to discover prospective disease-resistant germplasm. Screening techniques must also be enhanced, with a greater reliance on artificial screening methods using various races with a large population of the PRRC pathogen.

Author contributions

A.S. and M.R. drafted/wrote, reviewed and revised the manuscript. A.S. and M.R. contributed equally to this article. P.S., P.K., D.K.J. and R. S.R. reviewed the manuscript. H.L. and A.T. helped in manuscript writing.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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