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REGULAR ARTICLE

Role of phenolic compounds in resistance to chilli wilt

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ABSTRACT

Fusarium wilt is a principle disease of chilli crop in Kashmir and has assumed a serious proportion. The varieties identified as resistant to a particular pathogen may not have desirable traits, however, can be used as donors. Two resistant and 6 susceptible chilli (*Capsicum annum* L.) genotypes and their twelve F_1 hybrids showing variable degree of resistance to *Fusarium* wilt were analyzed for phenols and phenolic enzymes, under both uninoculated and inoculated conditions at different growth stages. Generally total phenols ortho-dihydroxy phenols and the enzyme activity were invariably high in resistant parents and hybrids irrespective of growth stages, while, in case of susceptible parents the phenols content and enzyme activities were comparatively less. There existed a positive correlation between the host resistance and the amount of phenols and increased enzyme activities while it was almost the opposite in susceptible lines. The positive association of higher phenols and enzymes with resistance could be of immense value for early and quick identification of resistant genotypes during screening of large populations.

Key Words: *chilli wilt; Fusarium pallidoroseum; phenolic compounds; resistance.*

INTRODUCTION

Chilli (*Capsicum annuum* L.) is one of the most important vegetable and spice crop in India. It is commercially important because of its pungency and colour. Plants being sessile organism are exploited as a source of food and shelter by wide range of parasites including bacteria, fungi and viruses (Gachomo et al., 2003). Chilli is no exception and a fungal pathogen that invades chilli is *Fusarium spp.* and causes *Fusarium* wilt. The disease is known to be caused by *Fusarium pallidoroseum* (Cooke) Sacc. Recently it has become a serious problem in Kashmir (India) and presents a formidable challenge to chilli producers. The

ability of a plant to ward off a pathogenic attack depends upon the coordination of different defense strategies. Phenolic compounds have long been correlated with the resistance of plants to infective agents (Link et al., 1929; Link and Walker, 1933). There has been little work in India on *Fusarium* wilt and its inheritance pattern. We screened various sources for resistance between 1994 and 2002 and also carried out the inheritance studies revealing that resistance was monogenically inherited. This study was undertaken to study the status and nature of changes in various biochemical factors such as phenols, orthodihydroxy phenols, polyphenol oxidase and peroxidase with the objective of helping in the early screening and selection of desirable genotypes in wilt resistance breeding programmes.

MATERIALS AND METHODS

Studies on biochemical factors were conducted on uninoculated and inoculated resistant and susceptible cultivars and their crosses. The parents and crosses studied are:
Resistant parents (Arka Lohit , SH-C-1154); Susceptible parents (Kashmir Long-I, SH-C-101, SH-PC-1, SH-C-101, SH-PC-1 SH-C-405, Pampori and their twelve hybrids.

Resistant and susceptible parents were grown in the experimental area 2004 rainy season and crosses were made to develop F_1's. Seed of parents and F_1's was sown in the nursery in the 2005 rainy season for evaluation in pots to assess biochemical traits. Pots were 22.5 cm diameter and filled with sterilized autoclaved soil.

POT EVALUATION

Two sets of pots were used. Each set consisting of resistant and susceptible parents and their crosses. One set was kept as uninoculated control, where no fungal inoculum was added to the sterilized soil. The other set received fungal inoculum that was thoroughly mixed with the sterilized soil within the top layer at the rate of one inoculum flask pot^{-1} and harboured *F. pallidoroseum* (38.92 x 103 cfu g^{-1}). Inoculated pots were placed under controlled conditions for 7 days. Nursery raised seedlings were then transplanted into pots after thoroughly washing their roots. There were 6 seedlings pot^{-1} and the design was a randomized complete block design with three replicates. Six seedlings of each genotype were used to evaluate both enzyme and phenol studies in stage 1, i.e. stage just before inoculation.

