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ASSESSMENT OF RESISTANCE TO THE PATHOGEN: SPECIAL REFERENCE TO ASCOCHYTA BLIGHT OF PEA

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Abstract

Pea (Pisumsativum L.) occupies a position of considerable importance as edible leguminous crop grown throughout the world. It occupies an area of 1.09 million hectares with an annual production of 8.26 million tonnes in the world. It is very nutritious as it contains high proportion of digestible proteins, carbohydrates, vitamins, along with minerals and consumed as fresh vegetable as well as pulse crop in the country. In India, pea is cultivated in an area of 282 thousand hectares with an annual production of 2.2 million tonnes. The taxonomic position of Ascochyta species on the morphological and pathological basis has been controversial for long, often leading to misidentification and hence erroneous control measures. Morphological criteria are highly variable, overlapping and influenced by environmental conditions making disease diagnosis a prolonged and cumbersome process. Accurate pathogen identification is most important to understand the intricacies of host pathogen relationship so as to implement judicious and sustainable disease management strategies. Molecular techniques are employed these days to differentiate the diverse species existing in a genus. These techniques are also employed to study variability existing within a pathogen. Therefore, variation existing in Ascochyta spp. attacking peas leading to blight complex can be studied through use of these techniques. Lack of monogenic resistance has greatly impaired the efforts in breeding disease resistant varieties in majority of crops including pea.

1. INTRODUCTION

Several Ascochyta species have been found to be associated with blights of many commercially important crops including edible legumes [1]. Of these, Ascochytapisi Lib., Ascochytapinodes L.K. Jones (syn. Didymellapinodes (Berk and Blox) Petrak; Mycosphaerellapinodes (Berk and Blox) Vestergn.) and Ascochytapinodella L.K. Jones (syn. PhomamedicaginisMalbr. Raum var. pinodella (Jones) Boerema; syn. Phomapinodella (L.K. Jones) Morgan Jones and K.B. Burch)

cause blight, leaf spot, pod blight and foot rot of pea, respectively [2]. A new species; Ascochytapisarum sp. nov.causing a new blight of pea in Himachal Pradesh. The taxonomic position of Ascochyta spp. infecting pea has been debated and is still ambiguous. It is considered Mycosphaerellapinodes to be ascigerous stage of A. pisi until it was pointed that disease caused by M. pinodes differed markedly from A. pisi [3]. It further contested the taxonomic position of Ascochytaspp and separated M. pinodes and Phomamedicaginis var. pinodella from A. pisi in his comprehensive and lucid description [4]. Recently it advocated transfer of M. pinodes to Didymellapinodes as nucleotide sequences of this fungus were more closely related to Didymellateleomorphs of other Ascochyta pathogens than Mycosphaerella.

The aforesaid species have been identified on the basis of symptoms by many workers, with A. pisi causing lighter brown leaf spots, A. pinodes; darker brown leaf and pod spots and A. pinodella; foot rot of pea. However, in many instances the symptoms overlap, as both A. pinodes and A. pinodella often cause foot rot and similar spots on leaves [5]. Correct identification becomes further problematic and error prone at early phases of disease expression often leading to misidentification and thus erroneous control measures. Similarly, identification by cultural and morphological characters like (i) size of pycnidia (ii) shape, size and septation of conidia (iii) presence/absence of chlamydospores have also been uncertain because of minute differences thus leading to wrong identification [6].

2. MATERIALS AND METHODS

2.1 Evaluation of pea germplasm

Four hundred and forty two pea genotypes were evaluated against Ascochytapinodes using detached leaf method. Three leaves per plant were placed in 11 cm diameter Petri plates lined with moist blotting sheets.

2.2 Evaluation of pea mutants

To evaluate resistance against Ascochytapinodes one hundred and twenty one mutants of different generations procured from Department of Vegetable Sciences and Floriculture were screened by detached leaf method.

2.3 Evaluation of induced resistance

In order to study induction of systemic acquired resistance an experiment was conducted under controlled environmental conditions in a growth chamber at 18^{o}C + 2^{o}C temperature with 12 hours of light and dark period. Healthy peas were sown in trays having sterilized potting mixture.

3. ASSESSMENT OF RESISTANCE TO THE PATHOGEN

3.1 Evaluation of pea genotypes for disease resistance

The screening of three hundred and sixty seven pea genotypes collected from various sources by detached leaf method are listed in Table 1. All the genotypes were found to be susceptible to Ascochytapinodes indicating lack of diversity for resistance.

