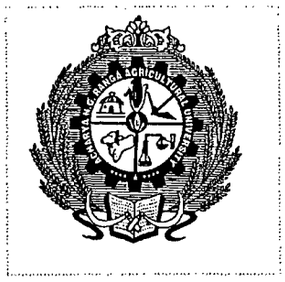
Studies on blast disease management in rice (*Oryza sativa* L.) incited by *Pyricularia grisea* Sacc

BY

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M. Sc (Ag.)

THESIS SUBMITTED TO THE
ACHARYA N.G. RANGA AGRICULTURAL UNIVERSITY
IN PARTIAL FULFILMENT OF THE REQUIREMENTS
FOR THE AWARD OF THE DEGREE OF

DOCTOR OF PHILOSOPHY
IN THE FACULTY OF AGRICULTURE



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2007

CERTIFICATE

Mr. G. Syamasundar Reddy has satisfactorily prosecuted the course of research and that, the thesis entitled "**Studies on blast disease management in rice (*Oryza sativa* L.) incited by *Pyricularia grisea* Sacc"** submitted is the result of original research work and is of sufficiently high standard to warrant its presentation to the examination. I also certify that, the thesis or part of thereof has not been previously submitted by him for a degree of any University.

Date : 06-9.07

Place : Hyderabad

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CERTIFICATE

This is to certify that the thesis entitled **"Studies on blast disease management in rice (*Oryza sativa* L.) incited by *Pyricularia grisea* Sacc"** submitted in partial fulfillment of the requirements for the degree of **"DOCTOR OF PHILOSOPHY IN AGRICULTURE"** of the Acharya N. G. Ranaga Agricultural University, Hyderabad, is a record of the bonafide research work carried out by **Mr. G. Syamasundar Reddy** under my guidance and supervision. The subject of the thesis has been approved by the Student's Advisory Committee.

No part of the thesis has been submitted for any other degree or diploma. The published part has been fully acknowledged. All the assistance and help received during the course of the investigation has been duly acknowledged by the author of the thesis.

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CONTENTS

Chapter I	Introduction	1
Chapter II	Review of Literature	5
Chapter III	Material & Methods	42
Chapter IV	Results	68
Chapter V	Discussion	247
Chapter VI	Summary	291
	Literature cited	296

LIST OF TABLES

S. No.	Title of the Table	Page No.
1	Details of *Pyricularia grisea* isolates collected from different parts of India	45
2	Details of fungicides employed in the investigation	55
3	Formulation and dosage details of fungicides used in the investigation for seed treatment and foliar spray	56
4	Mode of action and target sites of different fungicides employed in the investigation	66
5	Evaluation of rice cultivars for resistance against rice blast disease	70
6	Effect of systemic acquired resistance inducing chemicals on seed germination (%), *Kharif*, 2002	77
7	Effect of systemic acquired resistance inducing chemicals on seed germination (%), *Kharif*, 2003	81
8	Effect of systemic acquired resistance inducing chemicals on seed germination (%), *Kharif*, 2002 & 2003; pooled data	82
9	In vitro efficacy of systemic acquired resistance inducing chemicals against *Pyricularia grisea*	83
10	Effect of systemic acquired resistance inducing chemicals on blast severity (%) in the varieties HR-12, Swarnadhan and Rasi during *Kharif*, 2002	93
11	Effect of systemic acquired resistance inducing chemicals on blast severity (%) in the varieties HR-12, Swarnadhan and Rasi during *Kharif*, 2003	94
12	Effect of systemic acquired resistance inducing chemicals on blast severity (%) in the varieties HR-12, Swarnadhan and Rasi during *Kharif*, 2002 & 2003; pooled data	95
13	Effect of systemic acquired resistance inducing chemicals in the varieties HR-12, Swarnadhan and Rasi on different disease parameters during *Kharif*, 2002	111
14	Effect of systemic acquired resistance inducing chemicals in the varieties HR-12, Swarnadhan and Rasi on different disease parameters during *Kharif*, 2003	116

S. No.	Title of the Table	Page No.
15	Effect of systemic acquired resistance inducing chemicals in the varieties HR-12, Swarnadhan and Rasi on different disease parameters during *Kharif*, 2002 & 2003; pooled data	121
16	*In vitro* efficacy of different fungicides against *Pyricularia grisea*	127
17	Efficacy of fungicides as seed treatment against rice blast in nursery beds (*Kharif*, 2002)	136
18	Efficacy of fungicides as seed treatment against rice blast in nursery beds (*Kharif*, 2003)	141
19	Efficacy of fungicides as seed treatment against rice blast in nursery beds (*Kharif*, 2002 & 2003; pooled data)	146
20	Efficacy of fungicides as foliar spray against rice blast in nursery beds (*Kharif*, 2002)	155
21	Efficacy of fungicides as foliar spray against rice blast in nursery beds (*Kharif*, 2003)	160
22	Efficacy of fungicides as foliar spray against rice blast in nursery beds (*Kharif*, 2002 & 2003; pooled data)	165
23	Efficacy of fungicides as foliar spray against rice blast in the main field (*Kharif*, 2002)	172
24	Efficacy of fungicides as foliar spray against rice blast in the main field (*Kharif*, 2003)	179
25	Efficacy of fungicides as foliar spray against rice blast in the main field (*Kharif*, 2002 & 2003; pooled data)	186
26	Relative sensitivity of *Pyricularia grisea* isolates to carbendazim *in vitro*	197
27	*In vitro* sensitivity of three reference sensitive isolates of *Pyricularia grisea* to carbendazim	199
28	Relative sensitivity of *Pyricularia grisea* isolates to edifenphos *in vitro*	204
29	*In vitro* sensitivity of three reference sensitive isolates of *Pyricularia grisea* to edifenphos	207
30	Relative sensitivity of *Pyricularia grisea* isolates to iprobenfos *in vitro*	210
31	*In vitro* sensitivity of three reference sensitive isolates of *Pyricularia grisea* to iprobenfos	213

S. No.	Title of the Table	Page No.
32	Cross resistance of carbendazim resistant and sensitive isolates to other blasticides	২১৬
33	Cross resistance of edifenphos resistant and sensitive isolates to other blasticides	২২১
34	Cross resistance of iprobenfos resistant and sensitive isolates to other blasticides	২২৬
35	Persistence of acquired resistance in the resistant isolates after 12 serial transfers on OMA medium	২২৯
36	Efficacy of different fungicide combinations on carbendazim resistant isolate both *in vitro* and *in vivo*	২৩৪
37	Efficacy of different fungicide combinations on edifenphos resistant isolate both *in vitro* and *in vivo*	২৩৯
38	Efficacy of different fungicide combinations on iprobenfos resistant isolate both *in vitro* and *in vivo*	২৪৩

LIST OF FIGURES

S. No.	Title of the Figure	Page No.
1	Map showing the areas from which rice blast disease samples were collected in India and Andhra Pradesh state.	46
2	Disease reaction of different rice cultivars towards *Pyricularia grisea*	74
3	Disease reaction (0-9 scale) of different rice cultivars towards *Pyricularia grisea*	74
4	*In vitro* efficacy of systemic acquired disease resistance inducing chemicals against *Pyricularia grisea*	78
5	Effect SAR chemicals on seed germination (%) in the varieties HR-12, Swarnadhan and Rasi (*Kharif*, 2002 & 03)	78
6	Effect of SAR inducing chemicals on blast severity (%) at 5 DAI in the varieties HR-12, Swarnadhan and Rasi (*Kharif*, 2002&03)	96
7	Effect of SAR inducing chemicals on blast severity (%) at 10 DAI in the varieties HR-12, Swarnadhan and Rasi (*Kharif*, 2002&03)	96
8	Effect of SAR inducing chemicals on blast severity (%) at 15 DAI in the varieties HR-12, Swarnadhan and Rasi (*Kharif*, 2002&03)	97
9	Effect of SAR inducing chemicals on blast severity (%) at 20 DAI in the varieties HR-12, Swarnadhan and Rasi (*Kharif*, 2002&03)	97
10	Effect of SAR inducing chemicals as different treatments at 5 DAI on the varieties HR-12, Swarnadhan and Rasi (*Kharif*, 2002&03)	107
11	Effect of SAR inducing chemicals as different treatments at 10 DAI on the varieties HR-12, Swarnadhan and Rasi (*Kharif*, 2002&03)	107
12	Effect of SAR inducing chemicals as different treatments at 15 DAI on the varieties HR-12, Swarnadhan and Rasi (*Kharif*, 2002&03)	108
13	Effect of SAR inducing chemicals as different treatments at 20 DAI on the varieties HR-12, Swarnadhan and Rasi (*Kharif*, 2002&03)	108
14	Effect of SAR inducing chemicals in the variety HR-12 on different disease parameters (*Kharif*, 2002&03)	122
15	Effect of SAR inducing chemicals as different kind of treatments in the variety HR-12 (Kharif, 2002&03)	122