SAMPLING TECHNIQUES

Samples were collected at four stages i.e. stages after transplanting (S1, S2, S3 and S4) for enzyme activity and at three different stages (S_1, S_2 and S_3) for phenols, from both the sets separately. Where S_1: was just before transplanting, S_2: was five days after transplanting, S_3: was 10 days after transplanting and S_4: was 30 days after transplanting. For enzyme activity (peroxidase and polyphenol oxidase), fresh samples were used that were collected between 9 am. and 10 am. At all the stages 4^{th}, 5^{th} and 6^{th} leaves from top of each plant were picked from both inoculated and uninoculated plants of parents and hybrids. The collected leaves were washed and bulked and 2 g of the fresh bulked sample was used for enzyme analysis. The remaining bulked leaves were dried, powdered and used for phenol estimation. Peroxidase and Polyphenol oxidase enzymes were estimated by the method of Mahadevan and Sridhar (1986). Total phenols were determined by the method of Bray and Thorpe (1954).

Statistical analysis was carried out in R (R Development Core Team, 2009) for analysis of variance which is implementation of Box et al. (1978) design.

RESULTS AND DISCUSSION

Initially the total phenols ranged from 3.40 mg/g in (SH-C-405) to 6.50 mg/g (Kashmir Long-1) in the susceptible genotypes (Table 1). It ranged from 6.90 mg/g (SH-C-1154) to 7.60 mg/g (Arka Lohit) in the resistant parents. In the hybrids it ranged from 7.20 mg/g in Arka Lohit x SH-C-405 to 9.80 mg/g in Arka Lohit × Kashmir Long-1).

By the 2nd stage the total phenolic levels fell with inoculation in susceptible and resistant parents and the hybrids compared with uninoculated healthy, susceptible and resistant parents and hybrids.

At the 3rd phenological stage i.e. 30 days after inoculation there was a significant increase in the total phenols in uninoculated susceptible and resistant parents and hybrids. Without inoculation, total phenolic content decreased both in the resistant and susceptible parents and the hybrids. Variation in the total phenol content increased with the plant age irrespective of resistance level and inoculation treatment. In parental lines, the phenol content increased significantly irrespective of genotypes but the resistant parent and hybrids generally had a higher phenol content than susceptible lines. In the hybrids after initial increase in S_2, it decreased significantly from then on in uninoculated plants. However, with inoculation there was a significant decrease in total phenol content of the genotypes but it was significantly lower in susceptible parents than in resistant parents. In the hybrids total phenol content decreased significantly with inoculation as well as with the advanced growth stage. The significant decrease in the resistant types may be attributed to an increase in polyphenol oxidase and peroxidases. Similar observations were also made in three chilli varieties against fruit rot disease (Borua and Das, 2000) and in seedlings of pigeon pea resistant to wilt particularly during the early stages of growth (Bray and Thorpe, 1954).

Initial ortho-dihydroxy phenolic levels ranged from 0.17 to 0.19 mg g^{-1} in resistant parents while in susceptible parents, the ortho-dihydroxy phenolic levels was minimum (0.02 mg g^{-1}) than the resistant parents (Table 2). In hybrids a minimum value of 0.21 mg g^{-1} was recorded in SH-C-1154 × Kashmir Long-1 and maximum of 0.55 mg g^{-1} in Arka Lohit × SH-C-405. In the 1st phonological stage, the mean value of ortho-dihydroxy phenols was 0.18 mg/g in resistant parents, 0.03 mg g^{-1} in susceptible parents and 0.32 mg g^{-1} in hybrids. There was a higher level of ortho-dihydroxy phenols in the hybrids followed by resistant parents and susceptible parents. In phenological stage 2, the ortho-dihydroxy phenols significantly increased both under uninoculated and inoculated conditions as compared to S_1 and S_3. 30 days after inoculation. With advancing age and growth of plants, all the resistant parents and the hybrids had significantly increased ortho-dihydroxy phenols both under uninoculated and inoculated conditions,. However, the level of ortho-dihydroxy phenol was much higher in resistant parents and hybrids than susceptible genotypes. Generally, in total phenols, the level of ortho-dihydroxy phenols was high in resistant parents and hybrids in all the growth stages. They maintained their high level even after inoculation compared with susceptible genotypes whose ortho-dihydroxy phenols fell drastically after inoculation. It seems, that in resistant genotypes, the ortho-dihydroxy. phenols are continuously produced and maintain their level to provide protection from invading and infected wilt pathogen. There was a significant increase of ortho-dihydroxy phenols with the increased leaf age of groundnut affected by tikka leaf spot disease caused by *Cercospora arachidicola* (Sindhan and Jaglan, 1987) and in cowpea cultivar Co-A resistant to *Xanthomonas vigincola*, Burk. compared to a susceptible cultivar C-M-11 (Mohan et al., 1978). These findings conform to the findings of this study. The higher levels of ortho-dihydroxy phenols in resistant cultivars/hybrids after inoculation was expected because orthodihydroxy phenol compounds are possibly released and oxidized by polyphenol oxidase and peroxidase to the corresponding quinones before they become effective in combating the pathogen.

