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Review article

AN OVERVIEW OF RUST (*Uromyces viciae-fabae*) AND POWDERY MILDEW (*Erysiphe polygoni* DC) OF PEA (*Pisum sativum* L.)

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Pea is a self-pollinating, cool season leguminous crop with a diploid chromosome number of 14. Pea is cultivated extensively and because of high protein content, pea is a crop with great significance. However, cultivation of pea gets affected by numerous biotic and abiotic stresses. Fungal diseases such as rust, powdery mildew, fusarium wilt etc. comes under the biotic stresses which are most widespread. Rust and powdery mildew cause major damage to the crop in both tropical and temperate locales of the world. Use of fungicide to control plant diseases is a good approach but excessive use of fungicide can cause environmental pollution and disasters throughout the world and can also built resistance in the pathogens. Therefore, to remove these constraints, disease resistant varieties must be used. Use of resistant varieties is a safe and efficient alternative method to control plant diseases. Breeding for rust and powdery mildew resistance has been started globally and a number of resistant sources have been identified. To introgress resistant gene into commercial varieties of pea, molecular tools must be integrated with conventional breeding techniques. Till date only one linkage map has been generated for rust resistance in pea; while for powdery mildew, three genes have been mapped. Molecular markers linked to these genes can be used in breeding programs of resistance varieties. To improve the efficiency of selection for rust and powdery mildew resistance and enhance varietal development, the integrated approach of genomic resources, effective molecular tools and high resolution phenotyping tools must be used. An overview of pea rust and powdery mildew, pathogen structure, yield losses and breeding techniques implied to control these diseases, is provided in this review article.

Keywords: pea, rust, powdery mildew, molecular markers, resistant genes

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INTRODUCTION

Pea is a diploid (2n=2X=14), predominantly a self-pollinated annual herb with climbing or bush type of growth habit. It has a place with the vegetable family Fabaceae (Leguminaceae). It is a native plant of Central Asia whereas Asia Minor is the secondary centre of origin. Pea contains 7.2 g digestible protein which makes it a prime protein supplying vegetable crop (SIRWAIYA *et al.,* 2018). Pea is cultivated worldwide, and it is an important cool-season grain legume along with chickpea and cultivated lentil (KHAZAEI *et al.,* 2016). Dry peas occupy 34.2% area of the total pulse growing area, i.e. more than 1/3rd area under pulses (EUROSTAT, 2020). Canada is the largest producer of dry peas followed by Russia and China while India is at 4th position in production of dry peas (FAOSTAT, 2020). In India, dry peas are cultivated on an area of 616508 ha with a production of 796735 tonnes (FAOSTAT, 2020). Data of pea cultivation in India is presented in Table 1. Punjab is the fifth largest producer of pea in the country and accounts for 6.7 percent of India's production. In Punjab region of India, pea is the second most important vegetable crop after potato and is grown on an area of 31.3 thousand hectare with an annual production of 315.87 thousand tons (DHALL, 2017). From ecology point of view, pea is a very advantageous crop because it fixes atmospheric nitrogen. Thus it helps in blooming low-input farming system. Also, it is used as a break crop which further reduces the requirement of external inputs (SMYKAL *et al.,* 2012).

Table 1. Statics of pea cultivation in India (Source: FAOSTAT 2020)

Commodity	Area (Ha)	Production (Tonnes)	Productivity (Kg/ha)
Dry pea	616508	796735	12923
Green pea	563000	5703000	101297

In India, productivity of pulses, including pea, is influenced by many factors. Most of the pea cultivating areas has marginal lands with low rainfall which makes crop management difficult. This causes reduction in overall pulse productivity of the country. Fungal, viral and bacterial pathogens are major obstacles in pea production (PANDEY *et al.,* 2009). Fungal diseases such as rust and powdery mildew are widespread diseases of garden pea.