3.2 Evaluation of pea mutants

To evaluate resistance against Ascochytapinodes one hundred and twenty one mutants of different generations derived from Azad Pea-1 viz. [7], AP-0.2-2, AP-0.2-3-R, AP-0.3-4, AP-0.2-5, AP-0.2-6-1, AP-0.2-8-1, AP-0.2-9, AP-0.2-33-1, AP-0.2-07-177-1, AP-0.2-07-283-1, AP-0.2-07-320-1, AP-0.2-07-400-1, AP-0.2-07-567-1, AP-0.3-483-1, AP-0.3-503-3-2, AP-0.3-588-1, AP-7.5-07K-47-1, AP-7.5-07K-87-1, AP-7.5-07K-133-1, AP-7.5-07K-144-1, AP-15-bulk-3, AP-15-b-3, AP-15-b-2-12, AP-15-4-1, AP-15-b-3-1, AP-15-b2-10/2, AP-15-59-1, AP-15-0.7K-1-53-1, AP-15-0.7K-1-53-2, AP-15-0.7K-1- 97-2, AP-15-0.7K-1-201-1, AP 15-0.7K-1-377-1, AP-15-0.7K-1-377-2, AP-15-0.7K-1-377-3, AP-15-0.7K-1-393-1, AP20-1, AP-20-4-1, AP-20-05-2, AP-20-B-6-1, AP-20-22, AP-20-7-2, AP-20-62-2, AP-20- 65-3 and Arkel viz., A-0.2-2, A-0.2-143-1, A-0.2-33-1, A-0.3, A-0.3-1, A-0.3-1-1, A-0.3- 1-5, A-0.3-1-5-2, A-0.3-purple, A-0.3-1-5, A-0.3-4, A-0.3-3 A-0.3-06, A-0.3-483-1, A0.3-503-3-2, A-0.3-588-1, A-0.3-18-2, A-0.3-M5, A-0.3-1-1, A-0.3-1-5, A-0.3-06k-2-1, A-0.3-06K-6, A-0.3-06k-9-1, A-0.3-06k-16-1, A-0.3-06k-23-1, A-0.3-06k-31-1, A-0.3- 06k-34-1, A-0.3-06k-42-1, A-0.3-06k-52-1, A-0.3-06k-93-1, A-0.3-06k-102-1, A-0.3- 06k-113-1, A-0.3-06k-120-1, A-0.3-06k-130-1, A-0.3-06k-131-1, A-0.3-06k-300-1, A0.3-06k-122-1, A-0.3-06k-126-1, A-0.3-06k-144-1, A-0.3-06k-222-1, A-0.3-06k-226-1, A-0.3-06k-244-1, A-0.3-06k-251-1, A-0.3-06k-256-1, A-0.3-06k-256-2, A-0.3-06k-