Polyphenol oxidase (PPO) values ranged from 0.12 to 0.16 units $minute^{-1}$ in the resistant parent, 0.02 to 0.08 units $minute^{-1}$ in the susceptible parents at S_1. In the hybrids it ranged from 0.47 to 0.99 units $minute^{-1}$ (Table 3). At the S_2 the uninoculated genotypes had minimum polyphenol oxidase activity of 0.80 units $minute^{-1}$ in the susceptible parents while in the resistant parents it was 3.04 units $minute^{-1}$ and in the hybrids significantly higher PPO activity (3.56 units $minute^{-1}$) .The PPO activity varied significantly among genotypes under both inoculated and uninoculated conditions in all the three plant phenological stages. Polyphenol oxidase activity of different inoculated and uninoculated genotypes increased

with the advanced plant stage. In inoculated genotypes PPO activity showed an initial rapid increase but decreased by the final stage. However, in resistant parents and hybrids the PPO activity increased with the advanced plant growth stage.

The role of phenols becomes clear when the enzymes polyphenol oxidase and peroxidase are studied together. Polyphenol oxidase (PPO) and peroxidase activities (units minute^{-1}) during the initial stages were more in the hybrids followed by resistant parents, but were lower in the susceptible parents (Tables 3 and 4). The activity increased at the S_2, S_3 and S_4 in all the uninoculated genotypes. The increase was greater in the resistant parents than in susceptible ones. Immediately after inoculation (S_2) and later the enzyme activity increased rapidly in resistant hybrids and resistant parents as compared with controlled uninoculated healthy plants and their activity increased until S_4. In the susceptible parents PPO activity decreased immediately and continued down. By S_4 the activity was down to zero except for peroxidase activity in S_2 and S_3 where it increased slightly immediately after inoculation. Similar results of higher enzyme activity were observed in sunflower cultivars resistant to charcoal rot (*Rhizoctonia bataticola* (Taub) Butler) as compared to susceptible varieties (Pathak et al., 1998), in pea cultivars resistant to powdery mildew (*Erisiphe polygonic* DC) than in susceptible cultivars (Guleria et al., 1998), in leaf rust of wheat (Sharma and Sharma, 1998), in buckeye rot of tomato (Singh et al., 1997) and for peroxidase in tomato cultivars resistant to *Fusarium* wilt than susceptible ones (Jagadeesh et al., 2002), higher (PPO) activity was observed in urd bean resistant variety PU-35 as compared to the susceptible T-9 variety (Malik et al., 2002). An increased level of polyphenol oxidase and peroxidase was found in the resistant than in susceptible material infected with chilli cucumber mosaic leading to the formation of more quinones and other oxidative products was also observed (Singh et al., 2003) Similarly with Yellow Vein Mosaic Virus in okra (Ahmed et al., 1992) and in *Fusarium* wilt in tomato increased peroxidase and PPO activity was found in resistant hosts following inoculation with *Fusarium oxysporum* (Retig, 1974).

Peroxidase activity ranged from 0.80 to 19.42 units minute^{-1} under uninoculated and 0.00 to 19.53 units minute^{-1} under inoculated conditions. The differences were significant among the genotypes as well as between inoculated and uninoculated conditions at all the three plant growth stages. Peroxidase activity increased with the plant growth stage. In inoculated susceptible genotypes the peroxidase activity increased rapidly in the initial stage but later decreased to 0.00 units minute^{-1}. However, in resistant parents and hybrids the peroxidase level increased with the plant growth stage.