Pea rust can be caused by different pathogens in according to climatic conditions. In temperate regions of the world, pea rust is caused by *Uromyces pisi* (Pers.) Wint. (EMERAN *et al.,* 2005), while in tropical and subtropical regions, *Uromyces fabae* (Pers.) de-Bary is the rust causing fungus (RAI *et al.,* 2011). On the basis of morphology of telia and infection structures, these two species can be differentiated or these two can be differentiated by using internal transcribed spacer (ITS) markers (EMERAN *et al.,* 2005; BARILLI *et al.,* 2006). Aeciospores are infecting structures of *U. fabae* (KUSHWAHA *et al.,* 2006), while in case of *U. pisi*, urediospores are infecting spores (BARILLI *et al.,* 2009). *U. pisi* can cause more than 30% yield losses (EPPO, 2012) as compared to 50% yield losses caused by *U. fabae* (KUSHWAHA *et al.,* 2006). Reproductive stage of pea plant is the most susceptible for *U. fabae* infection (CHAND *et al.,* 2006) under high temperature and high humidity conditions (KUSHWAHA *et al.,* 2006).

Another fungal disease, powdery mildew (*Erysiphe polygoni* DC) is also one of the most devastating diseases of garden pea and causes severe damage throughout the pea cultivating

region. *Erysiphe polygoni* DC is an air borne pathogen (FONDEVILLA and RUBIALES, 2012). The loss due to powdery mildew is proportionate to the disease intensity and varies considerably depending on the stage of plant growth at which disease occurs. Climatic conditions with dry days and cool nights are most favourable for pathogen growth and development. Under dry weather conditions such as low humidity and low night temperature, *E. polygoni* can cause 25 to 50% loss in yield (FONDEVILLA *et al.*, 2007). In pea, powdery mildew usually appear late in the season, reaching at its maximum intensity during pod formation and making it the most critical stage which should not be coincided with the favourable environmental conditions, i.e. dry weather, for disease development (PRASAD and DWIVEDI, 2007).

In this review, basic information about the rust and powdery mildew pathogens has been provided along with the molecular work done to identify resistant genes for rust and powdery mildew.

CAUSAL ORGANISM

Rust

Rust in pea is caused by two species of *Uromyces* viz., *U. pisi* and *U. fabae*. In India, pea rust pathogen (*U. fabae*) was first reported on *Viciafaba* by SYDOW and BUTLER (1906) from Pusa (Bihar). BUTLER (1918) first reported occurrence of *U. fabae* on pea in India. PATEL (1933) reported the same pathogen on sweet pea crop from Poona where it caused severe damage.

Uromyces fabae is autoecious rust. It completes all its growth stages on the pea plant only and no alternate host is required to complete its life cycle. The fungus is heterothallic. On peas, the pycnia occur in small groups associated with the aecia. The aecia are cupulate and 0.3 to 0.4 mm in diameter. The peridium is short and whitish. The aeciospores are round to angular or elliptical with hyaline wall. The wall of aeciospores is verucose. The uredia measures were 14 to 26 µm in diameter. The uredia present a powdery appearance. The urediospores are round to ovate, light brown, echinulate, with 3-4 equatorial germ pores. The telia occur in the same sorus as the uredia. They are black brown to black. The teliospores are subglobose, ovate or elliptic with rounded or flattened apex, which is considerably thickened and appears papillate. These spores are smooth, brown. The wall of the teliospores is 1-2 µm thick at the sides and 5-12 µm at the apex. The pedicel is persistent, yellowish-brown, thick and upto 100 µ long.

In broad beans, rust is seed borne whereas in pea, it is soil borne. Aeciospores are the disease causing spores in pea and they remain in crop debris. The spores survive on weed hosts (*Lathyrus*, *Vicia* etc.) and infect the main crop by blowing through wind (GRUNWALD *et al.*, 2004). Wild hosts are the primary or secondary source of infection.

Environmental factors affect the initiation of infection by *Uromyces fabae* and its development on the pea plant. In plains of India where summer temperatures are very high, teliospores can survive under such temperature conditions. Thus, teliospores cause the disease in pea crop. Aeciospores and uredospores cannot survive under high temperature conditions of the Indian plains. On pea, the infection by *Uromyces fabae* increases with an increase in the duration of leaf wetness up to 24 h but it will not increase further significantly at optimum temperature 20°C (MORE *et al.*, 2020).

