Indian Phytopath. 64 (3) : 229-234 (2011)

RESEARCH ARTICLE

Histological studies of powdery mildew *(Erysiphe graminis* f. sp. *tritici*) resistance in wheat (*Triticum aestivum*)

A.K. BASANDRAI[1]*, R. PAUL[2], A. GULATI[3], P. D. TYAGI[2] and DAISY BASANDRAI[1]

[1]*CSK Himachal Pradesh Agricultural University, Hill Agricultural Research and Extension Centre, Dhaulakuan 173 001*
[2]*Department of Plant Pathology, CSK Himachal Pradesh Agricultural University, Palampur 176 062*
[3]*Plant Pathology Section, Institute of Himalayan Bioresource Technology, Palampur 176 061*

ABSRACT: Infection process of *Erysiphe graminis* f. sp. *tritici* pathotype 4 was examined on primary seedling leaves of eight wheat genotypes having specific gene(s) for powdery mildew resistance and two commercially grown cvs., in comparison with universal susceptible cv. Agra Local. In incompatible host parasite interactions, conidial germination and appressoriurn formation were less, particularly in Norka x Cc[8] (*Pm 1*). Germtubes appeared weak, shrivelled and distorted in Sappo (*Pm 2+4b*) and Kronjuwel (*Pm 4b+8*). Haustorial abortion, less secondary haustoriurn production, and restricted growth of mycelial wefts, with no subsequent conidiophore formation were observed in TP 114 (*Pm 2+6*). In Norka x Cc[8], Sappo and Kronjuwel, development of haustoriurn and elongated secondary hypha was completely absent. In compatible host parasite combinations, the sequence of infection events was similar to the susceptible check, differences were observed in components of rate reducing resistance. Incubation period and latent period were longer in Amigo (*Pm 17*), HS 240 and HS 295. In CS/Hope (*pm 5*), Kavkaz (*Pm 8*), Amigo, and HS 295 colony size and number per unit area and sporulation index were reduced, whereas in Transec (*Pm 7*) colony size and in HS 240 sporulation index were at par with the susceptible check. Sporulation capacity was also reduced in all the genotypes.

Key words: *Erysiphe graminis tritici*, histopathology, powdery mildew, resistance mechanism, wheat

In wheat and barley several race specific resistance mechanisms, such as failure of the pathogen to penetrate the host cell wall, abortion or inhibition of haustorial development and degeneration of haustoria in the epidermal cells, and necrosis of the tissues surrounding the infected cells have been reported against infection by *Erysiphe graminis* (Hyde and Colhoun, 1975; Wright and Heale, 1988; Kunoh *et al.,* 1990; Toyoda *et al.,* 1990; Nashaat and Moore, 1991; Wang *et al*., 2003). The rate reducing resistance has also been observed against wheat and barley powdery mildews in compatible interactions (Shaner, 1973; Nass *et al.,* 1981; Sharma *et al.,* 1991). Although, a large number of resistance sources to the pathogen have been identified and characterised in India (Sharma and Singh, 1990; Basandrai and Sharma, 1990; Upadhyay *et al.,* 1972) but no published account is available on powdery mildew resistance mechanisms in wheat.

The present investigation was undertaken to study the chronology of events in the infection process of *E. graminis* f sp. *tritici* in different genotypes of wheat to determine when and how compatible and incompatible interactions are differentiated. A quantitative analysis was also made on histological developments occurring during powdery mildew infection on both resistant and susceptible genotypes.

MATERIALS AND METHODS

Pathogen

Pathotype 4 of *Erysiphe graminis* f. sp. *tritici,* maintained on susceptible wheat cv. Agra Local, was used in the investigation. The pathotype was characterised on avirulence/virulence formula:

Pm 1,2,3a, 3b, 4, 2+6/Pm 3c, 5, 7, 8, Ma.

Host material

Eight wheat genotypes having specific genes for powdery mildew resistance were included in the study *viz*. Norka x Cc[8] (*Pm 1*), CS/Hope (*Pm 5*), TP 114 (*Pm 2+6*), Kavkaz (*Pm 8*), Amigo (*Pm 17*), Sappo (*Pm 2+4b*) and Kronjuwel (*Pm 4b+8*), two commercially grown cvs. HS 240 and HS 295, and universal susceptible cv. Agra local.

Inoculation of seedlings

Eight days old potted seedlings were spread horizontally on a glass sheet. The abaxial surface of leaves were kept in upwards position and inoculated by shaking over them heavily mildewed seedlings of Agra Local from 30 cm height. Uniformity in inoculum distribution on leaves was ensured by microscopic examination of conidia trapped on vaseline coated glass slides placed along with the leaves. The inoculated seedlings were incubated in a muslin cloth covered wooden chambers (temperature: maximum 20±3 °C, minimum 12±3 °C) under natural light.