Total phenols, ortho-dihydroxy phenols and enzyme activity were found generally, higher in the resistant parents and the hybrids irrespective of the growth stages. Their activity increased further after inoculation and continued untill S_4. The higher amounts of total phenols and ortho-dihydroxy phenols in the resistant parents and hybrids were accompanied by increased activities of PPO and peroxidase, resulting in more oxidation of phenolic substances to form more toxic quinones and other oxidative products. These oxidative products might be the key to combating the pathogen in the resistant host. On the other hand, lower amount of phenols and lower enzyme activities in the susceptible parents, failed to produce toxic quinones or other oxidative products to that extent as found in resistant hybrids and parents. In the later stages the enzyme activity was almost nil in the susceptible ones providing no protection against Fusarium wilt leading to death of the plants.

The increased peroxidase and polyphenol oxidase activity and changes in the phenolic constituents immediately after infection are normal responses of a host plant (irrespective of its ultimate reaction to disease) in putting up initial defense as observed and reported by Harbourne (1964). This mechanism breaks down in susceptible genotypes as found here probably due to lower synthesis of phenolic enzymes and substrates. However, it persists in the resistant hosts.

Table 1. Total phenol concentration (mg/g) in un-inoculated/inoculated chilli genotypes at different stages.

S. No.	Genotypes	Un-inoculated/ inoculated		Stage 1	Stage 2	Stage 3	Mean
	Resistant parents						
1.	Arka Lohit		UI	7.600	7.800	9.020	8.410
			I		7.580	6.510	7.045
2.	SH-C-1154		UI	6.900	7.450	9.420	8.435
			I		7.240	5.440	6.340
		Means	UI	7.250	7.630	9.220	
			I		7.410	5.980	
	Susceptible parents						
1.	Kashmir Long-1		UI	6.500	7.020	8.030	7.525
			I		6.210	6.400	6.305
2.	Pampori		UI	6.300	6.803	7.020	6.912
			I		6.000	5.230	5.615
3.	SH-C-1-01		UI	4.000	4.900	5.320	5.110
			I		3.810	3.710	3.760
4.	SH-PC-1		UI	4.800	5.220	5.910	5.565
			I		4.210	4.220	4.215
5.	SH-C-405		UI	3.400	3.730	4.040	3.885
			I		3.050	3.240	3.145
6.	Local Chilli		UI	6.000	6.490	7.050	6.770
			I		5.650	5.720	5.685
		Means	UI	5.170	5.690	6.230	
			I		4.820	4.750	
	Hybrids						
1.	Arka Lohit x Kashmir Long-1		UI	9.800	10.400	7.860	9.130
			I		9.830	5.550	7.690
2.	Arka Lohit x Local Chilli		UI	8.920	9.960	7.140	8.550
			I		9.600	4.340	6.970
3.	Arka Lohit x SH-C-101		UI	7.850	8.797	6.830	7.813
			I		8.303	5.600	6.952
4.	Arka Lohit x SH-PC-1		UI	7.530	8.420	6.550	7.485
			I		8.090	4.810	6.450
5.	Arka Lohit x SH-C-405		UI	7.200	8.230	6.140	7.815
			I		7.810	4.030	5.920
6.	Arka Lohit x Pampori		UI	9.250	10.560	7.800	9.180
			I		9.800	3.420	6.610
7.	SH-C-1154 x Kashmir Long-1		UI	7.540	8.690	6.620	7.655
			I		8.330	4.740	6.535
8.	SH-C-1154 x Local Chilli		UI	8.530	9.820	6.240	8.030
			I		9.100	3.310	6.205
9.	SH-C-1154 x SH-C-101		UI	7.600	8.430	5.030	6.730
			I		8.020	4.410	6.215
10.	SH-C-1154 x SH-PC-1		UI	7.200	9.540	5.740	7.640
			I		8.930	4.800	6.865
11.	SH-C-1154 x SH-C-405		UI	8.200	9.250	6.410	7.830
			I		7.840	4.620	6.230
12.	SH-C-1154 x Pampori		UI	8.360	9.450	6.220	7.835
			I		9.010	4.330	6.670
			UI	8.170	9.300	6.550	
		Means	I		8.720	4.500	