302- 1, A-0.3-06k-321-1, A-0.3-06k-468-1, A-0.3-06k-349-1, A-0.3-06k-336-1, A-0.3-06k392-1, A-0.3-06k-393-1, A-0.3-06k-401-1-2, A-0.3-06k-270-1, A-0.3-06k-270-2, A0.3-06k-472-1, A-0.3-06k-273-1, A-0.3-06k-274-1, A-0.3-06k-475-3, A-0.3-06k-276- 1, A-0.3-06k-277-1, A-0.3-06k-478-1, A-0.3-06k-280-1, A-0.3-06k-481-1-1, A-0.3- 06k-481-1-2, A-0.3-06k-483-2, A-0.3-06k-487-1, A-0.3-06k-487-2, A-0.3-06k-492-2, A-0.3-06k-495-1, A-0.3-06k-497-1, A-0.3-06k-498-1, A-0.3-07k-197-1, A-0.3-07k528-1, A-0.3-07k-528-2, A-0.3-07k-438-1, A-0.3-07k-633-1-1. Azad Pea-1 viz., AP0.2-2, AP-0.2-3-R, AP-0.3-4, AP-0.2-5, AP-0.2-6-1, AP-0.2-8-1, AP-0.2-9, AP-0.2-33-1, AP-0.2-07-177-1, AP-0.2-07-283-1, AP-0.2-07-320-1, AP-0.2-07-400-1, AP-0.2-07-567- 1, AP-0.3-483-1, AP-0.3-503-3-2, AP-0.3-588-1, AP-7.5-07K-47-1, AP-7.5-07K-87-1, AP-7.5-07K-133-1, AP-7.5-07K-144-1, AP-15-bulk-3, AP-15-b-3, AP-15-b-2-12, AP15-4-1, AP-15-b-3-1, AP-15-b2-10/2, AP-15-59-1, AP-15-0.7K-1-53-1, AP-15-0.7K-1- 53-2, AP-15-0.7K-1-97-2, AP-15-0.7K-1-201-1, AP-15-0.7K-1-377-1, AP-15-0.7K-1- 377-2, AP-15-0.7K-1-377-3, AP-15-0.7K-1-393-1, AP-20-1, AP-20-4-1, AP-20-05-2, AP-20-B-6-1, AP-20-22, AP-20-7-2, AP-20-62-2, AP-20-65-3 and Arkel viz., A-0.2-2, A-0.2-143-1, A-0.2-33-1, A-0.3, A-0.3-1, A-0.3-1-1, A-0.3-1-5, A-0.3-1-5-2, A-0.3- purple, A-0.3-1-5, A-0.3-4, A-0.3-3 A-0.3-06, A-0.3-483-1, A-0.3-503-3-2, A-0.3-588-1, A-0.3-18-2, A-0.3-M5, A-0.3-1-1, A-0.3-1-5, A-0.3-06k-2-1, A-0.3-06K-6, A-0.3-06k-9- 1, A-0.3-06k-16-1, A-0.3-06k-23-1, A-0.3-06k-31-1, A-0.3-06k-34-1, A-0.3-06k-42-1, A-0.3-06k-52-1, A-0.3-06k-93-1, A-0.3-06k-102-1, A-0.3-06k-113-1, A-0.3-06k-120-1, A-0.3-06k-130-1, A-0.3-06k-131-1, A-0.3-06k-300-1, A-0.3-06k-122-1, A-0.3-06k-126- 1, A-0.3-06k-144-1, A-0.3-06k-222-1, A-0.3-06k-226-1, A-0.3-06k-244-1, A-0.3-06k251-1, A-0.3-06k-256-1, A-0.3-06k-256-2, A-0.3-06k-302-1, A-0.3-06k-321-1, A-0.3- 06k-468-1, A-0.3-06k-349-1, A-0.3-06k-336-1, A-0.3-06k-392-1, A-0.3-06k-393-1, A0.3-06k-401-1-2, A-0.3-06k-270-1, A-0.3-06k-270-2, A-0.3-06k-472-1, A-0.3-06k273-1, A-0.3-06k-274-1, A-0.3-06k-475-3, A-0.3-06k-276-1, A-0.3-06k-277-1, A-0.3- 06k-478-1, A-0.3-06k-280-1, A-0.3-06k-481-1-1, A-0.3-06k-481-1-2, A-0.3-06k-483-2, A-0.3-06k-487-1, A-0.3-06k-487-2, A-0.3-06k-492-2, A-0.3-06k-495-1, A-0.3-06k497-1, A-0.3-06k-498-1, A-0.3-07k-197-1, A-0.3-07k-528-1, A-0.3-07k-528-2, A-0.3- 07k-438-1, A-0.3-07k-633-1-1. These mutants procured from Department of Vegetable Sciences and Floriculture were screened by detached leaf method. None of the above mutants showed effective resistance and were susceptible.

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Table 1: List of pea genotypes evaluated for disease resistance