Infection process and components of rate reducing resistance

The time course of the infection process was followed histologically using a Nikon Biophot microscope.The central

*Corresponding author: ashwanispp@gmail.com

portion of the inoculated leaves was cut into 0.5 and 1.0 cm segments after 12, 24, 48, 72, 96 and 144h of inoculation. The leaf segments were decolourised in Carnoy's solution (Haywood and Ellingboe, 1979), stained in lactophenol trypan blue solution (0.02%), and examined microscopically.

For quantitative determination of the pathogen development on both resistant and susceptible genotypes, observations were recorded on conidial germination, appressorium formation, elongated secondary hypha (ESH) production, haustorium production and their size, and rate reducing components including incubation and latent periods, colony number and size, sporulation index, and sporulation capacity (Shaner, 1973; Hyde and Colhoun, 1975; Nashaat and Moore, 1991).

RESULTS AND DISCUSSION

Sequence of events in the infection process

Compatible host pathogen interactions: On cv. Agra Local, the germinated conidia produced appressoria terminally on germtubes within 12h of inoculation. A mature appressorium was subtended by the septum formed across the germ tube. Appressoria were more or less foot shaped, closely appressed to the host surface, and produced primarily on periclinal host cell wall (Fig. 1a). Primary penetration of the host surface was direct and the infection peg formed at the undersurface of the appressorium penetrated through host cuticle and outer epidermal cell wall. Mostly, penetration was effected within 24h of inoculation through the penetration holes having smooth margins (Fig. 1b). Occasionally epidermal cell wall thickened and formed a collar like structure (papilla) around the infection peg (Fig. 1b). An oval shaped primary haustorial body developed within the epidermal cell at the tip of penetration hypha (Fig. 1b). Between 24 and 72h of inoculation, the haustorium increased in size and digitate, elongated finger like appendages developed from both ends of the haustorium. Concomitant with haustorial development, formation of elongated secondary hyphae from appressoria on the host surface was also observed after 48h of inoculation (Fig. 1c). A mycelial weft developed on the leaf surface with continued growth and branching of elongated secondary hyphae. Growth of mycelial weft was accompained by secondary penetration of the leaf surface and subsequent production of secondary haustoria between 48 to 72h of inoculation (Fig. 1d). In stained material, blue "halos" were often observed 6 days after inoculation along the sites of subsequent penetrations. Secondary penetrations were effected through the penetration holes produced within "halos" (Fig. 1e, 1f). Conidiophore development sites were deeply stained and terminally swollen appendages were formed on the mycelial weft after 48 to 72h of inoculation (Fig, 1g). These had swollen bases and produced conidial chains with mature conidia in acropetal succession after about 6 days of inoculation.

A similar sequence of events was observed during infection in compatible host pathogen interactions, involving pathotype 4 of *E. graminis f. sp. tritici* of other susceptible