LSD= Genotypes (0.068***), Stage (0.021***), Inoculation (0.021***), Genotype x Stage (0.096***), Genotype x Inoculation (0.096***), Stage x Inoculation (0.030***), Genotype x Stage x Inoculation (0.136***)

Table 2. Orthodihydroxy phenol concentration (mg/g) in un-inoculated/inoculated chilli genotypes at different stages

S. No.	Genotype	Uninoculated/ Inoculated		Stage 1	Stage 2	Stage 3	Mean
	Resistant parents						
1.	Arka Lohit		UI	0.190	0.430	1.960	0.860
			I		0.450	1.946	0.875
2.	SH-C-1154		UI	0.170	0.390	1.420	1.290
			I		0.400	1.400	0.900
		Means	UI	0.18	0.41	1.69	
			I		0.43	1.67	
	Susceptible parents						
1.	Kashmir Long-1		UI	0.040	0.130	1.060	0.595
			I		0.090	0.470	0.280
2.	Pampori		UI	0.040	0.230	1.240	0.735
			I		0.110	0.390	0.250
3.	SH-C-1-01		UI	0.030	0.150	1.400	0.775
			I		0.080	0.190	0.135
4.	SH-PC-1		UI	0.020	0.110	1.020	0.565
			I		0.060	0.430	0.245
5.	SH-C-405		UI	0.020	0.150	1.060	0.605
			I		0.050	0.500	0.275
6.	Local Chilli		UI	0.030	0.200	1.620	0.910
			I		0.090	0.710	0.400
		Means	UI	0.03	0.16	1.23	
			I		0.28	0.45	
	Hybrids						
1.	Arka Lohit x Kashmir Long-1		UI	0.220	0.740	1.790	1.265
			I		0.700	1.740	1.220
2.	Arka Lohit x Local Chilli		UI	0.250	0.670	1.050	0.860
			I		0.650	1.100	0.875
3.	Arka Lohit x SH-C-101		UI	0.270	0.960	1.620	1.290
			I		0.930	1.650	1.290
4.	Arka Lohit x SH-PC-1		UI	0.440	1.020	1.920	1.500
			I		1.000	1.940	1.470
5.	Arka Lohit x SH-C-405		UI	0.550	1.100	1.960	1.530
			I		1.210	1.990	1.600
6.	Arka Lohit x Pampori		UI	0.330	0.750	1.430	1.090
			I		0.800	1.510	1.155
7.	SH-C-1154 x Kashmir Long-1		UI	0.210	0.680	1.720	1.200
			I		0.690	1.700	1.195
8.	SH-C-1154 x Local Chilli		UI	0.230	0.590	1.000	0.795
			I		0.540	1.020	0.780
9.	SH-C-1154 x SH-C-101		UI	0.220	0.870	1.650	1.260
			I		0.820	1.640	1.230
10.	SH-C-1154 x SH-PC-1		UI	0.390	0.920	1.490	1.205
			I		0.900	1.470	1.185
11.	SH-C-1154 x SH-C-405		UI	0.450	0.990	1.330	1.160
			I		0.930	1.310	1.120
12.	SH-C-1154 x Pampori		UI	0.300	0.930	1.070	1.000
			I		0.900	1.050	0.975
			UI	0.32	0.85	1.50	
		Means	I		0.84	1.55	

LSD = Genotypes (0.035***), Stage (0.011***), Inoculation (0.011***), Genotype x Stage (0.050***), Genotype x Inoculation (0.050***), Stage x Inoculation (0.016***), Genotype x Stage x Inoculation (0.071***)

Table 3: Polyphenol oxidase activity (units/minute) in un-inoculated/inoculated chilli genotypes at different stages.