Powdery mildew

The causative of pea powdery mildew, *Erysiphepisi* DC ex St. Amans. was recorded for the first time in 1805 in Europe. In India it was reported as early as 1910 (SINGH, 2005). Erysiphaceae are widely distributed throughout the world.

The disease is caused by *Erysiphepisi* DC. *Erysiphepisi* DC. has complex life cycle and is obligate biotroph in nature. The mycelium of *E. pisi* is generally fined, persistent and rarely thick. It is ectophytic i.e. the entire thallus, except the saccatehaustoria in epidermal cells, is present on the surface of the affected parts of the plant. The haustoria develop as outgrowths from lobed swellings (appressoria) on the sides of the hyphae adjacent to the host surface. The haustorium arises as an exceedingly narrow tube from the appressorium, penetrating the cell wall and swelling into a rounded sac in the epidermal cell.

The conidiophores arise vertically from the superficial hyphae on the host surface. Conidiophores are septate and having uninucleate cells. Conidia are formed singly or in short chains. These conidia are ellipsoid to ovate in shape with vacuolated cytoplasm. The ripe conidia fall off quickly and are dispersed by wind.

Late in the season, but not always on pea leaves in the field, the cleistothecia appear. Cleistothecia are sharp, minute black bodies scattered in the superficial mycelium. These sexual fruit bodies are globose. The peridium of the fruit body is composed of distinct polygonal cells and is provided with a number of hypha-like appendages whose number varies from 10 to 30. Usually, 4 to 8 asci are formed in each cleistothecium. They appear to arise from a single point and hence give a fascicled appearance. The asci are ovate to broadly ovate or subglobose, nearly sessile. The asci contain 3-5 (rarely 6) ascospores which are elliptical, hyaline and 1-celled (SINGH *et al.,* 2012).

The pathogen is seed borne. When pods are affected by the mildew, the fungus grows through the pod into the seeds so that the seeds adhere to the pods. In addition, cleistothecia serve as source of primary inoculum for the next season. They develop on dead plant debris. Ascospores developed in these fruit bodies are released by decay of the fruit and blown by wind to lower leaves where they cause infection and produce the primary mass of spores. Secondary spread occurs through these wind-blown conidia. However, SINGH *et al.* (2012) reported that the fungus is present throughout the year in the plains or at different altitudes in the hills in conidial stage and the latter are blown by wind as the source of primary inoculum.

SYMPTOMS

Rust

Uromyces fabae (Pers.) de-Bary upon infection reduce the area for photosynthesis by developing pustules on stems, leaves and sometimes on pods also (BARILLI *et al.,* 2010). Infecting plants produce pseudo flowers, similar to true flowers in colour and shape and also leaves at the top of stem appear in rosette pattern (PFUNDER and ROY, 2000). Many authors have described rust symptoms on pea (BARILLI *et al.,* 2009; BARILLI *et al.,* 2014; KUSHWAHA *et al.,* 2006; KUSHWAHA *et al.,* 2010; CHAND *et al.,* 2006; XUE *et al.,* 2002). In pea plant, early rust symptoms develop on abaxial side of older leaves and form round to oval aecidia. Initially aecidia form creamy white to light yellow to bright orange coloured pustules on the leaf and stem (Fig. 1).

Under high humidity and high temperature conditions, pustules spread on other plant parts also. Aeciospores are the infecting structure of *U. fabae* (Pers.) de-Bary and are released from aecidia and deposited as yellow powder. In case of *U. pisi*, uredial pustules are found on both the sides of leaf and also on stem. Urediospores appear in light brown powdery mass. After aecial or uredial development on plant, telial symptoms appear on plant parts. Teliospores are found mainly on stem and tendril and are produced from the aecial/uredial pustules. Telial infection affects the size and quality of grain. Due to telial infection, grain remains small in size and also grain colour become dull.