Accession No.	Accession No.	Accession No.	Accession No.	Accession No.	Accession No.
EC-329562	IC-199764		KP-1	PI413669	PalamPriya
EC-329554	IC-208366	LFP-430	PMR-31	PI142441	PG-3
EC-329570	IC-208378	LFP-431	HPPC-63	PI381132	HPG-7
EC-328761	IC-208379	LFP-432	DPP-107	PI404221	IPBB-4
EC-313635	IC-208399	LFP-433	Pb 29B	FP-258	DPP-63
EC-329572	IC-208395	LFP-434	DPP-LMR	9142-10-67	P-1291
EC-389376	IC-209118	LFP-435	KS-245	Bialspurilinclon	IPF-P-2-5
EC-292173	IC-209101	P-102	FP-207	DPR-66	IPFD-26
EC-328760	IC-212631	HFP-4	9160-29-5	IFDDI-10	NDP-25
EC-292171	IC-212669	P-1368	PMR-10	IPF-1-17	P-1401
EC-324129	IC-219028	DPP-60	HFP-8909	KDMR-663	P-1542
EC-292160	IC-218985	KFMR-622	KPMR-522	DMR-49	HMQ-22
EC-328758	IC-218998	AFW-166	HFP-9910	HFP-4	IPF-P-2-5
EC-329560	IC-267120	BUP-7839	KPML-678	DPR-63	IPFD-26
EC-292166	IC-267152	P-2112	NBP-1	KDMR-641	NDP-25
EC-329552	IC-267162	PPM-2	IPFP-2-6	IFP-93-131	P-1401
EC-32125	IC-267171	VRP-16	P-1401	DPR-21	P-1542
EC-329549	IC-268275	VRPMR	P-1542	Wide Aphaca	HMQ-22
EC-329561	IC-296737	Acacia	HMQ-22	KMMR-696	DDR-66
EC-329566	IC-374352	DPP89(A)	IPF-P-2-5	P-1799	HVDP-15
EC-329568	IC-386802	VRP-8	IPFD-26	P-1806	KDMR-129
EC-329753	IC-424893	DGP-164	NDP-25	P-1347	KFPD-1
EC-329579	IC-424894	DPP-100	P-1401	P-11743	P-1808
EC-334160	NIC-11184	HPPC-96	P-1542	DMR-48	P-1503
EC-412883	NIC-11183	DPP-362	HMQ-22	Milt	HFP-9910
EC-341907	NIC-11205	NDVP-104	DDR-66	DPR-23	DPP-49
EC-342007	NIC-11199	DPP-102	HVDP-15	HPG-1-1-1	PDR-67
EC-381858	LFP-48	FP-259	KDMR-129	DPR-67	P-264
EC- 381854	LFP-83	EC-38154	P-1368	P-264	DPP-63
EC-387115	LFP-84	PL-8	DPP-60	KFP-01	P-1291
EC-389377	LFP-89	NDVP-24	KFMR-622	DPP-53	IPF-P-2-5

Table 2: List of pea genotypes evaluated for disease resistance

Accession No.	Accession No.	Accession No.	Accession No.	Accession No.	Accession No.
EC-538004	LFP-305	Bonneville	AFW-166	P-1363	IPFD-26
EC-538009	LFP-362	DPP-362	BUP-7839	NPP-250	NDP-25
EC-548807	LFP-363	VP-5	P-2112	P-1301	DPP-4
EC-548809	LFP-393	NDVP-24	PPM-2	KDMR-67	DPP127R
EC-6620	LFP-413	VP-101	P-212B	IPF-2-10	PMR-4
EC-388602	LFP-417	PB-(B-14)	MPPC-75	IP-8005	KS-221
EC-385247	LFP-419	P-2111	P-1360	NDVP-10	LMR-4
EC-218997	LFP-420	JI1412	Spiti Local	DPP-168	PHPMR-1
EC 381865	LFP-421	T-10	Linclon	C-400	JI 2437
EC 381864	LFP-422	DPP-19	NDVP-8(B)	DPG-4	JI1210
EC 381860	LFP-423	Vn 53	PB-294	UU-11	Kinnauri
EC-341725	LFP-427	Vl-2436	MatarAgeta	IM-25	VP 8902
EC-381859	LFP-428	JI 2432	DPP113T	NDV-10	DPP-120
IC-32978	LFP-429	DPP-113	DPP-54	JDKP8	KS-245
IC -209123	LFP-430	PB- 87	VN-52	PB-29	JI-2433
EC-381855	LFP-429	VL-2436	P-1542	PMR-21	JI-1210
NDVP-4	EC507771	HPCC 63	Dpp-35	15MA-6	Vn-52
EC-381857	IC 199776	NDVP 86	3818-54	MA-6	DPP-13
PMP-21	EC6620	Sugar gaint	JI 2433	EC381864	PMP-21
VL-3	IC-208399	Little marble	IC-356172	VI 2434	Kc 286
NDVP-250	VKG-28187	JDKP 8	IC-212622	FP207	HPP-6
DPP-40	P-183	JI 24-34	IC2677171	9160-29-5	DPP 54
EC381853	HPCC-16	Matri Black	EC-398598	FP257	PMP 21
DPP-54	FP-206	PI 542XEP	IC396743	FP280	VP-8005
DPP-13	FP-258	Sugar Sprint	IC212669	9412-5-55	NDVP-10 (A)
VKP-2	9142-4-54-2	Mr. Big	Ks 221	FP-183	DPP-102-T
Sd-82	FP211	Alaska	Ks 245	FP-259	IC-32178
DPP-6	FP261	Sugar snap	KTP 4	FP-182	DPP-100
JI-1210	FP180	Green arrow	LMR-20	VL-3	
PB-29b	Acacia	DPP-LMR-41	DPP-89 (A)	9412-10-67	

3.3 Induction of resistance

Effect of different treatments on induction of resistance in pea against A. pinodes was studied

by measuring activity of two key enzymes viz., phenylalanine ammonia lyase (PAL) and peroxidase (PO) involved in plant defense and manifestation of systemic resistance [8]. Time taken for appearance of disease symptoms was recorded as a parameter to evaluate disease resistance [9].