S. No.	Genotype	Uninoculated/ Inoculated		Stage 1	Stage 2	Stage 3	Stage 4	Mean
	Resistant parents							
1.	Arka Lohit		UI	0.160	3.040	3.520	5.200	3.920
			I		4.920	4.210	5.150	4.760
2.	SH-C-1154		UI	0.120	2.680	3.440	5.060	3.727
			I		4.030	4.220	5.300	4.517
		Means	UI	0.140	2.860	3.480	5.130	
			I		4.480	4.220	5.230	
	Susceptible parents							
1.	Kashmir Long-1		UI	0.080	2.920	3.210	4.200	3.443
			I		1.080	1.540	0.000	0.873
2.	Pampori		UI	0.060	2.560	3.450	4.590	3.533
			I		1.920	1.950	0.000	1.290
3.	SH-C-1-01		UI	0.060	2.080	3.240	3.960	3.093
			I		1.120	1.440	0.000	0.853
4.	SH-PC-1		UI	0.020	2.470	3.350	4.180	3.333
			I		1.860	1.900	0.000	1.253
5.	SH-C-405		UI	0.020	2.350	3.050	4.250	3.217
			I		1.450	1. 670	0.000	1.040
6.	Local Chilli		UI	0.020	2.460	3.240	4.130	3.277
			I		1.330	1.480	0.000	0.937
		Means	UI	0.040	2.470	3.260	4.220	
			I		1.460	1.660	0.000	
	Hybrids							
1.	Arka Lohit x Kashmir Long-1		UI	0.860	2.840	3.600	5.560	4.000
			I		3.840	4.400	5.450	4.563
2.	Arka Lohit x Local Chilli		UI	0.690	3.560	3.840	5.220	4.207
			I		4.640	4.990	5.070	4.900
3.	Arka Lohit x SH-C-101		UI	0.530	2.560	3.880	5.310	3.917
			I		3.440	5.150	5.120	4.570
4.	Arka Lohit x SH-PC-1		UI	0.990	2.440	3.840	5.470	3.917
			I		3.210	4.640	5.360	4.403
5.	Arka Lohit x SH-C-405		UI	0.630	2.600	3.320	5.190	3.703
			I		3.990	4.440	5.000	4.463
6.	Arka Lohit x Pampori		UI	0.960	3.040	3.400	5.340	3.927
			I		3.580	4.440	5.320	4.433
7.	SH-C-1154 x Kashmir Long-1		UI	0.65.0	2.560	3.280	5.110	3.650
			I		3.410	3.800	5.140	4.117
8.	SH-C-1154 x Local Chilli		UI	0.52.0	2.640	3.480	5.060	3.727
			I		3.040	4.000	5.080	4.040
9.	SH-C-1154 x SH-C-101		UI	0.610	2.200	3.920	5.240	3.787
			I		3.160	4.040	5.140	4.113
10.	SH-C-1154 x SH-PC-1		UI	0.530	2.800	3.240	5.330	3.790
			I		3.440	4.200	5.200	4.280
11.	SH-C-1154 x SH-C-405		UI	0.470	2.280	3.240	5.110	3.543
			I		3.330	4.080	5.210	4.207
12.	SH-C-1154 x Pampori		UI	0.640	2.880	3.600	5.330	3.937
			I		3.160	4.080	5.630	4.290
		Means	UI	0.670	2.700	3.550	5.270	
			I		3.520	4.360	5.230	

LSD= Genotypes (0.027***), Stage (0.10**), Inoculation (0.008***), Genotype x Stage (0.046***), Genotype x Inoculation (0.038***), Stage x Inoculation (0.014***), Genotype x Stage x Inoculation (0.065***)

Table-4: Peroxidase activity in un-inoculated/inoculated chilli genotypes at different stages (units/minute)