Fig. 1. Rust symptoms on pea plant: light yellow pustules on leaves and stem

Powdery mildew

Development of white powdery blotches on leaves is the primary sign of powdery mildew. Upper leaf surface is infected first and then entire leaf is covered with mildew. Due to powdery fungal growth, leaves become chlorotic or necrotic, stems and fruits are also covered with mycelium and fruiting bodies of the fungus. Powdery growth on leaves and stem weaken a plant but do not kill the plant. The lower leaves are the most affected, but the mildew can appear on any above ground part of the plant (FONDEVILLA *et al.*, 2011). At the later stages of disease, powdery spots become larger and thicker, and a large number of spores formed. Whereas rain controls the disease by washing off the spores and making them burst instead of germinating (GHAFOOR and MCPHEE, 2011). Rust disease of pea is most destructive in late sown crops (JHA *et al.*, 2019) or in late maturing cultivars.

YIELD LOSSES

Rust

In the middle of 1980s, rust of pea has been emerged as one of the most serious pathogens of pea and now, it causes heavy crop loss in many parts of world including Europe, North and South America, Asia, Australia and New Zealand. Warm and humid environment favours the growth of disease. Usually, the rust pathogen ceases the biochemical and physiological functions of plant and as pathogens grows on leaves; it also reduces the

photosynthetic activity of plant (EPPO, 2012). When the disease is sever, is affects leaves dry up. Depending on the rust severity, leaves fall down and also the pods do not mature properly which leads to yield losses of more than 30% (BARILLI *et al.,* 2012). Usually, rust cause severe damage in late crops with about 20% reduction in faba bean crop (SAYED *et al.,* 2011). But yield losses can be as high 45 to 50%, if disease appear early in the crop growth period (STODDARD *et al.,* 2010). During 1990s, rust in India was observed in epiphytotic form when rust infection initiated during early season of crop growth, i.e., from the mid of November (SHARMA, 1998). Rust infection during early crop growth stages may cause complete failure of the crop. Thus, as the rust symptoms appear on pea plant, management of rust is a prior requirement for sustainable pea production (DAS *et al.,* 2019).

Powdery mildew

Powdery mildew (*Erysiphe pisi*) is one of the most severe diseases of peas in India and in many other countries (RANA *et al.,* 2013). Powdery mildew pathogen can cause yield losses up to 30% (FONDEVILLA *et al.,* 2007). This pathogen majorly affects the pea crop when it is grown for seed production as powdery mildew is more severe towards the crop maturity. Use of fungicidal sprays can improve the grain yield of susceptible varieties by 48.7% (FONDEVILLA and RUBIALES, 2012). The disease can cause 25–50% yield losses (FONDEVILLA *et al.,* 2007) as it decrease total yield biomass, pods per plant, seeds per pod, plant height and number of nodes (SMITH *et al.,* 2003). The powdery mildew pathogen can also hasten crop maturity, rapidly raising tenderometer values beyond optimal green pea harvesting levels (FALLOON and VILJANEN-ROLLINSON, 2001). Seed quality can also be degraded due to powdery mildew as mildew fungus can cause severe pod infection which leads to seed discolouration. Conidias and fungal debris from heavily infected crops can cause breathing and allergy problems for machinery operators. Late sown crop or late maturing varieties are more susceptible to powdery mildew infection. If the disease appears during early crop growth season, it can cause heavy yield losses (FONDEVILLA and RUBIALES, 2012).

GENETICS OF RESISTANCE

Rust

Several scientists performed several studies to know the genetics of rust resistance in pea and also identified resistant germplasm both in wild and cultivated species of pea (CHAND *et al.,* 2006; BARILLI *et al.,* 2009). Many scientists have reported a single dominant gene that controls the rust resistance in pea (PAL *et al.,* 1979; KATIYAR and RAM, 1987). In some other studies, scientists found more than one gene controlling rust resistance in pea (SINGH and SRIVASTAVA, 1985; KUMAR *et al.,* 1994; CHAND *et al.,* 2006). Many scientists found that the resistance for *Uromyces fabae* in pea is controlled by a single recessive gene (SINGH and RAM, 2001; COX, 1995). Resistant to pea rust is not linked with the flower and stipule base pigmentation (ABUSALEHA *et al.,* 1987). The first report in molecular mapping of pea shows that one major (*Qruf*) and one minor (*Qruf1*) QTLs control the resistance to rust in pea (RAI *et al.,* 2011). These two QTLs are located on LGIV of pea. Another study was conducted using *Pisum fulvum* accession IFPI3260 and results showed that a single QTL, *UP1*, is responsible for resistance to *Uromycespisi* (BARILLI *et al.,* 2010).