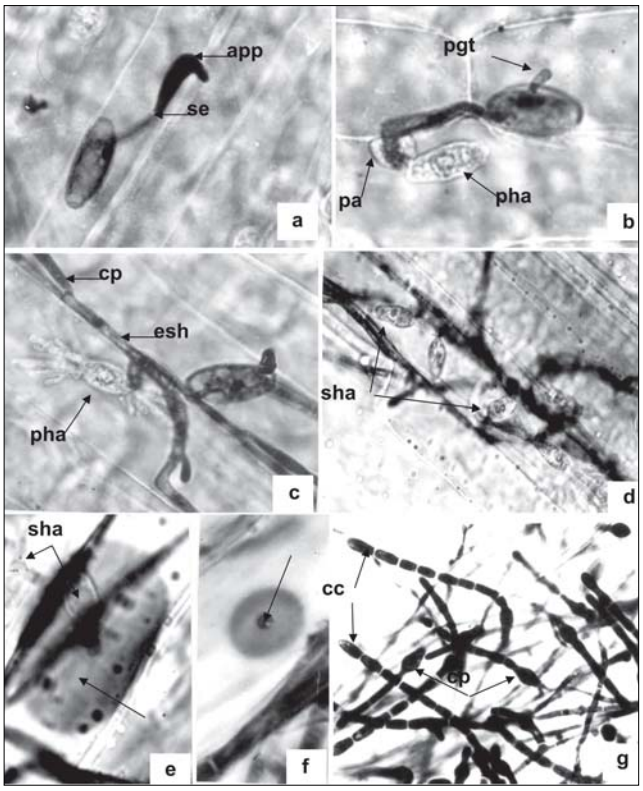

Fig. 1. Infection process of *Erysiphe graminis* f. sp. *tritici* 'pathotype 4' on *Triticum aestiuum* cv. Agra Local: (a) Germinated conidium showing appressorium (app) with subtending septum (se) (12h after inoculation), x 1000; (b) Primary penetration of the host-surface by a conidium, showing primary germtube (pgt), papilla (pa) and primary haustorium (pha) (24h after inoculation), x 2000; (c) Conidium with elongated secondary hypha (esh) and condiophore (cp) on host-surface and primary haustorium with digitate processes (pha) within the epidermal cell (48h after inculcation) x 1600; (d) Secondary penetration of host-surface showing secondary haustoria (sha) (72h after inoculation) x 1600; (e) Secondary penetration showing blue "halos" around the penetration site (arrow) and secondary haustoria (sha) within epidermal cells (6 days after inoculation), x 2000; (f) smooth penetration hole in the "halo", indicated by an arrow (6 day after inoculation), x 2800; (g) Conidiophores (cp) with conidial chains (cc) (6 days after inoculation), x 800.

host genotypes, viz. CS/Hope (*Pm 5*), Transec (*Pm 7),* Kavkaz (*Pm 8*), Amigo (*Pm 17*), HS 240, and HS 295. However, development of sporulating colonies in Amigo (*Pm 17*), HS 240 and HS 295 were observed after seven days of inoculations instead of six days in CS/Hope (*Pm 5*), Transec (*Pm 7*) and Kavkaz (*Pm 8*). Although histological studies have been carried out to understand mechanisms of resistance associated with some known powdery mildew resistance genes (*Pm* genes) in wheat (Slesinski and Ellingboe, 1969; Hyde and Colhoun, 1975; Haywood and Ellingboe, 1979), such investigations involving susceptible genes *Pm 7*, *Pm 17* and resistant gene combinations *Pm 2+4b*, and *Pm 4b+8* are being reported here for the first time.

Incompatible host pathogen interactions: Various levels of resistance mechanisms were observed in resistant genotypes Norka x Cc^8 (*Pm 1*), TP 114 (*Pm 2+6*), Sappo

(*Pm 2+4b*) and Kronjuwel (*Pm 4b+8*). Conidial germination was less on resistant genotypes, particularly on Norka x Cc[8] and Sappo, as compared to susceptible ones. Germtube growth was normal on all the resistant genotypes except Sappo on which growth was inhibited (Fig. 2a). On Norka x Cc[8] and Sappo mature appressoria formed after 24h of inoculation (Fig. 2b), while on Kronjuwel and TP 114 mature appressoria developed after 12h (Fig. 2d, 3a). On Sappo (Fig. 2b, 2c) and Kronjuwel (Fig. 2e, 2f) the germtubes appeared weak, shriveled and distorted when examined after 24 to 72h of inoculation. Appressoria appeared reduced in size, flattened and deformed. Growth of the pathogen stopped after papilla formation on Kronjuwel (Fig. 2e). Further development of pathogenic units was inhibited after appressoria formation in Sappo. In genotypes Norka x Cc[8], Sappo and Kronjuwel mesophyll cells underlying the infection court assumed loose disorganised appearnce within 48h of inoculation (Fig. 2g). This indicated that resistance mechanisms probably became operative at the cuticular level. However, in other investigations cuticle provided no serious barrier to penetration (Jarosz *et al*., 1982; Nashaat and Moore, 1991). Svec and Mikulova (2009)