S. No.	Title of the Figure	Page No.
16	Effect of SAR inducing chemicals in the variety Swarnadhan on different disease parameters (*Kharif*, 2002&03)	123
17	Effect of SAR chemicals as different kinds of treatments in the variety Swarnadhan (*Kharif*, 2002&03)	123
18	*In vitro* efficacy of different fungicides against *Pyricularia grisea*	138
19	Progress of leaf blast severity in relation to efficacy of fungicides as seed treatment in nursery beds (*Kharif*, 2002&03)	147
20	Development of initial disease symptoms in the seed treated nursery beds (*Kharif*, 2002&03)	148
21	Progress of leaf blast severity in relation to efficacy of fungicides as foliar spray in the nursery beds (*Kharif*, 2002&03)	166
22	Progress of leaf blast severity in relation to efficacy of fungicides against leaf blast in the main field (*Kharif*, 2002&03)	187
23	Neck blast incidence in relation to efficacy of fungicides against rice blast in the main field (*Kharif*, 2002&03)	188
24	Grain yield (kg/ha) in relation to efficacy of fungicides against rice blast in the main field (*Kharif*, 2002&03)	189
25	Relative sensitivity of *Pyricularia grisea* isolates to carbendazim *in vitro*	198
26	Relative sensitivity of *Pyricularia grisea* isolates to edifenphos *in vitro*	205
27	Relative sensitivity of *Pyricularia grisea* isolates to iprobenfos *in vitro*	211
28	Cross resistance of Carbendazim resistant (Cuttak) and sensitive (Medchel) isolates to other blasticides (Mycelial growth)	217
29	Cross resistance of Carbendazim resistant (Cuttak) and sensitive (Medchel) isolates to other blasticides (Spore germination)	217
30	Cross resistance of Edifenphos resistant (Mandya) and sensitive (Pondicherry) isolates to other blasticides (Mycelial growth)	222
31	Cross resistance of Iprobenfos resistant (Maruteru) and sensitive (Pondicherry) isolates to other blasticides (Spore germination)	222
32	Persistence of acquired resistance in the resistant isolates after 12 serial transfers on OMA medium (Mycelial growth)	230

S. No.	Title of the Figure	Page No.
33	Persistence of acquired resistance in the resistant isolates after 12 serial transfers on OMA medium (Spore germination)	230
34	Efficacy of different fungicide combinations on carbendazim resistant isolate both *in vitro* and *in vivo* (ED_{50})	235
35	Efficacy of different fungicide combinations on edifenphos resistant isolate both *in vitro* and *in vivo* (ED_{50})	240
36	Efficacy of different fungicide combinations on iprobenfos resistant isolate both *in vitro* and *in vivo* (MIC)	244

LIST OF PLATES

S. No.	Title of the plate	Page No.
1	Leaf blast symptoms caused by *Pyricularia grisea* in the main field	47
2	Neck blast symptoms (broken necks) caused by *Pyricularia grisea*	47
3	Neck blast symptoms (lesioned necks) caused by *Pyricularia grisea*	47
4	Leaf blast symptoms caused by *Pyricularia grisea* in the nursery	48
5	Conidia of *Pyricularia grisea* (400X)	48
6	Laboratory culture of *Pyricularia grisea*	59
7	Mass multiplication of *P. grisea* on sorghum grain	59
8	Uniform Blast Nursery (UBN) pattern	60
9	Sprinklers used to increase the relative humidity	60
10	Screening of rice cultivars for host plant resistance against rice blast	61
11	Evaluation of fungicides as foliar spray against rice blast in the nursery	61
12	*In vitro* inhibition of mycelial growth of *P. grisea* by BTH	79
13	*In vitro* inhibition of mycelial growth of *P. grisea* by ferric chloride	79
14	*In vitro* inhibition of mycelial growth of *P. grisea* by sodium selenite	79
15	Evaluation of SAR inducing chemicals against rice blast in the glass house	98
16	Effect of BTH as seed treatment alone (1), seed treatment + one foliar spray (2) and seed treatment + two foliar sprays (3) in highly resistant variety, Rasi	98
17	Effect of salicylic acid as seed treatment alone (1), seed treatment + one foliar spray (2) and seed treatment + two foliar sprays (3) in moderately resistant variety, Swarnadhan	99
18	Effect of BTH as seed treatment alone (1), seed treatment + one foliar spray (2) and seed treatment + two foliar sprays (3) in moderately resistant variety, Swarnadhan	99

S. No.	Title of the plate	Page No.
19	Effect of salicylic acid as seed treatment alone (1), seed treatment + one foliar spray (2) and seed treatment + two foliar sprays (3) in highly susceptible variety, HR-12	100
20	Effect of BTH as seed treatment alone (1), seed treatment + one foliar spray (2) and seed treatment + two foliar sprays (3) in highly susceptible variety, HR-12	100
21	*In vitro* inhibition of mycelial growth of *P. grisea* at different concentrations of carbendazim 50 WP	129
22	*In vitro* inhibition of mycelial growth of *P. grisea* at different concentrations of pyraclostrobin 25 EC	129
23	*In vitro* inhibition of mycelial growth of *P. grisea* at different concentrations of diniconazole 25 WP	130
24	*In vitro* inhibition of mycelial growth of *P. grisea* at different concentrations of isoprothiolane 40 EC	130
25	*In vitro* inhibition of mycelial growth of *P. grisea* at different concentrations of edifenphos 50 EC	131
26	*In vitro* inhibition of mycelial growth of *P. grisea* at different concentrations of kasugamycin 2 L	131
27	*In vitro* inhibition of mycelial growth of *P. grisea* at different concentrations of pyroquilon 50 WP	132
28	*In vitro* inhibition of mycelial growth of *P. grisea* at different concentrations of tricyclazole 75 WP	132
29a	Evaluation of fungicides as seed treatment in the nursery beds	149
29b	Evaluation of fungicides as seed treatment in the nursery beds (contd.)	150
30	Evaluation of fungicides against rice blast in the main field	190
31	*In vitro* sensitivity of Tirupathi isolate towards carbendazim	200
32	*In vitro* sensitivity of Kadapa isolate towards edifenphos	200
33	*In vitro* sensitivity of Naidupeta isolate towards iprobenfos	200
34	Inhibition of mycelial growth by Cuttak isolate (highly resistant) at different concentrations of carbendazim	201

S. No.	Title of the plate	Page No.
35	Inhibition of mycelial growth by Medchel isolate (highly sensitive) at different concentrations of carbendazim	২০১
36	Inhibition of mycelial growth by Mandya isolate (highly resistant) at different concentrations of edifenphos	২০৬
37	Inhibition of mycelial growth by Pondicherry isolate (highly sensitive) at different concentrations of edifenphos	২০৬
38	Inhibition of mycelial growth by Maruteru isolate (highly resistant) at different concentrations of iprobenfos	২১২
39	Inhibition of mycelial growth by Pondicherry isolate (highly sensitive) at different concentrations of iprobenfos	২১২
40	Inhibition of mycelial growth of carbendazim resistant isolate (Cuttak) at different concentrations of pyraclostrobin	২১৪
41	Inhibition of mycelial growth of carbendazim sensitive (Medchel) isolate at different concentrations of pyraclostrobin	২১৪
42	Inhibition of mycelial growth of edifenphos resistant isolate (Mandya) at different concentrations of pyraclostrobin	২২৩
43	Inhibition of mycelial growth of edifenphos sensitive (Pondicherry) isolate at different concentrations of pyraclostrobin	২২৩
44	Comparative inhibition of mycelial growth of *P. grisea* by carbendazim 50 WP, edifenphos 50 EC and tricyclazole 75 WP at different concentrations	২৫৪

Acknowledgement

*I earnestly revere my beloved parents **Sri G. Chandra Reddy and Smt Dhanamma** for their boundless love and blessings which accompanied me in all my endeavours.*

*I deem it privilege to extol my profound and sincere feelings of gratitude to the Chairman of my Advisory Committee, **Dr. T. Vithal Reddy**, Professor & Head, Dept. of Plant Pathology, College of Agriculture, Rajendranagar, Hyderabad for his unflagging help, constructive criticism in planning and presentation of the investigation.*

*I take it as an extreme privilege to express my heartfelt thanks and sincere gratitude to the Co-chairman of my Advisory Committee, **Dr C. S. Reddy,** Principal Scientist & Head, Dept. of Plant Pathology, Directorate of Rice Research, Rajendranagar, Hyderabad for his help, guidance, cooperation and encouragement which have instilled in me the spirit of confidence to successfully complete this research work at DRR. Without his personal involvement at every stage, it would have never been possible to see this work in the present shape.*