S. No.	Genotype	Uninoculated/ Inoculated		Stage 1	Stage 2	Stage 3	Stage 4	Mean
	Resistant parents							
1.	Arka Lohit		UI	0.700	0.900	3.450	16.450	6.933
			I	-	0.890	3.250	16.900	7.013
2.	SH-C-1154		UI	0.650	0.950	3.230	15.010	6.397
			I	-	0.820	3.150	15.453	6.474
		Means	UI	0.680	0.930	3.340	15.370	
			I		0.860	3.200	16.170	
	Susceptible parents							
1.	Kashmir Long-1		UI	0.700	0.920	2.990	9.300	4.403
			I	-	1.830	3.990	0.000	1.940
2.	Pampori		UI	0.680	0.800	3.410	8.200	4.403
			I	-	1.950	3.990	0.000	1.940
3.	SH-C-101		UI	0.700	1.100	4.020	9.700	4.940
			I	-	2.250	4.700	0.000	2.317
4.	SH-PC-1		UI	0.450	0.950	2.820	7.200	3.657
			I	-	2.100	3.960	0.000	2.020
5.	SH-C-405		UI	0.550	8.830	3.010	8.420	4.087
			I	-	2.990	3.680	0.000	2.223
6.	Local Chilli		UI	0.770	0.850	3.780	8.330	4.320
			I	-	2.920	3.647	0.000	2.189
		Means	UI	0.640	0.910	3.340	8.530	
			I		2.340	3.990	0.000	
	Hybrids							
1.	Arka Lohit x Kashmir Long-1		UI	3.750	5.500	6.540	14.500	8.847
			I	-	6.800	7.420	14.590	9.603
2.	Arka Lohit x Local Chilli		UI	3.200	4.000	7.050	15.210	8.753
			I	-	5.250	8.350	15.290	9.630
3.	Arka Lohit x SH-C-101		UI	3.000	3.550	6.800	18.530	9.627
			I	-	5.100	8.450	18.710	10.753
4.	Arka Lohit x SH-PC-1		UI	2.850	3.550	5.740	16.740	8.677
			I	-	5.100	6.350	16.900	9.250
5.	Arka Lohit x SH-C-405		UI	3.350	3.600	5.020	19.420	9.347
			I	-	4.000	6..150	19.530	9.893
6.	Arka Lohit x Pampori		UI	3.500	4.300	5.050	15.200	8.183
			I	-	5.950	8.000	15.220	9.723
7.	SH-C-1154 x Kashmir Long-1		UI	3.050	3.500	5.260	13.310	7.357
			I	-	4.580	7.040	13.370	8.330
8.	SH-C-1154 x Local Chilli		UI	3.250	4.050	6.740	14.730	8.507
			I	-	5.230	8.850	14.810	9.730
9.	SH-C-1154 x SH-C-101		UI	2.120	3.100	5.850	16.850	8.600
			I	-	5.230	7.850	16.900	9.993
10.	SH-C-1154 x SH-PC-1		UI	3.320	3.920	5.280	15.410	8.203
			I	-	4.880	6.690	15.480	9.009
11.	SH-C-1154 x SH-C-405		UI	3.050	3.620	5.050	15.440	8.037
			I	-	4.880	6.690	15.480	9.017
12.	SH-C-1154 x Pampori		UI	3.000	3.340	5.950	15.410	8.233
			I	-	4.720	6.800	15.420	8.980
		Means	UI	3.120	3.840	5.860	15.900	
			I		5.190	7.390	15.980	

LSD= Genotypes (0.038***), Stage (0.014**), Inoculation (0.012***), Genotype x Stage (0.066***), Genotype x Inoculation (0.054***), Stage x Inoculation (0.020***), Genotype x Stage x Inoculation (0.093***)

From the biochemical results it can be concluded that there is a positive correlation between host resistance and the amount of phenols and increased enzyme activity. The opposite occurs in the susceptible plants. The positive association of higher phenols and enzyme activity with resistance could be of value for early identification of resistant genotypes during population screening.

REFERENCES

Ahmed N., Thakur, M.R., Bajaj, K.L., Cheema, S.S. (1992). Biochemical basis of resistance to YVMV disease in okra. *Plant Disease Research* 9, 20–25.