Powdery mildew

Various workers have reported inheritance of powdery mildew resistance by a single recessive gene *er1* (SAXENA *et al.*, 1975; VAID and TYAGI, 1997; LIU *et al.*, 2003). Others reported two different recessive genes (*er1* and *er2*) (RAM, 1992; TIWARI *et al.*, 1997, 1998). FONDEVILLA *et al.*, in 2011 reported a new dominant gene in *Pisum fulvum*, *Er3*. Polygenic inheritance is also reported by GUPTA *et al.* (1995). Gene *er1* provide complete and durable resistance under field and controlled environmental conditions, whereas *er2* provides only leaf resistance and also is not durable under heavy disease infection. According to SU *et al.* (2004), during the growth cycle, a combination of gene *er1* and *er2* can enhance resistance. Recently, COBOS *et al.* (2018) located the *Er3* gene in pea LGIV at 0.39 cM downstream of marker AD61. The location of *Er3* gene in the pea map is a first step toward the identification of this gene. Till now, three genes have been mapped on pea genomes which are responsible for powdery mildew resistance in pea (Table 2).

Table 2. Molecular mapping of powdery mildew resistant gene(s)

Gene	Linkage group	Linked marker	Marker type	Distance (cM)	Reference
er1	VI	$PD10_{650}$	SCAR	2.0	TIMMERMAN *et al.*, 1994
er2	III	ScX17_1400	SCAR	2.6	KATOCH *et al.*, 2010
Er3	IV	AD61	SSR	0.39	COBOS *et al.*, 2018

MOLECULAR MARKER ASSISTED BREEDING FOR RESISTANCE

Now-a-days various techniques are used to control different pea disease such as sowing before the main season i.e. early sowing, use of chemical fungicides and sowing of resistant cultivars. Chemical control is effective when protective and systemic fungicides are used to control the diseases. Use of resistant varieties is the most effective, economic and environmentally friendly method of control.

There is a typical requirement for release of an improved commercial variety, development and implementation of new molecular genetic tools that will help in conversion of conventional approaches into genomics-assisted breeding approaches and this conversion will accelerate the release of improved pea cultivars. Molecular tools, including marker-assisted selection, have the potential to accelerate and improve the effectiveness of breeding for disease resistance in pea. For these reasons, many efforts have been made to understand the genetics and genomics of pea, including a focus on understanding the genetic basis of resistance to *U. fabae* and *E. pisi*. Genetic linkage map of pea have been constructed based on a range of molecular genetic marker types such as randomly amplified polymorphic DNAs (RAPDs), simple sequence repeats (SSRs) and single nucleotide polymorphism (SNPs) (LAUCOU *et al.*, 1998; LORIDON *et al.*, 2005; SINDHU *et al.*, 2014). Through the use of these maps, a number of genomic regions controlling rust and powdery mildew resistance can be identified. Earlier, for different molecular studies, 4.3 GB size of pea genome was in use (MACAS *et al.*, 2007). But now, a reference

genome of 3.92 GB (KREPLAK *et al.*, 2019) can be used to identify new genomic regions of disease resistance and molecular markers linked to them.

Rust

Resistance to *Uromyces fabae* in pea is non-hypersensitive type and the disease initiation and development is greatly influenced by environmental conditions. Due to these constraints, selection is difficult for rust resistance. And these situations also are the major limitation to find out genetics of rust resistance in pea. Molecular markers can be used in field for selection of rust resistance plants and this will accelerate the breeding program for rust resistance (VIJAYALAKSHAMI *et al.*, 2005). Linkage between molecular marker and the gene(s), responsible for a particular trait is a pre requisite for use of molecular markers. For example, resistance for a particular disease can be identified by using this approach at initial stages of plant growth.