*I humbly record my heart felt thanks to **Dr. T. Madhusudhan**, Principal Scientist (Rtd.), Pathology, Farmers' Call Center, ANGRAU, Hyderabad, member of my advisory committee for his keen interest, valuable guidance and constant encouragement during my research work and thesis preparation.*

*With profound and unfading sense of gratitude to express my sincere thanks to the member of my advisory committee, **Dr. M. Ganesh**, Principal Scientist & Head, National Seed Project, ANGRAU, Rajendranagar for his valuable advice and constant inspiration.*

*I wish to proffer my thanks to **Dr B. S. Kulkarni,** Professor & University Head, Dept. of Mathematics and Statistics, ANGRAU, Rajendranagar and member of my advisory committee for giving me valuable guidance and timely suggestions during the preparation of the manuscript.*

*I sincerely extend my gratitude to **Dr V. Krishna Rao** and **Dr P. Narayan Reddy,** both Professors in the Dept. of Plant Pathology, College of Agriculture, Rajendranagar for their kind cooperation and valuable suggestions throughout the course of investigation.*

*I would like to express my humble, most reverence, heartful respect and profound gratitude to **Dr. T. Narsi Reddy**, Professor and Head, Dept. of Plant Pathology for his kind cooperation, valuable advice.*

With deep sense of gratitude, I would like to convey my thanks to ***Dr. Muralidharan****, Principal Scientist,* ***Dr. Laha****,* ***Dr. Krishnaveni****, &* ***Dr. Srinivasa Prasad****, Senior Scientists and* ***Smt Susheela****, Research Associate, Department of Plant Pathology, DRR, Rajendranagar for their generous help and constant cooperation and guidance during the course of study. I also wish to express my sincere thanks to* ***Mr Prasad****,* ***Mr Roseswar Rao****,* ***Mr Ananth Reddy*** *and* ***other field staff*** *at DRR who helped me during the entire course of my research.*

Some rare, auspicious moments come in life when words are totally insufficient to express the heartfelt emotion. At this transitory phase, words foil my pen, to acknowledge those unique, valuable personalities, who has made a vital contribution in accomplishing this thesis work. I humbly place on record my respect and deep sense of gratitude to ***Dr P. Krishna Reddy,*** *Professor, IIIT, Hyderabad;* ***Dr B. Bhaskar Reddy,*** *Director (International Programmes), ANGRAU;* ***Dr M. Mallikarjun Reddy,*** *Dy. Director (Research). ANGRAU;* ***Dr N. P. Eswar Reddy & Dr T.V.N. Sastri,*** *Professors, S.V. Agricultural college, Tirupathi,* ***Dr B. Sahadeva Reddy*** *and* ***Dr B. Radhika,*** *Scientists, ANGRAU for their unparalleled affection, moral support and persistent encouragement to enrich my life.*

I am in dearth of words to express my feelings towards my beloved best friends ***Raji*** *and* ***Bujji*** *who shared my fears, tears and joys and tuned hopeless moments to wonderful things which put forth new tender leaves in my life. I feel it a rare opportunity to express my bountiful regards, affection and gratitude to my beloved brother, sisters, brothers-in-law who constantly inspired me to study.*

The wholehearted encouragement, cooperation and splendid company extended by my dear friends ***Parameswar, Vikram, Diyavar, Dinakar, Ashok, Sunil, Anil, Madhav, Rammohan, Umamahesh*** *and* ***Chandrasekhar*** *who helped me in their own sphere in many ways.*

I cordially concede my special gratitude to my colleagues at the International Institute of Information Technology, Hyderabad, ***Smt. Seetha, Gopalaswamy, Sucharitha, Karunakar, Jagadeesh, Sivasankar, Konda Reddy, Hari, Lavanya, Raghunathan, Uday, Kumaraswamy*** *and* ***Venugopal Reddy*** *for their help and co-operation during the course of thesis writing.*

I affectionately acknowledge the cooperation and encouragement of my ***Juniors*** *and* ***Seniors****, in whose presence I never felt my work a burden.*

Finally, a word of appreciation goes to ***Mr Shree Kanth*** *and* ***Mr Pradeep*** *for their computer work and statistical analysis, respectively.*

Date: 06.09.2007 ***(G. SYAMASUNDAR REDDY)***

DECLARATION

I, G. SYAMASUNDAR REDDY hereby declare that the thesis entitled **"Studies on blast disease management in rice (*Oryza sativa* L.) incited by *Pyricularia grisea* Sacc"** submitted to **ACHARYA N. G. RANGA AGRICULTURAL UNIVERSITY** for the degree of **DOCTOR OF PHILOSOPHY** in Agriculture is the result of original research work done by me. I also declare that the material contained in the thesis has not been published earlier.

Date : 05/09/2007

Place : Hyderabad

05/09/07

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ABSTRACT

Rice blast caused by *Pyricularia grisea* Sacc. is the most serious and damaging disease affecting rice production in temperate and humid countries including India. Different rice cultivars were evaluated for blast disease resistance against *P. grisea* during the years 2002 and 2003 by adapting Uniform Blast Nursery (UBN) pattern developed by the International Rice Research Institute (IRRI). Forty six cultivars were highly resistant (0-1 score), 32 were resistant (2-3 score), 15 were moderately resistant/susceptible (4-5 score), 19 were susceptible (6-7 score) and 33 were highly susceptible (8-9 score).

Green house experiments were conducted to assess the effect of selected SAR inducing chemicals *viz.* salicylic acid (10^{-3} M), BTH (10^{-3} M), ferric chloride (10^{-4} M), sodium selenite (10^{-5} M) and zinc sulphate (10^{-3} M) for inducing systemic acquired resistance against blast disease in HR-12, Swarnadhan and Rasi cultivars during *Kharif*, 2002 and *Kharif*, 2003. In the susceptible variety HR-12, BTH 10^{-3} M was superior in minimizing the leaf blast severity followed by salicylic acid 10^{-3} M. Sodium selenite 10^{-5} M and ferric chloride 10^{-4} M had shown some SAR inducing potential. The SAR inducing effect of zinc sulphate 10^{-3} M was negligible. The SAR inducing chemicals applied as seed treatment followed by either one foliar spray or two foliar sprays were found significantly superior to those of seed treatment alone. However, the 2nd spray did not show any significant effect in inducing any additional systemic resistance. In the moderately resistant variety Swarnadhan, BTH 10^{-3} M and salicylic acid 10^{-3} M were more successful in reducing the leaf blast severity, while the effect of ferric chloride 10^{-4} M, sodium selenite 10^{-5} M and zinc sulphate 10^{-3} M was found negligible. The SAR inducing capabilities of the chemicals did not differ with the method and number of applications *viz.* seed treatment alone, seed treatment followed by either one or two foliar sprays. In the highly resistant variety Rasi, disease incidence was observed neither in untreated control nor in chemical treatments and hence the SAR inducing capacity can not be assessed in Rasi due to its resistance against blast disease.

Among the fungicides tested, the highest *in vitro* efficacy was shown by those fungicides that affect mitosis and cell division (carbendazim 50 WP) followed by the ones that disrupt respiration (azoxystrobin 25 SC, pyraclostrobin 25 EC and RIL010 25 SC). On the other hand, melanin biosynthesis inhibitors *viz.* pyroquilon 50 WP, tricyclazole 75 WP and fenoxanil 20 SC have failed to inhibit mycelial growth *in vitro*. The next best control was offered by edifenphos 50 EC, iprobenfos 48 EC and isoprothiolane 40 EC whose mode of action is lipid and membrane synthesis. Sterol biosynthesis inhibitors *viz.* propiconazole 25 EC, epoxiconazole 7.5 EC, difenconazole 25 EC and diniconazole 25 WP were next in the order of their *in vitro* efficacy. The *in vitro* efficacy of the antifungal antibiotic kasugamycin was found as good as DMI-fungicides.

In the seed treatment experiments, pyroquilon 50 WP was found the best fungicide with 6.66 per cent leaf blast severity at 20 days after inoculation (DAI) followed by azoxystrobin 25 SC and pyraclostrobin 25 EC with 7.33 and 8.33 per cent disease severity, respectively when compared to cent per cent disease severity in the untreated control. The next best protection was offered by RIL010 25 SC, tricyclazole 75 WP and fenoxanil 20 SC with 11.16, 14.16 and 24.99 per cent disease severity, respectively. Carbendazim 50 WP and propiconazole 25 EC were comparatively good with 32.49 and 39.99 per cent disease severity, respectively. The remaining fungicides were not good enough as seed treatment when compared to their superior counterparts even though they performed far better than the untreated control.