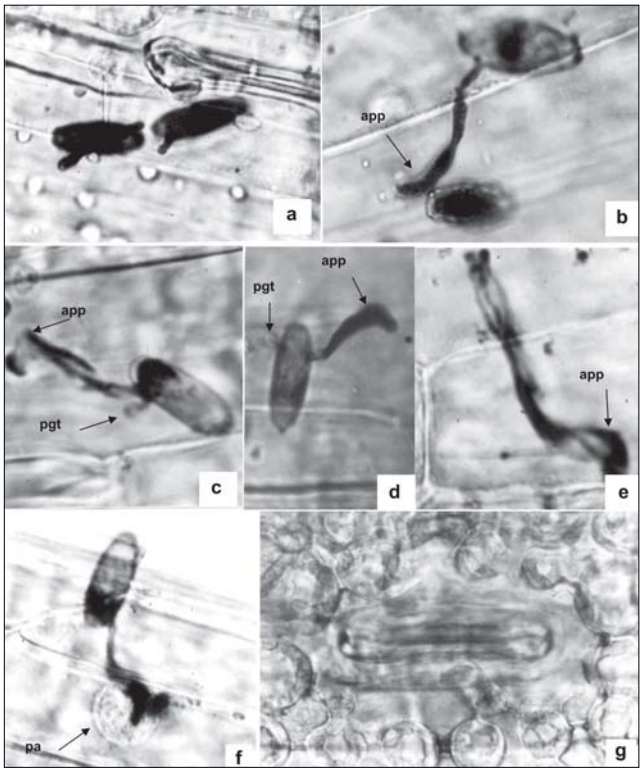

Fig. 2. Infection process of *Erysiphe graminis* f. sp. *tritici* 'pathotype 4' on *Triticum aestivum* genotypes Sappo (a-c) and Kronjuwel (d-g): (a) Germinated conidium with inhibited germtube growth on Sappo (12h after inoculation), x 1600; (b) An ungerminated conidium and a germinated conidium with mature appressorium on Sappo (24h after inoculation), x 1200; (c) Shrivelled and distorted appressorium and germtube on Sappo (48h after inoculation), x 1200; (d) Conidium with primary germtube (pgt) and mature appressorium (app) on Kronjuwel (12 h after inoculation), x 1800; (e) Papilla (pa) formation around the penetration site in Kronjuwel (24h after inoculation) x 1200; (f) Shrivelled and distorted growth of the pathogen on Kronjuwel (48 h after inoculation), x 2400; (g) Disorganization of mesophyll cells in Kronjuwel (48 h after inoculation) x 1600

compared the values of pre-haustorial resistance in the supersensitive hexaploid control cultivar Ai-bian 1 and in dicoccum wheat genotypes CGN 11486 and TRI 6158. It was reported that in the *dicoccum* samples less than 80% of pathogen's units were arrested in appressorial stage and more than 20% infection units could penetrate the host's cell and developed into secondary germ tube stage by 48 hours after inoculation. Lupton (1956) reported that *E. graminis* germinated and produced appressoria on the leaves on *Triticum carthlicum*, *T. dicoccum* var. *farrum* and *T. dicoccum* f sp. *georgicum*, *T. timopheevi and Aegilops caudata* but it was unable to penetrate the epidermal wall. In another investigation, almost complete inhibition of ESH formation was observed in near isogenic line CI 14123 (*Pm 4*) and cv. Khapli (*Pm 4* donor) inoculated with either isolate W 9/30 or W 71/178 of *E. graminis f. sp. tritici* (Hyde and Colhoun, 1975). The reaction was a marked epidermal hypersensitivity which resulted in the rapid abortion of developing haustoria after penetration.

Formation of papillae was observed in Kronjuwel (*Pm 4b+8*). Some workers have found association of papillae with race specific incompatibility (Stolzenburg *et al*., 1984; Nashaat and Moore, 1991) whereas, others did not find evidence of their involvement in resistance (Hyde and Colhoun, 1975; Wright and Heale, 1988; Serezhkina *et al*., 1996; Hu and Kang, 1998). In the present investigations papillae were also observed in the universal susceptible cv. Agra local also. This showed that papillae probably have no role in resistance.

As stated above, Sappo and Kronjuwel containing genes *Pm 2+4b* and *Pm 4b+8*, respectively, showed resistance to pathotype 4, however, Kavkaz containing gene *Pm* 8 was susceptible. It has been shown by other workers that the *P2*/*Pm2* gene combination did not appear incompatible until approximately 3 days after inoculation, when mesophyll cells underlying the infection court turned necrotic (Slesinski and Ellingboe, 1969; Hyde and Colhoun, 1975). However, in the present investigation a genotype containing gene *Pm* 2 could not be tested. It may be concluded -from the available evidence in the present studies that gene *Pm 4b* in combination with the genes *Pm 2* and *Pm 8* in genotypes Sappo and Kronjuwel, respectively resulted in incompatibility with pathotype 4.

On TP 114 penetration of host cell walls, primary haustorial development, elongation of secondary hyphae, secondary penetrations, lesser development of secondary haustoria, and restricted growth of mycelial weft were observed (Fig. 3b, 3c, 3d). However, further production of primary and secondary haustoria stopped after 96h of inoculation (Fig. 3d). Although conidiophore initials were formed, no conidiophore or conidia developed. As has been observed in the present studies, Wu *et al*. (1985) and Wang *et al*. (2003) reported resistance to *E. graminis* f. sp. *tritici* in some cvs of wheat was conferred by reduction in number and final size of haustoria. A reduction in the size of haustoria in incompatible interactions were also found by Haywood and Ellingboe (1979). It is thus clear that the resistance mechanisms in TP 114 (*Pm2+*6) are different from those in Norka X Cc[8] (*Pm1*), Sappo (*Pm 2+4b*) and Kronjuwel (*Pm 4b+8*).