The phenylalanine ammonia lyase activity upto 96 hours at six different time intervals after treatment with various induces as compared to untreated check is presented in Table 3. Initially highest PAL activity of 150.4 g t-cinnamic acid hrs^{-1} g fresh $weight^{-1}$ was recorded with 5 mM SA treated plants which was at par with 150.2 g t-cinnamic acid hrs^{-1} g fresh wt^{-1} in 3 mM SA. In all other treatments no significant change in PAL activity was observed as compared to untreated check. After 12 hours of treatment a significant increase in PAL activity was observed in all the cases in comparison to the untreated check (147.60) and was maximum in case of 5 mM SA (192.60) followed by 1 mM SA (154.40) and A. pinodes (154.40) inoculated plants. All other treatments were statistically at par with 153.60, 152.60 and 151.40 g t-cinnamic acid hrs^{-1} g fresh $weight^{-1}$ of PAL activity in 3 mM SA, A. rabiei inoculated and Ascochyta spp. (common bean) inoculated plants [7]. After 24 hrs of treatment this further increased in all cases with maximum in 5 mM SA (211.80) followed by 3 mM SA (188.20), 1 mM SA (161.40), A. pinodes (158.20), Ascochyta spp. (common bean) (155.0) and A. rabiei (154.80) inoculated plants.

Inoculations of treated plants at different time intervals of 0, 12, 24, 48, 72 and 96 hrs with test fungus revealed that there was a significant delay in appearance of symptoms with 3 mM SA also showing maximum mean PAL and PO activity as compared to plants inoculated with Ascochytapinodes only [10]. There was delay in appearance by 12 hrs in case of 3 mM SA when inoculation was made after 0 and 12 hr of treatments. Inoculation after 24 hr of treatment delayed the symptom expression further by 24 hrs with no disease expression thereafter in subsequent inoculations at 48, 72 and 96 hrs [7].

Table 3:Effect of SAR inducers on appearance of Ascochytapinodes symptoms

Treatment	Time of Inoculation (hrs)					
	0	12	24	48	72	96
Untreated	-	-	-	-	-	-
Inoculated with A. pinodes	48	48	48	48	48	48
Treated with 1mM SA	48	48	48	60	60	48
Treated with 3mM SA	60	60	72	-	-	-
Treated with 5mM SA	48	-	-	-	-	-
Inoculated with A. spp. (common bean)	48	48	48	48	48	48
Inoculated with A. rabiei	48	48	48	48	48	48

In case of 1 mM SA there was a delay in appearance of symptoms by 12 hours when inoculations were done after 48 and 72 hrs of treatment. In all other cases there was no delay in appearance of symptoms [11]. In 5 mM SA treatment no disease was recorded after 12 hours of treatment as there was injury (phytotoxicity) to plants which died after 96 hrs.

4. CONCLUSION

The screening of three hundred and sixty seven pea genotypes collected from various sources by detached leaf method revealed all the genotypes to be susceptible to Ascochytapinodes indicating lack of diversity for resistance. Several workers have also reported absence of resistance against this pathogen in different parts of the world. It is a well-established fact that acquired resistance in plants is induced in plants as a result of plant-pathogen interactions or by application of chemicals like salicylic acid (SA) or 2,6-dichloroisonicotinic acid. Salicylic acid is known to play a key role as an exogenous inducer in systemic acquired resistance. The preliminary results obtained in the present study also indicate that SA acid is a key inducer of systemic acquired resistance in pea against A. pinodes as phenylalanine ammonia lyase and peroxide activity was found to be high in SA treated pea plants. The PAL and peroxidase activity significantly increased after 12 hr of treatment with 3 and 5 mM SA as compared to the untreated check. The 5 mM SA caused the highest increase upto 24 hr, however, the activity of these two enzymes declined after it. The decline in activity of two enzymes after 12 hr with 5 mM SA was due to injury caused by it to leaves due to its phytotoxicity and complete death of plants was observed with it after 96 hr of treatment.

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