In the nursery experiments where the fungicides were used as foliar sprays, all the fungicides tested were found significantly superior to untreated control upto 20 days after inoculation. The efficacy of pyroquilon 50 WP, azoxystrobin 25 SC, RIL 010 25 SC, pyraclostrobin 25 EC and tricyclazole 75 WP was excellent. Fenoxanil 20 EC, iprobenfos 48 EC, isoprothiolane 40 EC, edifenphos 50 EC, propiconazole 25 EC and kasugamycin 2 L were also found superior in controlling leaf blast incidence. Carbendazim 50 WP exhibited good amount of control compared to rest of the fungicides. Diniconazole 25 WP was the least effective of all the fungicides tested even though it was significantly superior over control.

In the main field, the supremacy of pyroquilon 50 WP was inadvertently continued in controlling both leaf blast severity and neck blast incidence. The same trend was also continued in increasing the total grain yield while reducing the quantity of ill-filled grain. The performance of tricyclazole 75 WP and fenoxanil 20 SC was next only to azoxystrobin 25 SC, pyraclostrobin 25 EC and RIL 010 25 SC. There were 2.66 per cent of lesioned necks with out any broken ones in the plots treated with tricyclazole 75 WP while fenoxanil 20 SC produced 4 per cent lesioned necks and 1.66 per cent broken necks. Conversely, pyroquilon 50 WP had produced neither lesioned necks nor broken necks against 21.66 per cent lesioned necks and 17.33 per cent broken necks in the untreated control. The highest grain yield of 2.9 t/ha was obtained from the plots treated with pyroquilon 50 WP as against 1.8 t/ha from the untreated control plots.

Rice blast samples were collected from 12 different parts of India including Andhra Pradesh to test the sensitivity of *P. grisea* towards carbendazim, edifenphos and iprobenfos *in vitro*. For carbendazim, Cuttak isolate recorded the highest ED_{50} values of *54.63 and 56.18 µg/ml for mycelial growth (poisoned food) and spore germination*, respectively. Medchel isolate recorded the lowest ED_{50} values of 0.39 and 0.55 µg/ml for inhibition of mycelial growth (poisoned food) and spore germination, respectively. For

edifenphos, Mandya isolate recorded the highest ED_{50} values of 30.56 and 32.35 µg/ml for mycelial growth (poisoned food) and spore germination, respectively. Pondicherry isolate recorded the lowest ED_{50} values of 0.73 and 0.77 µg/ml for mycelial growth and spore germination, respectively. For iprobenfos, Maruteru isolate recorded the highest ED_{50} values of 31.85 and 32.57 µg/ml for mycelial growth and spore germination, respectively. For iprobenfos also, Pondicherry isolate recorded the lowest ED_{50} values of 0.66 and 0.68 µg/ml for mycelial growth and spore germination, respectively.

Development of resistance towards edifenphos has contributed to the development of positive cross resistance not only to iprobenfos but also to isoprothiolane. Similarly, development of resistance towards iprobenfos had also contributed towards the tolerance of edifenphos and isoprothiolane. Development of resistance to either edifenphos or iprobenfos had no correlation with the fungicides *viz.* pyraclostrobin, kasugamycin and carbendazim. Similarly, development of resistance towards carbendazim had no correlation with the fungicides *viz.* pyraclostrobin and kasugamycin. The carbendazim resistant isolate had shown partial resistance towards iprobenfos, isoprothiolane and edifenphos. The isolates of *P. grisea* were grown in oat meal agar medium for 12 successive generations to study the persistence of acquired resistance. The results show that the resistance to carbendazim was lost very rapidly when compared to those of edifenphos and iprobenfos.

Tricyclazole 75 WP and BTH 50 WG did not show any inhibitory effect on the fungus *in vitro* individually, while mancozeb 75 WP showed the highest inhibition of both mycelial growth and spore germination when compared to carbendazim 50 WP, kasugamycin 2L and propiconazole 25 EC. But, tricyclazole 75 WP recorded the lowest blast severity of 20.67 per cent as compared to 66.67 per cent in mancozeb 75 WP and 64.67 per cent in BTH 50 WG. There was significant reduction in leaf blast severity due to the combination of kasugamycin 2L, propiconazole 25 EC and mancozeb 75 WP with carbendazim 50 WP.

LIST OF ABBREVIATIONS

%	:	Per cent
µg/ml	:	microgram per milliliter
@	:	at the rate of
BTH	:	Acebenzolar-S-methyl 50 WG
C	:	celsius
CD	:	Critical Difference
cm	:	centimeters
DAS	:	days after sowing
DASP	:	days after spraying
EC	:	Emulsifiable Concentrate
ED_{50}	:	Effective Dose 50
et al.	:	and others
Fig.	:	Figure
g	:	grams
h	:	hours
i.e.	:	that is
l	:	liter (s)
m	:	meters
mg	:	milligrams
mg/l	:	milligrams per liter
MIC	:	minimum inhibitory concentration
ml	:	milliliter
mm	:	millimeter
no.	:	number
OMA	:	Oat Meal Agar
P (0.05)	:	CD at 5% level
PDA	:	Potato Dextrose Agar
ppm	:	parts per million
psi	:	pounds per square inch
SC	:	Soluble concentrate
SE	:	Standard error
WG	:	Water dispersible granules
WP	:	Wettable powder

Introduction

CHAPTER I

INTRODUCTION

Rice is the world's most important staple food crop. More than half of the world's population relies on rice as the major daily source of calories and protein. Rice is being cultivated in about 151 m. ha around the world accounting to the total production of 597.8 m. t. of paddy grain with an average yield of 4 tonnes ha^{-1}, while in India, rice is grown in an area of 42.8 m. ha accounting to the total production of 129.2 m. t. of paddy grain with an average yield of 3 tonnes ha^{-1} (FAO, 2006). In Andhra Pradesh, rice is grown in an area of 3.86 m. ha accounting to the total production of 9.6 m. t. of paddy grain with an average yield of 3.11 tonnes ha^{-1} (Rice in India, 2006). By 2030, the world population is expected to reach 8.2 billion as against 6.5 billion in 2006 which would require approximately 533 m. t. of milled rice by 2030 as compared to 420 m. t. in 2006. There are still some 815 million people in the world suffering from hunger and malnutrition today, and most of them live in areas that depend upon rice production for food, income and employment.

Rice blast disease caused by *Pyricularia grisea* Sacc. is the most serious and damaging among the plant diseases affecting rice production in temperate and humid countries including India. Chemicals for this particular disease *viz.* carbendazim, edifenphos, iprobenfos, kasugamycin, isoprothiolane, ferimzone, metaminostrobin etc. have been developed. These chemicals mostly act on the growth of *P. grisea* by exhibiting specific fungicidal effects on the pathogen. The wide use of these site-specific chemicals sometimes causes development of fungicide resistance in the pathogens. In fact, resistance against kasugamycin and organophosphorus thiolate fungicides was

recognized in the field, where they were intensively used. Solutions to the problem were contrived for the effective use of chemicals at hand, such as application of mixtures with different action mechanisms or their use in rotation. All modern fungicides are developed through extensive safety evaluation while there is a deep public concern about their side effects on non-target organisms and the environment. Thus, there is a great deal of interest in those chemicals that are supposedly specific to target organisms and are less likely to lead to resistance problems. Some of such chemicals currently in the market include melanin biosynthesis inhibitors (MBIs) *viz.* tricyclazole, pyroquilon, carpropamid, fenoxanil, fthalide and diclocymet. In addition, there is an other group of chemicals called priming effectors or plant defense activators such as probenazole, acibenzolar-S-methyl (BTH) and tiadinil which induce host resistanee against the pathogen's attack (Yamaguchi, 2004).

Resistance can be defined as the ability of an organism to tolerate higher doses of chemicals. Such ability might be stable and inheritable adjustment by the fungus to a fungicide, resulting in less than normal sensitivity to the fungicide (Delp and Dekker, 1985). Resistance arises through the survival and multiplication of initially rare mutants in the pathogen population, during exposure to applied fungicides which at the same time kill or inhibit growth of the initially predominant sensitive forms. Several reports and excellent reviews have been published on the phenomenon of fungi developing resistance to fungicides throughout the world (Ashida, 1965; Dekker, 1976; Georgopoulos, 1977; Staub and Sozzi, 1984; Staub, 1991; Thind, 1993; Brent, 1995; Russell, 1995; Gangawane, 1997; Brent and Hollomon, 1998; Sharma *et al.*, 1998; Russell, 1999 and Brent and Hollomon, 2000).