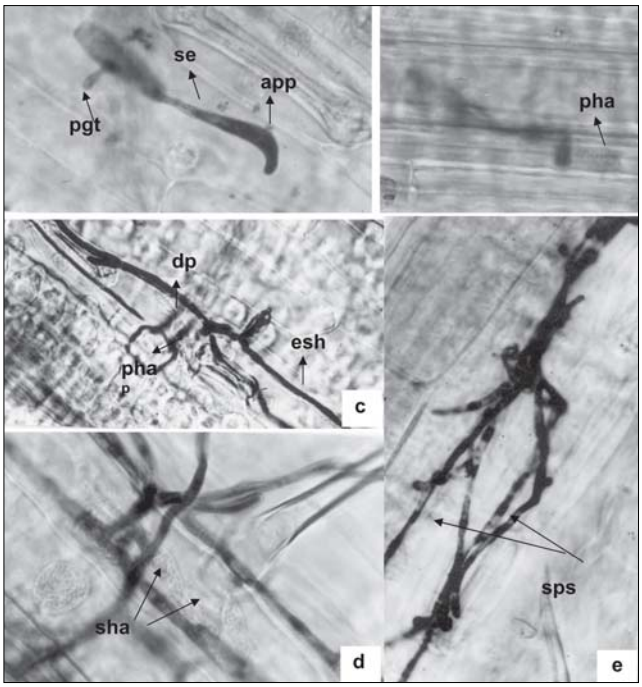

Fig. 3. Infection process of *Erysiphe graminis* f. sp. *tritici* pathotype 4 on *Triticum aestiuum* genotype TP 114: (a) Germinated conidium showing primary germtube (pgt) and appressorium (app) with subtending septum (se) (after 12h after inoculation), x 1200; (b) Primary penetration by a conidium showing primary haustorium (pha) (24 h after inoculation), x 1800; (c) Primary penetration showing haustorium (pha) with digitate process (dp) and elongated secondary hyphae (esh) (48h after inoculation), x 800; (d) secondary haustoria (sha) developing within epidermal cells and conidiophore intials (ci) developing on mycelial weft (72h after inoculation), x 1600; (e) Restricted growth of mycelial weft with secondary penetration sites (sps) (96h after inoculation), x 1600.

Quantitative analysis of histological development during infection: Data was recorded on conidial germination, appressorial formation, elongated secondary hyphae and haustorial production for quantitative determination of powdery mildew development on different genotypes (Table 1). Conidial germination was at par on all the susceptible cultivars. However, it was significantly less on resistant genotypes Norka x Cc[8], TP 114, Sappo and Kronjuwel as compared to susceptible ones. Appressorial formation was significantly less on all resistant genotypes as compared to Agra local and other susceptible genotypes. Appressorial formation was least on genotype Norka x Cc[8], followed by Sappo and Kronjuwel. On susceptible genotypes the mature appressoria produced ESH. However, inhibition in ESH production occurred on resistant genotype TP 114, while on others such as Norka x Cc[8], Sappo and Kronjuwel no elonagated secondary hyphe (ESH) were produced.

In susceptible genotypes 80-90% parasitic units produced primary haustoria after 48 h of inoculations against 30 % in the resistant genotype TP 114. Primary haustorial production was not observed in Norka x Cc[8], Sappo and Kronjuwel. The size of primary haustoria was the largest in genotypes Transec and HS 240, and the smallest in TP 114.

In susceptible genotypes 5.1-6.1 secondary haustoria per parasitic unit were produced as against 0.6 haustoria per parasitic unit in the resistant genotype TP 114. No secondary haustorial production was observed in Norka x Cc[8], Sappo and Kronjuwel.

Components of rate reducing resistance

Observations on components of rate reducing resistance in compatible combinations were recorded on incubation and

Table 1. Conidial germination, appressorial formation, elongated secondary hyphae (ESH) and haustorial production by *Erysiphe graminis* f. sp. *tritici* pathotype 4 on different wheat genotypes