Borua, I., Das, P. (2000). Changes in activities of polyphenol oxidase, acid phosphatase and phenol content in developing chilli varieties susceptible and resistant to *Collectotrichum capsici*. *Crop Research (Hissar)* 19(2): 230–234

Box, G.E., Hunter, W.G., Hunter, J.S. (1978). *Statistics for Experimenters*. New York: John Willey and Sons.

Bray, H.G., Thorpe, W.V. (1954). Analysis of phenolic compounds of interest in metabolism. In: Glick, D. (Ed.) *Methods of Biochemical Analysis*, Interscience Publishers, New York, USA 1, 27–52.

Gachomo, E.W., Shonukam, O.O., Kotchoni, S.O. (2003). The molecular initiation and subsequent acquisition of disease resistance in plants. *American Journal Biotechnology* 2, 26–32

Guleria, S., Paul. B., Bajaj, K.L (1998). Level of phenol metabolising enzymes in powdery mildew (*Ersiphe polygoni* DC) of pea. *Plant Disease Research* 13, 181–183

Harbourne, J.B. (1964). *Biochemistry of phenolic compounds*. Academic Press, London, New York.

Jagadeesh, S., Banerjee, M.K., Chakraborty, S., Kallo, G., Singh, J. (2002). Role of phenolics and peroxidase in resistance to Fusarium wilt in tomato (*Lycoperiscon esculentum* Mill). *Annals of Agri-Bio-Research* 7(1), 41–46

Link, K.P., Angel, H.R., Walker, J.C. (1929). The isolation of protocatechuic acid from pigmented onion scales and its significance in relation to disease resistance in onion. *Journal of Biological Chemistry* 81, 368.

Link, K., Walker, J.C. (1933). The isolation of catechol from pigmented onion scales and its significance in relation to disease resistance in onion. *Journal of Biological Chemistry* 100, 379–383.

Mahadevan, A., Sridhar, R. (1986). *Methods in Physiological Plant Pathology, 3rd ed*. Sivakami Publications, Chennai.

Malik, S., Kumar, P., Panwar, J.D.S., Anjali, Rathi, Y.P.S. (2002). Physiological and biochemical alterations induced by urdbean Leaf Crinkle Virus in *Vigna mungo* (L.) Hepper. *Annual Plant Protection Science* 10(1), 91–94.

Mohan, R., Alagianagalingam, M.N., Govindaswamy, C.V. (1978). Changes in phenolics and soluble carbohydrates in cowpea varieties infected by *Xanthomonas vignicola* Burk. *Current Science* 47, 431–432

Pathak, D., Srivastava, M.P., Deka, S.C. (1998). Biochemical basis of charcoal rot [*Rhizoctonia bataticola* Taub. (Butlur)] resistant and susceptible cultivars of sunflower in relation to total phenols free amino acids; insoluble pectin and polyphenoloxidase activity. *Annals of Biology Ludhiana* 14, 189–193.

R Development Core Team (2009). *R: A language and environment for statistical computing*. R Foundation for Statistical Computing, Vienna, Austria. ISBN 3-900051-07-0, URL http://www.r-project.org.

Retig, N. (1974). Changes in peroxidase and polyphenol oxidase associated with natural and induced resistance of tomato to Fusarium wilt. *Physiology and Plant Pathology* 4, 145–150.

Sharma, A.K., Sharma, S.K. (1998). Peroxidase and polyphenol oxidase activity changes in relation to leaf rust of wheat. *Journal of Maharashtra Agricultural Universities* 22, 286–291.

Sindhan, G.S., Jaglan, B.S. (1987). Role of phenolic compounds and carbohydrates in resistance of groundnut to tikka leaf spot. *Indian Journal of Mycology and Plant Pathology* 17, 141–144

Singh, R., Hundal, J.S., Chawla, N. (2003). Evaluation of chilli (*Capsicum annuum*) genotypes for quality components. *Indian Journal of Agricultural Sciences* 73(1), 51–53.

Singh, Y., Jamwal, R.S., Pathania, N.K., Kalia, P. (1997). Inheritance of biochemical traits in relation to buckeye fruit rot (*Phytophthora nicotianae* var. *parasitica*) in tomato. *Indian Journal of Hill Farming* 10, 10–15