Besides understanding the nature of fungicide resistance, knowledge of cross resistance is also important. Resistance to a fungicide is frequently associated with resistance to other fungicides with the same mode of action. Resistance management is vital to prolong the active life of the high risk fungicides, without which the number and effectiveness of the fungicides available to the farmers would be seriously reduced and the fungicides could become ineffective before they have generated sufficient revenue to the manufacturer to support the research and development programmes necessary to evolve new fungicides for crop protection.

Varietal resistance, the most desirable control measure, has been investigated by researchers in several countries. Unavailability of donor parents with stable levels of resistance has been the main factor for the limited progress in the varietal improvement programme (Reddy and Reddy, 1986). The existing levels of resistance can be further enhanced by inducing systemic resistance. Induced resistance is a state of enhanced defensive capacity developed by a plant when appropriately stimulated. Induced resistance is not the creation of resistance where there is none, but the activation of latent resistance mechanisms. Induced resistance primes the plant for resistance against further attacks by a broad spectrum of microorganisms. Once established, systemic acquired resistance lasts for weeks, perhaps throughout that generation and it will be an additional tool for effective disease management (Kessmann *et al.*, 1994)

Rice blast management at present represents a paradoxical situation where chemical control has been the most predominant method of disease control despite its potential toxic effects on humans, wild life and the environment if persist for longer period. But, the application of fungicides for blast control is inevitable; otherwise the food security of any rice growing nation will be at risk. However, a cautious effort should

be made to derive maximum mileage from other methods of control such as host plant resistance, induced systemic acquired resistance etc. besides effective fungicide resistance management strategies. With this back ground, an investigation was carried out on rice blast disease management under the title **"Studies on blast disease management in rice (*Oryza sativa* L.) incited by *Pyricularia grisea* Sacc** with the following objectives-

1. To study the relative resistance in the available rice cultivars against *Pyricularia grisea* Sacc.
2. To test the fungitoxic effects of systemic acquired resistance inducing chemicals on the pathogen.
3. Induction of systemic acquired resistance in rice against *Pyricularia grisea* using chemicals.
4. To study the efficacy of new fungicides on rice blast fungus.
5. Testing the isolates for the development of resistance to the most commonly used fungicides.
6. Testing the cross-resistance of the resistant strains to other structurally similar fungicides.
7. Testing the persistence of acquired resistance of the resistant strains.
8. Testing different fungicidal combinations to prevent/delay resistance development in the fungus.

Review of Literature

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CHAPTER II

REVIEW OF LITERATURE

2.1 EVALUATION OF RICE CULTIVARS FOR RESISTANCE AGAINST BLAST DISEASE

Baboy *et al.* (1995) found some rice varieties with moderately stable resistance from cultivars of different origin screened to identify field resistance to *Pyricularia grisea* under conditions provoking blast infection. The results that showed the resistance or susceptibility in different varieties varied between sites during the same year or between years.

Saifulla and Manjunatha (1995) screened several genotypes for blast disease resistance in the field. Ten genotypes *viz.,* KHRS 24, 27, 30, 38, 46, 57, 62, 64, 65 and KHRS 66 were highly resistant, KHRS 47, 59, 67, 68, 69, 70 and KHRS 71 were moderately resistant and HR-12 was highly susceptible.

Sharma and Ghimire (1996) screened 292 rice genotypes for blast resistance using Janaki and Laxmi as resistant controls and Jumli Pokhara as the susceptible control. The cultivars Khumal 4, Milyang 55, Janaki, Sabitri, Philips, Kalyani, Tai Chung Senglutions and IR 46 showed resistance. Hybrid IR 36 x Chhomrong was immune, while hybrid Fuji x Chhomrong had medium resistance. The variety IR 3941-4-1 was also immune while CH 45 and Radha Krishna were highly susceptible.

Singh and Dodan (1996) screened 175 rice genotypes during 1993-94 for resistance against rice blast in Haryana. Among them, 19 genotypes were resistant to leaf blast and 10 were resistant to neck blast while the genotypes HKR 90-403 and KHR 91-405 were resistant to both leaf and neck blast.

Kumari *et al.* (1998) screened 381 rice genotypes for field tolerance to major diseases of rice and identified resistant sources. The results indicated the existence of a remarkably rich diversity in the cultivated and traditional rice varieties which offered a pool of resistant genes against rice blast.

Twenty day old seedlings of 51 elite rice lines were evaluated for their resistance to rice blast in a field experiment conducted in Malan, Himachal Pradesh, India during 1997-98. Elite rice lines HPR 1057, HPR 1171 and RP 2421 were highly resistant to blast while IR-64 exhibited delayed reaction to the disease, whereas Himalaya 799 and 2216 exhibited some degree of slow blasting resistance (Sharma and Kapoor, 2002).

Rao and Reddy (2004) evaluated ten new plant types (NPTs) of rice for their disease resistance along with IR-64 and Rasi as resistant controls and HR-12 as susceptible control. The NPTs were tested in the Uniform Blast Nursery (UBN) using 0-9 scale of Standard Evaluation System (SES, 1996) developed by IRRI. Among the NPTs, five cultivars were resistant, three were moderately resistant, while the remaining 2 were reported as susceptible.

Amanzadeh *et al.* (2004) reported that the rice cultivars Fujiminori, Onda and Hassan Saraii were semi susceptible to leaf blast in the nursery and Haraz is a resistant cultivar to blast disease in Iran.

Sengultuvel *et al.* (2006) reported that the cross IR 50 × Tetep recorded the lowest score for blast resistance (2.1) followed by the crosses ASD 16 × Tetep (2.3) and IR 50 × Moroberekan (2.5). Among the crosses, 4 were resistant, 9 were moderately resistant, 6 were susceptible and one was highly susceptible to *Pyricularia grisea* infection.

Tripathi (2006) screened 12 rice cultivars viz. Indrajal, Vandana, Gurmatia, Basmati, Govinda, Jaya, Kalinga, Dehula, IR-64, Ajan, Lochai and Newari. Higher degree of resistance was observed in the cultivars *viz.* Kalinga (14.6%), Vandana (16.2%), IR-64 (18.2%) and Govinda (19.1%) while Gurmatia (58.9%), Indrajal (57.2%) and Dehula (57.3%) were found susceptible.

2.2 INDUCTION OF SYSTEMIC ACQUIRED RESISTANCE WITH CHEMICALS

Resistance can be induced in susceptible plants by activating the signal transduction system. This method of making susceptible plants as resistant ones is knows as "Induced Systemic Resistance" (M'Piga *et al.*, 1997).

A variety of constitutive barriers (physical and chemical) present in plants prior to infection are collectively responsible for natural resistance of plants. When a physical, chemical or biological agent induces the production or accumulation of defense components in the host or activates defense mechanism, it may be regarded as "Induced or Acquired Resistance" (Purkayastha, 1998).

The two most clearly defined forms of induced resistance are Systemic Acquired Resistance (SAR) and Induced Systemic Resistance (ISR) which can be differentiated based on the nature of the elicitor and the regulatory pathways involved (Gary and Robert, 2004). The classic form of SAR can be triggered by exposing the plants to virulent, avirulent and non-pathogenic microbes or artificially with chemicals such as salicylic acid, 2,6-dichloro-isonicotinic acid (INA) or benzo (1,2,3) thiadiazole-7-carbothioic acid S-methyl ester (BTH). Depending on the plant and elicitor, a set period of time is required for the establishment of SAR that corresponds to the time required for the coordinated accumulation of pathogenesis-related proteins and salicylic acid throughout the plant (Cameron *et al.*, 1994; Uknes *et al.*, 1992; Ward *et al.*, 1991). ISR is

potentiated by plant growth-promoting rhizo-bacteria (PGPR), of which the best characterized are strains within several species of *Pseudomonas* that cause no visible damage to the plant's root system. Unlike SAR, ISR does not involve the accumulation of pathogenesis-related proteins or salicylic acid, but instead, relies on pathways regulated by jasmonate and ethylene (Knoester *et al.*, 1999; Pieterse *et al.*, 1998; Yan *et al.*, 2002).

Hirooka and Umetani (2004) studied the effect of Tiadinil, a systemic fungicide for the control of rice blast. They considered it as a plant activator that induces plant resistance against pathogens.

Nishioka *et al.* (2005) reported that a pyrazole derivative, 3-chloro-1-methyl-1H-pyrazole-5-carboxylic acid (CMPA), exhibited high anti rice blast activity with out any significant antimicrobial activity up to 1 mg/ml. The treatment of CMPA induced the expression of PBZ1, a defense related gene which is evoked by several plant activators. This ability to induce PBZ1 expression and enhance disease resistance with out anti-microbial activity suggest that CMPA can activate acquired resistance in rice.