Genotype	Powdery mildew resistance gene(s)	Reaction to pathotype 4	Conidial germination (%) (12h*)	Appressorial formation (%) (24h*)	ESH/Appr. (%) (48h*)	Primary haustorial production (%)		Mean primary haustorial size (mm) (72h*)	Mean number of secondary haustoria/ parasitic unit(72h*)
						(24h*)	(48h*)		
Agra local	None	S	86 (68.0)	85 (67.2)	100	80	85	107.3 x13.5	5.1
Norkax Cc[8]	*Pm 1*	R	13 (21.1)	13 (21.1)	—	—	—	—	—
CS/Hope	*Pm 5*	S	93 (74.7)	90 (71.6)	100	70	85	107.3x13.5	5.4
TP 114	*pm 2+6*	R	62 (51.9)	60 (50.8)	80	25	30	68.8x13.5	0.6
Transec	*Pm 7*	S	92 (73.6)	90 (71.6)	100	75	82	118.4x13.5	5.9
Kavkaz	*Pm 8*	S	96 (78.5)	96 (78.5)	100	78	80	99.9x13.5	5.8
Amigo	*Pm 17*	S	92 (73.6)	90 (71.6)	100	85	90	108.0x13.5	5.8
Sappo	*Pm 2+4b*	R	50 (45.0)	34 (35.7)	—	—	—	—	—
Kronjuwel	*Pm 4b+8*	R	48 (43.9)	48 (43.9)	—	—	—	—	—
HS 240	?	S	89 (70.6)	88 (69.3)	100	80	80	118.4x13.5	6.0
HS 295	?	S	91 (72.5)	90 (71.5)	100	75	85	107.3x13.5	6.1
S.E.			±2.3	±1.3					
CD (5%)			4.8	2.7					

*hours after inoculation; ?=not known; R=resistant; S=susceptible
Data was taken on 90 units from three replicates and analysed following completely randomised design (CRD)

Table 2. Components of rate reducing resistance in compatible wheat-*Erysiphae graminis* f. sp. *tritici* 'pathotype 4' combinations

Genotype	Incubation period (days)	Latent period (days)	Colonies* per cm^2	Colony size (mm^2)	Sporulation* index	Sporulation* capacity (%)
Agra local	4	6	32.4	0.8	87.6	100.0
CS/Hope	4	6	11.9	0.4	24.9	10.4
Transec	4	6	16.6	0.7	58.0	33.9
Kavkaz	4	6	22.1	0.3	33.6	26.2
Amigo	5	7	20.3	0.4	26.4	18.9
HS 240	5	7	18.9	0.6	73.3	48.8
HS 295	5	7	20.2	0.5	44.4	31.6
S.E.	—	—	±3.1	±0.1	±7.8	—
CD (5%)	—	—	6.6	0.1	6.7	—

*8 days after inoculation

Data on colonies per cm^2 was taken on 30 leaf bits, and on colony size, sporulation index and sporulation capacity on 30 colonies from each of the three replicates and analysed following completely randomised design (CRD)

latent periods, colony number and size, sporulation index, and sporulation capacity (Table 2).

In Agra Local, CS/Hope, Transec and Kavkaz development of macroscopically visible colonies and spore production were recorded after 4 and 6 days. Genotypes Arnigo, HS 240, and HS 295 exhibited incubation and latent periods of 5 and 7 days, respectively. Similarly, longer incubation and latent periods vis a vis Agra Local as reported earlier in Chul x Cc^8 having gene *Pm 3b* (Sharma et al., 1991) and HS 240 (Kanwar, 1993). A significant reduction in colony number/unit leaf area was recorded in all the susceptible genotypes as compared to Agra Local. In Transec, colony size was at par with Agra local, while in other susceptible genotypes/cvs. colony size was significantly smaller. Sporulation index in HS 240 was statistically at par with susceptible cv Agra Local, while in other compatible genotypes sporulation index was signicantly less. Sporulation capacity was markedly reduced in all compatible combinations, particularly CS/Hope and Amigo, over Agra Local.

All genotypes and cvs. developed significantly less colonies per unit area as compared to Agra Local. These genotypes/cvs., except Transec and HS 240, also exhibited smaller colony size and low sporulation index, respectively, in comparison to Agra local. Aggarwal and Bahadur (1995) studied the development of *E. graminis tritici* on susceptible and resistant wheat genotypes through scanning electron microscopy. Scanty growth and few conidial chains were observed on resistant genotypes, HD 29 and HD 30 and heavy growth with 24-30 conidial chains per microscopic field were observed in susceptible Cu, WL 711. Similarly, Ryabchenko *et al*. (2003) reported that development of *E. graminis* f. sp. *tritici* on wheat- *Aegilops* derived line 135/99 was characterized by longer incubation period and sparse colonies from mycelial hyphae with multiple hyphal lobes with often disturbed adhesion to the surface of epidermal cells. Low sporulation has also been reported in near - isogenic lines having genes *Pm 3c*, *Pm 4* and *Pm (Ma) vis a vis* susceptible cv. Agra Local (Nass *et al*., 1981). Likewise, smaller colonies and low spore production was observed in lines with genes *Pm 3a*, *Pm 3b*, *Pm 3c*, and *Pm* 8 compared to cv Agra local (Sharma *et al*., 1991). The rate reducing resistance in CS/Hope, Transec, Kavkaz, and Arnigo appears related to genes *pm 5*, *Pm 7*, *Pm 8*, and *Pm17*, respectively. In commercially cultivated line HS 240, the rate reducing resistance is possibly related to gene *Pm 8*, incorporated by genotype Veery in the pedigree. The results also clearly indicated existence of gene(s) for residual resistance in HS 295.