Till now many linkage maps of pea have been constructed by using morphological, physiological and pigmentation characters (BLIXT, 1974), isozyme data (WEEDENN and MARX, 1987) and also by using the morphological characters, isozyme data and DNA markers together (WEEDENN and WOLKOI, 1990). Three linkage maps in pea has been constructed using different molecular marker technologies viz., RAPD, SSR and SNP (LAUCOU *et al.*, 1998; LORIDON *et al.*, 2005; SINDHU *et al.*, 2014). Molecular marker technology has been used in a number of independent studies to determine the genetics of rust resistance in pea (VIJAYALAKSHAMI *et al.*, 2005; BARILLI *et al.*, 2010; SINGH *et al.*, 2015). Also, many scientists developed molecular markers linked to the rust resistance in pea by using different generations of marker technology (VIJAYALAKSHAMI *et al.*, 2005; SINGH *et al.*, 2015). Till today, only one linkage map of pea rust have been constructed using SSR markers and reported the location of resistant gene on LG VII (RAI *et al.*, 2011). One linkage map for pea rust has been developed with the help of RAPD markers by using two wild pea (*Pisum fulvum* L.) accessions, IFPI3260 (resistant) and IFPI3251 (susceptible) and showed the location of resistant gene on LG VI (BARILLI *et al.*, 2010).

Powdery mildew

Till now only three genes (*er1*, *er2* and *Er3*) have been identified in *Pisum* germplasm for powdery mildew resistance and out of these, only *er1* is most commonly used to incorporate resistance in commercial varieties. Resistance with only one gene can cause occurrence of new races of the pathogen and can lead to a breakdown of the resistance, which is the major hindrance in the expansion of cultivation areas of the pea. This problem can be solved only by using polygenic resistance or combining several major genes which will provide durable resistance against pathogen.

Combination of different resistant genes in a single genotype can provide durable resistance in most of the legumes for different diseases. But only one to three powdery mildew resistant genes have been reported for pea powdery mildew which makes it difficult to attain durable resistance. Marker-assisted breeding technique can be used in which markers linked to resistance genes are selected. The PCR-based markers are most commonly used in marker-assisted breeding because these markers require only a small amount of DNA as template DNA and can be efficiently applied to large populations. Microsatellites and other Co-dominant

markers, which allow the detection of heterozygotes, are preferred. SSR markers or microsatellites are predominantly used because of their ease of use.

All the three genes confirming resistance against *E. pisi* have been sequenced and mapped now. The gene *er1* is mapped on pea LGVI by TIMMERMAN *et al.* (1994). TIWARI *et al.* (1999) and KATOCH *et al.* (2010) mapped the location of gene *er2* on pea LGIII. Most recent, COBOS *et al.* (2018) mapped the location of *Er3* gene on pea LGIV. COBOS *et al.* (2018) also reported a SSR marker, AD61, which is tightly linked to *Er3* gene. *Er3* is located 0.39 cM downstream of AD61 marker. A number of experiments have been conducted to study the linkage between the *er1* locus and various genetic markers. SARALA (1993) reported the location of *er1* gene on pea LGVI with the use of morphological markers. Marker-assisted breeding is the most appropriate method to transfer *er1* gene based resistance into commercially cultivated varieties. EK *et al.* (2005) developed three SSR markers, PSMPSAD60, PSMPSAA374 and PSMPA5 linked to *er1* gene but these markers are located at a distance of 10.4, 11.6 and 14.9 cM from *er1*, respectively. These distances are too large to be useful for MAS (RIBAUT *et al.*, 2002).