Adrina *et al.* (2006) studied the induction of SAR mediated by the chemical activator DL-3-aminobutyric acid (BABA) and the fungicide fosetyl-aluminium in potato cultivars with different levels of resistance against *Phytophthora infestans* (Mont.) de Bary 1876. They have demonstrated the enhancement of the defence response in the post harvest tuber samples by measuring the protein content of several hydrolytic enzymes as well as the phenol and phytoalexin content. Thus, they opined that the protective effect persist through out the whole crop cycle which may offer the possibility of controlling both foliage and tuber blight and could have a major impact in reducing over-winter survival of *P. infestans* in the tubers.

2.2.1 Salicylic acid

Injection of cucumber cotyledons with polyacrylic acid (PA), acetylsalicylic acid (ASA) and salicylic acid induced resistance to *Colletotrichum lagenarium* (Pass.) Ellis & Halst. 1893. Resistance was observed 96 hrs after treatment, but, not by 24 hrs (Mills and Wood, 1984).

Salicylic acid induced systemic resistance in sickle pod (*Cassia obtusifolia*) against *Alternaria cassiae* Jurair & A. Khan 1960. Full expression of resistance occurred within 24 hrs of induction, but persisted for only nine days (Weete, 1992).

Conti *et al.* (1996) suggested that the application of salicylic acid before incubation with *Sphaerotheca fuliginea* (Schltdl.:Fr.) Pollacci. reduced the intensity of the infection process and that salicylic acid was involved in the expression of systemic resistance in cucumber to *S. fuliginea.*

The effect of SA on systemic resistance of cacao against *Phytophthora palmivora* (E. J. Butler) E. J. Butler 1919 was investigated using 2 clones, ICSI and TMC 67. The number of lesions per leaf were significantly reduced in SA treated plants compared with those in the control. Seed treatment, foliar spray and soil drench induced systemic resistance in cacao (Okey *et al.*, 1997).

Exogenous application of salicylic acid solutions to pea leaves induced systemic resistance to *Erysiphe pisi* DC. 1805 and fungal germlings were reduced by 20-30 per cent that successfully infected untreated leaves of SA treated plants. The induced resistance persisted for atleast 13 days after treatment, but excision of treated leaves one day after SA application prevented full induction of systemic resistance and the resistance was not expressed, if treated leaves were inoculated less than 3 days after SA application. The effect of SA was transmitted to leaves at nodes both above and below treated leaves (Frey and Carver, 1998).

Quintanilla and Brishammar (1998) treated the potato plants with salicylic acid to induce resistance against late blight caused by *Phytophthora infestans* under green house conditions. Their results showed that the higher salicylic acid concentrations increased resistance in genetically resistant cultivars while the lower SA concentration increased susceptibility.

Exogenous applied SA does not induce systemic resistance to cucumber root rot caused by *Pythium aphanidermatum* (Edson) Fitzp. 1923, but endogenous SA, accumulated by *Pseudomonas* treatment in cucumber roots may be involved in induced systemic resistance (Chen-Chunquan *et al.*, 1999).

2.2.2 Benzothiadiazole (BTH)

A novel synthetic chemical, Benzo (1, 2, 3)-thiodiazole-7-carbothioic acid S-methyl ester (BTH) was shown to induce acquired resistance in wheat. BTH (0.3 mM) protected wheat systemically against powdery mildew infection by affecting multiple steps in the life cycle of the pathogen, *Erysiphe graminis* (DC.) Speer 1975 (Gorlach *et al.*, 1996).

Benzothiadiazole-S-methyl ester (BTH) is the new generation compound of plant protection products which activates the plants own defense mechanisms leading to an increased plant resistance against diseases. It was introduced in 1996 as a 'plant activator' for the control of wheat powdery mildew in Germany and Switzerland (Ruess *et al.*, 1996).

Jorgensen *et al.* (1997) suggested that dipping germinating seeds in BTH gave significant reduction of barley mildews caused by *Erysiphe graminis* compared with untreated seeds. Spraying BTH on green plants gave the best effect on leaves just above the treated leaf compared with leaves at higher levels.

Benhamou and Belanger (1998) concluded that exogenous application of a non-toxic, synthetic chemical, Benzo (1, 2, 3), ethiodiazole-7-carbothioic acid S-methyl ester (BTH, CGA 245704) sensitize susceptible cucumber plants to react more rapidly and more efficiently to *Pythium ultimum* Trow 1901 attack on cucumber, mainly through the massive accumulation of phenolic compounds at sites of attempted pathogen penetration.

Colson (1998) reported that treatment of cotton plants with BTH or SA reduced the percentage of leaf area covered with Alternaria lesions (2-15 fold), reduced the leaf area blighted (2-22 fold) and reduced the defoliation.

Delon *et al.* (1998) proved that one foliar application of CGA 245704 (Benzothiodiazole) at 1.6 g a.i./ha every 14 days partially protected tobacco plants against blue mold (*Peronospora tabacina* D. B. Adam 1933).

The effect of seed treatment with a chemical activator of systemic acquired resistance, CGA 245704 against two Brassica diseases with contrasting infection biologies the air borne downey mildew pathogen, *Peronospora parasitica* (Pers.:Fr.) Fr. 1949 and the soil borne fungus, *Rhizoctonia solani* J.G. Khun. 1958 were tested. Seeds of rape and cabbage were either imbibed with various concentrations of CGA 245704 in suspension or imbibed and then dried. There was a significant effect on the sporulation of *P. parasitica* for all concentrations of the compound used in both treatments. Some control of damping off caused by *Rhizoctonia solani* was provided by the imbibition treatment, but the degree of control was highly dependent on the concentration applied to the seed. It is concluded that seed treatment with CGA 245704 may simultaneously control several seedling diseases and therefore, provide a novel strategy for the management of these Brassica diseases (Jensen *et al.*, 1998).

Laun (1998) observed that treatments with Bion (30 and 60 g/ha in 400 litres of water) resulted in crop damage in cucumber and melons. Studies were also carried out during 1995-97 with Bion (30-60 g/ha, 2-3 times) in radish against *Peronospora destructor* (Berk.) Casp. 1860 and *Albugo candida* (Pers.:Fr.) Kuntze 1891 and got good control of these diseases.

Tomato plants infected by *Fusarium oxysporum* f.sp. *radicis-lycopersici* were treated with plant activator Bion (a product of CIBA GEIGY) at 0.01-1%. Treated plants became less infected with the disease than untreated ones, indicating the importance of systemically acquired resistance in plant protection not only in cereals but also in horticultural crops (Takacs and Dolej, 1998).

Csosz *et al.* (1999) carried out field trials in which Bion 50 WG was applied to approximately 50 varieties of wheat to investigate its effect on *Erysiphe graminis* f.sp. *tritici*. Bion induced greater resistance than triadimefon (Bayleton 25 WP).

Dann *et al.* (1998) observed the induction of resistance in soybean plants against white mould disease in which seeds were treated with 2, 6-dichloro isonicotinic acid (INA) and benzothiadiazole (BTH).

Godard *et al.* (1999) reported that the crucifer seedlings were sprayed with a water solution containing 0.05 mg/ml of elicitor CGA 245704 and challenged 4 days later with 14 different isolates of *Peronospora parasitica*. Results showed that resistance to the disease was induced against all the isolates. These results also showed that no resistance was induced when the inoculation was done just after BTH treatment. The seedlings as well as the young plants exhibited induced resistance when challenged 1-30 days after treatment.

On potted plants, acibenzolar-S-methyl (BTH) showed control on anthracnose and scab of cucumber and rust of Japanese pear but not on *Fusarium* wilt of

cucumber. In field trials, the occurrence of both rust and scab on Japanese pear was suppressed with this compound. These studies suggested that acibenzolar-S-methyl induced resistance to some but not to all diseases on cucumber and Japanese pear (Ishii *et al.*, 1999).

Narusaka *et al.* (1999) worked on systemic acquired resistance against cucumber scab caused by *Cladosporium cucumerinum* Ellis & Arthur 1889 by using Acibenzolar-S-methyl and salicylic acid and they concluded that a protective signal(s) can be transferred from leaves treated with acibenzolar-S-methyl to the untreated upper and lower leaves when systemic resistance was elicited. In contrast, exogenously applied SA is not likely to function as a mobile systemic resistance-inducing signal, because SA induces localized acquired resistance.

Schweizer *et al.* (1999) reported that rice seedlings treated with the synthetic compound, benzo (1, 2, 3) thiadiazole-7-carbothioic acid-S-methyl ester (BTH) acquired resistance to subsequent attack by the rice blast fungus *Pyricularia grisea.*

Ge-XiuChun *et al.* (1999) reported that the blast disease severity of the upper untreated leaves markedly reduced after the second leaf was treated with 0.5 mM/litre at 7 days before inoculation with *Pyricularia grisea.* They also reported that treatment of rice seeds with BTH enhanced SAR to *Pyricularia grisea* at the 3 to 4 leaf stage of rice seedlings.