The lack of development of visible colonies in Norka x Cc^8 (*Pm 1*), Sappo (*Pm 2+4b*), Kronjuwel (*Pm 4b+8*) and TP 114 (*Pm 2+6*) implied that resistance mechanisms operating in these genotypes were capable of inactivating build up of the initial inoculum. Race specific vertical resistance genes retard epidemic development by reducing the effective initial inoculum (Vanderplank, 1963, 1968). Hence, genotypes Norka x Cc^8, TP 114, Sappo, and Kronjuwel showed race specific vertical resistance. As is evident from the longer incubation and latent periods, less number of colonies per unit area, smaller colony size and reduced sporulation in CS/Hope, Transec, Kavkaz, Amigo, HS 240, and HS 295 on inoculation with Egt pathotype 4 with matching virulence, race-specific resistance also reduced the apparent infection rate. Thus genotypes possessing major genes for resistance also showed rate reducing/residual resistance upon confronting the matching virulence. Reduction in the apparent infection rate is a trait generally attributed to gene(s) for horizontal resistance (Vanderplank, 1963, 1968). Race specific major and non race specific minor genes have also been described as expression of different actions of the same gene(s) in different genetic backgrounds of the pathogen (Nelson, 1975, 1978). Furthermore, the residual or ghost effect of polygenes is reflected in components of rate reducing resistance (Clifford, 1975). This residual resistance can be effectively employed in the development of multiline varieties with buffering effect to disease development.

ACKNOWLEDGEMENTS

Authors are thankful to the Director, Institute of Himalayan Bioresource Technology, Palampur, Head, Plant Pathology Department, Himachal Pradesh Agricultural University,

Palampur and Associate Director (R&E) HAREC, Dhaulakuan Palampur for providing necessary facilities.

REFERENCES

Aggarwal, R. and **Bahadur, P.** (1995). Host pathogen interaction in powdery mildew of wheat through SEM. *Cereal Rusts Powd. Mild. Bull.* **23**: 1-6.

Basandrai, A.K. and **Sharma, S.C.** (1990). Sources of powdery mildew resistance in wheat and triticale and inheritance of resistance in selected *Triticum aestivium* L. and *T durum* Desf genotypes. *J. Genet. Plant Breed.* **44**: 253-257.

Clifford, B.C. (1975). Stable resistance to cereal diseases: Problems and progress. In: *Report Welsh Plant Breeding Station.* 1974, 107-13.

Haywood, M.J. and **Ellingboe, A.H.** (1979). Genetic control of primary haustorial development of *Erysiphe graminis* on wheat. *Phytopathology* **69**: 48-53.

Hu, D., Li, Z. and **Kang, Z.** (1998). Ultrastructure and cytochemistry of papilla response in wheat against attack by *Blumeria graminis* f sp. *tritici. J. Zhenjiang Agric. Univ.* **24**: 502-508.

Hyde, H. and **Colhoun, J.** (1975). Mechanism of resistance of wheat to *Erysiphe graminis* f sp. *tritici. Phylopath. Zeitscrift* **82:** 185 206

Jarosz, A.M., Sheets, M. and **Levy, M,** (1982). Cuticle thickness in phlox and resistance to powdery mildew: an unreliable line of defence. *American J. Bot.* **69**: 824-828.

Kanwar, N. (1993). Investigations on powdery mildew *(Erysiphe graminis* f. sp. *trifici)* resistance and intensity loss relationship in wheat.*:* Ph.D. Thesis, Himachal Pradesh Agricultural University Palampur, India pp. 118.

Kunoh, H., Nicholson, R.L., Yosioka, H., Yamaoka, N. and **Kobayshi, I.** (1990). Penetration of infection court by *Erysiphe graminis.* Degradation of the host cuticle. *Physiol. Mol. Plant Path.* **36:** 397 407.

Lupton, F.G.H. (1956). Resistance mechanisms of species of *Triticum* and *Aegilops* and of amphidiploids between them to *Erysiphe graminis* DC. *Trans. British Mycol. Soc.* **39**: 51-9.