According to WERNER *et al.* (2000) if single marker is located far away from the gene of interest, then two flanking markers can be used in combination. As mentioned earlier, markers identified by EK *et al.* (2005) are located away from the *er1* gene but the gene *er1* is flanked by the markers PSMPSAD60 and PSMPS5 and by using these two markers in combination, effective selection can be made for resistant plants with an error of only 1.6%. An RAPD marker $OPB18_{430,}$ which is linked to *er1* gene at 11.2 cM distance, was identified by NISAR and GHAFOOR (2011).

CONCLUSION

Mendel's discovery of the laws of inheritance was done by using pea as a model organism that makes it the foundation of modern plant genetics. Rust and powdery mildew resistance are the two major components of pea breeding. Phenotypic expression of both the disease resistance is not easily observable because initiation and development of rust and powdery mildew is greatly influenced by the environmental conditions. As a result of this, genetic basis of resistance is still not clear. Also, available information on host-pathogen interaction is very less. That is a major limitation for breeding programs of rust and powdery mildew resistance. Knowledge about the biology of the pathogen and host-pathogen interactions can improve efficiency of resistant varietal development program.

FUTURE CHALLENGES

Use of resistant varieties is the most efficient, economic and also environmental friendly approach to control pea diseases. But most of the powdery mildew resistant commercial varieties carries the gene *er1* only which can lead to development of new races of pathogen by breaking down of the resistance. Therefore, it is necessary to develop varieties which carry resistance from multiple genes. Incomplete polygenic resistance is durable as it cannot be easily broken by a single mutation of the pathogen. More than one gene into the same cultivar would provide durable resistance and it will also increase the level of resistance. It is difficult to exploit minor genes in disease resistance programs because identification of minor genes is difficult.

The other way to provide a durable resistance is by combining different major genes into the same variety. Combined resistances first limit colony establishment and, if this fails, to cause death of established colonies by hypersensitive response would provide a double barrier to disease development and also this will provide durable resistance. By this approach, a complete resistance can be provided. Further the use of molecular markers linked to the resistant genes, can aid to this strategy.

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PRISUSTVO RĐE (*Uromices viciae-fabae*) I BUĐI (*Erisiphe poligoni* DC) KOD GRAŠKA (*Pisum sativum* L.)

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Izvod

Grašak je samooplodna mahunarka sa diploidnim brojem hromozoma 14. Grašak se ekstenzivno gaji i zbog visokog sadržaja proteina je kultura od velikog značaja. Međutim, na uzgoj graška utiču brojni biotički i abiotički stresovi. Gljivične bolesti poput rđe, pepelnice, fuzarioznog uvenuća itd. su najrašireniji biotički stresovi. Rđa i pepelnica nanose veliku štetu usevima I u tropskim I u umerenim krajevima sveta. Upotreba fungicida za suzbijanje biljnih bolesti je dobar pristup, ali prekomerna upotreba fungicida može prouzrokovati zagađenje životne sredine i katastrofe širom sveta, a takođe može izazvati otpornost kod patogena. Stoga, da bi se uklonila ova ograničenja, moraju se koristiti sorte otporne na bolesti. Upotreba otpornih sorti je siguran i efikasan alternativni metod za suzbijanje biljnih bolesti. Globalno je započeto oplemenjivanje na otpornost na rđu i pepelnicu i identifikovan je veliki broj otpornih izvora. Da bi se gen otporanosti uneo u komercijalne sorte graška, potrebno je integrisati molekularne metode sa konvencionalnim tehnikama oplemenjivanja. Do danas je napravljena samo jedna *linkage map* za otpornost na rđu graška; dok su za pepelnicu mapirana tri gena. Molekularni markeri povezani sa ovim genima mogu se koristiti u programima oplemenjivanja otpornih sorti. Da bi se poboljšala efikasnost selekcije za otpornost na rđu i pepelnicu i poboljšalo stvaranje sorti, mora se koristiti integrisani pristup genomskih resursa, efikasni molekularni alati i alati za fenotipizaciju visoke rezolucije. Pregled rđe graška i pepelnice, strukture patogena, gubitaka prinosa i tehnika oplemenjivanja koje se podrazumevaju za suzbijanje ovih bolesti dat je u ovom preglednom radu.

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