A foliar application of BTH at tillering induced systemic resistance in wheat to powdery mildew as well as to speckled leaf blotch (Septoria tritici Roberge 1842). BTH treatment at the end of tillering provided better protection than when applied at mid tillering stage (Stadnik and Buchenauer, 1999).

Tosi *et al.* (1999) reported that a novel systemic chemical CGA 245704 (BTH) applied as a soil drench and foliar spray was shown to provide good protection against *Plasmopara helianthi* in sunflower plants. Soil drenches at the rate of 150 and 200 mg/kg soil (3000 µg/ml and 4000 µg/ml), were applied 3 days before the inoculation and induced 80-82% protection against downy mildew. Even though BTH offered 90 per cent protection against the disease at 300 mg/kg soil (600 µg/ml), the plants exhibited phytotoxicity symptoms at this concentration.

Cotyledonary treatment with wettable granule (WG) formulation supplying 35 µg/ml of Benzo (1, 2, 3)-thiodiazole-7-carbothioic acid S-methyl ester (BTH) significantly reduced the formation of lesions on the subsequent two leaves when challenged with *Alternaria macrospora* Zimm. 1904 (Colson and Deverall, 2000).

BTH treatment (100 µg a.i./ml) caused a significant reduction in the number of lesions following challenge inoculation with *Mycosphaerella pinodes* (Berk. & Bloxam) Vestergr. 1912 compared with control (Dann and Deverall, 2000).

Acibenzolar-S-methyl (BION) at low concent *Ustilago maydis* (DC.) Corda 1842rations activates resistance in many crops against a broad spectrum of diseases including fungi, bacteria and viruses. In monocots, activated resistance by Bion typically was very long lasting, while it was less pronounced in dicots. Bion was translocated systemically in plants and can take the place of salicylic acid in the natural SAR signal pathway inducing the same spectrum of resistance and the same set of molecular markers (Oostendrop *et al.*, 2001).

Jansen *et al.* (2006) reported that treatment of rice seedlings with BTH was unable to prevent colonization of roots by *P. grisea* although a reduction in disease levels was observed. Moreover, BTH was not able to protect barley roots against infection with *P. grisea*. Taken together, the results suggested that although roots show

intrinsic variation in their ability to resist colonization by *P. grisea*, neither gene-for-gene incompatibility nor acquired resistance are as effective as blocking the pathogen as they are in leaves.

2.3 NON-CONVENTIONAL CHEMICALS

Bhattacharyya and Roy (1998) tested 21 non-conventional chemicals each at two concentrations in wet seed treatment, root-dip treatment and spray treatment against sheath blight of rice (*Rhizoctonia solani)* and concluded that sodium selenite at 10^{-5} M, zinc sulphate at 10^{-4}, lithium sulphate at 10^{-4} M, calcium nitrate at 10^{-2} M, sodium fluoride at 10^{-3} M and ferric chloride at 10^{-4} M could check the disease in excised rice leaves as well as in standing plants. When tested by poison food technique, these chemicals showed no *in vitro* fungitoxicity at their effective concentrations. Highest efficacy was recorded with the combination of all three methods used stage by stage.

Manandhar *et al.* (1998) reported that ferric chloride, dipotassium hydrogen phosphate and salicylic acid significantly reduced rice blast disease severity when applied as either soil drench or foliar spray but not as seed treatment. Neck blast incidence was significantly reduced by all 3 chemicals when applied as foliar spray at least 2 times from the seedling to the heading stage, while greatest reduction was obtained with ferric chloride.

Chowdhury (2000) studied the effect of four different non-conventional chemicals of diverse nature at 10^{-3} to 10^{-6} M concentration in controlling fusarium wilt of chickpea. Of these, IAA, cycloheximide and cycocel showed very strong protective effect at lower concentrations compared to higher concentrations. These chemicals reduced wilt symptoms by 45 to 57 per cent, preventing mortality appreciably and also limited vascular colonization by *Fusarium oxysporum* f.sp. *ciceri* in the susceptible chickpea cultivar, ICC-4951.

Senapaty *et al.* (2000) tested 21 non-conventional chemicals against *P. grisea in vitro* by poison food technique and concluded that cadmium chloride (10^{-4} M), ferric chloride (10^{-4} M), lithium sulphate (10^{-3} M), magnesium sulphate (10^{-3} M), potassium chloride (10^{-3} M), sodium fluoride (10^{-3} M), sodium molybdate (10^{-4} M), sodium silicate (10^{-4} M) and zinc sulphate (10^{-4} M) could effectively check the disease. These chemicals showed no *in vitro* fungi toxicity at their effective concentrations.

Kamalakannan *et al.* (2001) tested the effect of some heavy metal salts *viz.*, barium chloride, cadmium chloride, ferric chloride, mercuric chloride and nickel nitrate at 10^{-3} and 10^{-5} M on mycelial growth, biomass production and spore germination of *Pyricularia grisea in vitro*. Mycelial growth, biomass production and spore germination were lowest with nickel nitrate at both the concentrations. Foliar spray and seed treatment at 10^{-3} m concentration of all the heavy metal salts provided moderate to substantial protection to rice seedlings at different stages.

Mills and Wood (1984) reported that the liquid shake cultures incorporated with polyacrylic acid (PA), Acetyl salicylic acid (ASA) and salicylic acid did not significantly affect the growth of the pathogen *Colletotrichum lagenarium.*

Brock *et al.* (1994) worked with 2, 6-dichloro isonicotinic acid against *Alternaria macrospora in vitro*. They concluded that there is no direct effect of 2, 6-dichloro isonicotinic acid on the fungus *Alternaria macrospora.*

Coquoz *et al.* (1995) reported that SA inhibited the release of zoospores of the fungus *Phytophthora infestans in vitro* at 2 mM whereas the germination of sporangia was not significantly altered in the concentration range of 0 to 2 mM. Mycelial growth of *Phytophthora infestans* was not affected *in vitro* when the culture medium was amended with SA.

Dann *et al.* (1998) reported that 2,6-dichloro isonicotonic acid (INA) and benzothiodiazole (BTH) did not result in observable pathotoxicity *in vitro* or inhibit the growth of *Sclerotinia sclerotiorum* (Lib.) de Bary 1884.

SA did not inhibit mycelial growth of *Pythium aphanidermatum* at 100-200 µg/ml *in vitro*, but higher levels inhibited mycelial growth. Zoospore germination increased at concentration of 100-500 µg/ml, but decreased at 1000 µg/ml compared to lower concentrations (Chen-Chunquan *et al.*, 1999).

Ishii *et al.* (1999) reported that there is no remarkable activity of BTH on mycelial growth and conidia germination of many fungi tested like *Cladosporium cucumerinum, Colletotrichum lagenarium, Fusarium oxysporum* f.sp. *cucumerinum, Corynespora cassicola, Alternaria alternata* (Fr.:Fr.) Keissl. 1912 and *Botrytis cinerea* Pers.:Fr 1794.

The CGA 245704 treatment did not inhibit zoosporangial germination of *Plasmopara helianthi in vitro* at 50, 100, 200 and µg/kg soil (Tosi *et al.*, 1999).

2.4 CHEMICAL CONTROL

Kapooria and Hairwadzi (1982) studied the effect of the systemic fungicide carbendazim *in vitro* on *Pyricularia grisea* at different concentrations *viz.*, 100, 200, 400, 600, 800 and 1000 µg/ml. They concluded that inhibition of growth started at 800 µg/ml when the fungicide was added to the solidified medium and at 400 µg/ml when it was incorporated in to the molten medium.

Mbodj *et al.* (1987) reported that tricyclazole reduced infection by *Pyricularia grisea* using resistant cultivars. Tricyclazole at 300 and 600 a.i./ha gave the same or better control than tricyclazole at 150 g a.i./ha or edifenphos at 600 g a.i./ha.

Moletti *et al.* (1988) concluded that pyroquilon (1.5 kg/ha) applied twice gave good results against leaf blast followed by tricyclazole at 0.75 kg/ha used twice. But, tricyclazole gave good control of neck blast followed by pyroquilon.

Huang and Yu (1989) concluded that unit disease incidence (1%) resulted in 21-229 kg/ha yield loss of rice with average percentage yield loss of 0.89%. They also said that yield loss was reduced to 0.37 per cent with the best fungicide treatment (0.35% Shinmonate D).

Naidu and Reddy (1989) reported that carbendazim, edifenphos, pyroquilon, isoprothiolane, iprobenfos and thiophanate-methyl reduced leaf blast but not neck blast on a susceptible rice cultivar IR-50 when applied as foliar sprays at tillering, panicle initiation and flowering stages.