Nashaat, N.I. and **Moore, K.** (1991). The expression of components of seedling resistance to *Erysiphe graminis* f. sp*. trifici* in *Triticum timopheevii* and a hexaploid derivative. *Plant Path.* **40**: 495-502.

Nass, H.A., Peterson, W.L., Mackenzie, D.R. and **Nelson, R.R.** (1981). The residual effect of some "defeated' powdery mildew resistance genes in isolines of Chancellor winter wheat. *Phytopathology* **71**: 1315-1318.

Nelson, R.R. (1975). Horizontal resistance in plants. Concepts, controversies and application. In: Galvez GE, eds. *Proceedings of the Seminar on Horizontal Resistance to Blast Disease of rice. CIAT* publication, series CE 9 Cali, Columbia, 1 20.

Nelson R.R. (1978). Genetics of horizontal resistance to plant disease. *Annu. Rev. Phytopath.* **16**: 359-378.

Ryabchencko, A.S., Serezhkina, G.V., Mishina, G.N. and **Andreev, L.N.** (2003). Morphological variability of wheat – powdery mildew in context to wheat- *Aegilops* lines with different resistance. *Biol. Bull. Rus. Acad. Sci.* **30**: 255-261.

Serezhkina, G.V., Andreev, L.N., Avetisyan, T.V., Batova. S.N. and **Poleva, L.V.** (1996). Role of primary reactions in interactions between the parasite and host plant with special reference to resistance of the wheat-wheatgrass hybrids to *Erysiphe graminis tritici* at the stage of penetration. *Biol. Bull. Rus. Acad. Sci.* **23**: 346-352.

Shaner, G. (1973). Reduced infectability and inoculum production as factors of slow mildewing in knox wheat. *Phytopathology* **63**: 1307-1311.

Sharma, S.C., Prakash Vidya, Basandrai, A.K. and **Aulakh, K.S.** (1991). Residual resistance in wheat to *Erysiphe graminis* f sp. *tritici. Plant Dis. Res.* **6**: 70-74.

Sharma, T.R. and **Singh, B.M.** (1990). Development of powdery mildew on different wheat cultivars. *Indian Phytopath.* **43**: 170-74.

Slesinski, R.S. and **Ellingboe, A.H.** (1969). The genetic control of primary infection of wheat by *Erysiphe graminis* f. sp. *tritici. Phytopathology* **59**: 1833-1837.

Stolzenurg, M.C., Aist, M. and **Israel, H.W.** (1984). The role of papillae in resistance to powdery mildew conditioned by *Mla* gene in barley. II. Experimental evidence. *Physiol. Plant Path.* **25**: 347-364.

Švec, Miroslav and **Mikulová, Katarína** (2009). Laboratory method for identification of resistance genetic sources to powdery mildew on wheat. In: *Determination and Evaluation of Cereal Diseases*. Presented in 7th specialist Seminar on Influence of biotic and abiotic stresses on disease occurrence and development held at Crop Research Institute, Division of Plant Genetics, Breeding and Product Quality, Prague – Ruzyne.

Toyoda, H., Mayama, S. and **Shishiyama, J.** (1978). Fluorescent microscopical studies on the hypersensitive necrosis in powdery mildewed barley leaves. *Phytopath. Zeitscrift* **68**: 181-187.

Upadhyay, M.K., Kumar, R. and **Singhal, N.C.** (1972).Sources of resistance to powdery mildew of wheat. *Indian J. Genet. Plant Breed.* **32**: 242-246.

Vanderplank, J.E. (1963). *Plant Disease Epidemic and Control.* New York, USA: Academic Press.

Vanderplank, J.E. (1968). *Diseave Resistance in Plants,* New York, USA: Academic Press.

Wang, ZhangMing., Chen, HouDe., Bi, HuaSong., Fang Zheng., Wang, Z.M., Chen, H.D., Bi H.S. and **Fang, Z.** (2003). Studies on haustorium' s development of *Blumeria graminis* f sp *tritici* and papillae formation of host cell. *J. Yangzhou Univ. Agri Life Sci.* **24**: 64-67.

Wright, A.J. and **Heale. J.B.** (1988). Host response to fungal penetration in *Erysiphe graminis* f sp*. hordei* infection in barley. *Plant Path.* **37**: 131-40.

Wu, W., Yang, J.S. and **Wu, Y.S.** (1985). Relation of hausroria to wheat powdery mildew resistance. *Acta Phytopath. Sinica* **15**: 31-5.

Received for publication: March 15, 2011
Accepted for publication: July 29, 2011