Okhovot (1989) evaluated four fungicides namely tricyclazole, benomyl, edifenphos and thiabendazole sprayed first at the panicle stage and then 15 days later at a concentration of 0.2%. They concluded that tricyclazole was the most effective in reducing neck blast though the differences between grain yields were not statistically significant.

Awoderu (1990) reported the yield loss upto 58.8% which might be attributed to the neck blast caused by *Pyricularia grisea* in Cote 9' Ivore.

El Kazzaz *et al.* (1990) reported that Hinosan (edifenphos) and Kitazin (iprobenfos) inhibited growth of *Pyricularia grisea in vitro* at 75 and 100 ppm, respectively, while the minimum inhibitory concentration of tricyclazole and pyroquilon were 500 and 700 ppm, respectively. All test fungicides were effective against blast disease, pyroquilon being the best followed by tricyclazole, edifenphos and iprobenfos. The best control was given by treating the plots once in the nursery and 2 or 3 times after transplanting.

Nikolaev *et al.* (1990) suggested that the fungicide tricyclazole inhibited melanin biosynthesis in *Pyricularia grisea* and induced fungitoxic activated oxygen in rice leaves.

Nikolaev and Aver Yanov (1990) reported that tricyclazole added to a spore suspension of *P. grisea* completely inhibited repigmentation. It is suggested that the fungicidal activity of tricyclazole (and probably other melanin-inhibiting fungicides) may be associated with the inhibition of repigmentation of germinated spores.

Peterson *et al.* (1990) opined that tricyclazole, a systemic fungicide with selective activity against *P. grisea* interferes with appressorial function by inhibiting melanin biosynthesis and host penetration. They also suggested that tricyclazole was effective as a preventive treatment for the control of rice blast which can be applied by a variety of methods at any growth stage.

Sader *et al.* (1990) studied the effect of seed treatment with quintozene, benomyl, carbendazim, thiram, captafol and captan on 100 seed weight, seed germination and vigour in healthy and *Pyricularia* infected seeds of rice varieties IAC 164 and IAC 165. They found that 100 seed weight was more in healthy compared with infected seeds. No effect of the fungicides was observed on seed germination except quintozene which exhibited some phytotoxic effect on seed germination. No significant effect of fungicides was found on seedling vigour and dry weights of above ground parts of seedlings.

Goulart (1991) reported that captan, totylfluanid, ethyltrianol, iprodione and thiram reduced the incidence of *P. grisea* in naturally and artificially contaminated rice seeds of cultivar IAC-47, but ethyltrianol and iprodione + thiram gave the best control and highest yield increase. Captan was least effective and none of the treatments improved germination.

Awoderu and Bangura (1992) obtained significant yield increase with benomyl (60.1 %), copper oxychloride (75.2 %) and PP 389 (117.4 %) high yielding on susceptible rice cultivar C22.

Ishiguro *et al.* (1992) reported that application of isoprothiolane, edifenphos, iprobenfos, kasugamycin, blasticidin-S or ferimzone to rice plants after the appearance of rice blast lesions inhibited lesion enlargement and spore production. A linear relationship was found between days after application of each fungicide and the area of a lesion during a particular period.

Iprobenfos, copper oxychloride and edifenphos were the most inhibitory to spore germination of *Pyricularia grisea.* Germ tube elongation was drastically reduced by edifenphos and IBP. Edifenphos was the best fungicide in the field followed by IBP, pyroquilon and kasugamycin both in controlling leaf and neck blast and in increasing grain yield (Mustaq Ahmad and Ahmad, 1992).

Saifulla and Seshadri (1992) suggested that seed treatment with pyroquilon and tricyclazole (both 4 g/kg seed) was effective in controlling *Pyricularia grisea* in rice nurseries.

Sharma and Kumar (1992) reported that soil treatment with 3.75 kg a.i./ha of iprobenfos granules controlled infection of rice by *Pyricularia grisea* in Nagaland.

Viswanathan and Narayanasamy (1992) reported that both tricyclazole and mancozeb at 500 ppm effectively inhibited spore germination, germ tube elongation, mycelial growth and sporulation of the rice blast fungus *P. grisea in vitro.*

Paul *et al.* (1993) tested seven different fungicides namely iprobenfos, carbendazim, metiram, edifenphos, tricyclazole, carbendazim + metiram and pyroquilon against rice blast on the early maturing cultivar Rustic. They said that of the fungicides tested, tricyclazole gave more effective control than iprobenfos the fungicide mostly used then.

Prabhu and Filippi (1993) studied the effect of seed treatment with pyroquilon on rice leaf blast. In the field, pyroquilon (4 g/kg seed) treatment kept leaf blast severities below 5 per cent at 38 days after sowing in susceptible cultivars IAC 47, IAC 25, IAC 164 and L-50 in the blast nursery. Seed treatment with pyroquilon (4 g/kg seed) significantly reduced leaf blast at 38 days after sowing in cultivars IAC 25, IRAT 112 and IAC 47. They also suggested that under low disease pressure pyroquilon at the rate of 2 g/kg seed significantly reduced leaf blast severity. But, at high disease pressure, higher rates of application (4 g/kg seed) were necessary for maximum disease control in a susceptible cultivar. Seed treatment has little or no effect in resistant cultivars.

Rodriguez *et al.* (1993) evaluated the effectiveness of seed treatment with carboxin (2.5 g/kg seed), benomyl (2.5 g/kg seed), edifenphos (2 ml/kg seed), kasugamycin (2.0 ml/kg seed) and tricyclazole (2.0 g/kg seed) to control leaf blast. The results showed that leaf blast was reduced by at least 50% and plant height increased by 27% when compared with the control. There was no difference between dry seed treatment and wet (soaked) seed treatment.

Variar *et al.* (1993) tested tricyclazole (Beam 75WP), IBP (Kitazin 17G) and edifenphos (Hinosan 50EC) on susceptible rice varieties IR 50 and HR-12. They concluded that application of IBP granules @ 25 kg/ha at 40 days after sowing supplemented with one spray of edifenphos @ 1 l/ha at booting stage recorded significantly low disease score and maximum grain yield. Seed treatment with tricyclazole @ 4 g/kg seed supplemented with IBP @ 25 kg/ha at 40 days after sowing proved equally effective.

Ram Singh *et al.* (1994) evaluated 7 fungicides against *Pyricularia grisea* on the susceptible rice cultivar Taraori Basmati and found that tricyclazole and propiconazole were the most effective in reducing neck blast and increasing grain yield.

Saifulla (1994) tested tricyclazole, blasticidin, carbendazim, cyproconazole, chlorothalonil, bitertenol, propiconazole and hexaconazole for the control of *Pyricularia grisca* in the field using the susceptible variety Mandya Vijaya and concluded that the best disease control and yield were achieved with tricyclazole and blasticidin.

The differential effect of seven fungicides on growth against three isolates of *P. grisea* was evaluated *in vitro* by food poisoned technique at 1, 5, 10, 25, 50 and 100 µg/ml for edifenphos, IBP, pyroquilon, copper oxychloride and mancozeb; 0.1, 0.25 and 0.50 µg/ml for carbendazim and 1.0 and 5.0 µg/ml for thiophanate methyl (Arun Kumar and Singh, 1995). Rice and fingermillet isolates of *P. grisea* exhibited maximum sensitivity to carbendazim followed by thiophanate methyl, edifenphos, IBP, mancozeb and copper oxychloride while pyroquilon being least effective *in vitro*.

Shirane *et al.* (1995) reported that SSF-126 (E)-2-methoxyimino-N-methyl-2-(2-phenoxyphenyl) acetamide inhibited mycelial growth (IC_{50} 7.7 µM) of *P. grisea*. The inhibited mycelium, which was incubated for 30 h in the presence of SSF-126 grew vigorously after transfer to a fungicide free medium indicating that SSF-126 is a fungi static compound.

Enyinnia (1996) reported that one application of tricyclazole at 400 g a.i./ha at maximum tillering stage of rice successfully suppressed blast development and resulted in a significantly higher grain yield than untreated control plots by an average of 42.17 per cent, whereas benomyl at 400 g a.i./ha produced 18.94 per cent more rice grain than the control.

Kurahashi *et al.* (1996) found that the mode of action of KTU 3616 differs from other derivatives of cyclopropane carboxylic acid as it inhibits melanin biosynthesis by blocking scytalone and vermelone dehydration and thereby prevents *Pyricularia grisea* from penetrating rice epidermal cells.

Viswanathan *et al.* (1996) reported a maximum detectable level of 0.5 ppm of tricyclazole in grain and straw which was well below the maximum residue limit when the rice cultivar IR 50 was sprayed with tricyclazole at 800 ppm 35 days before harvesting during the late boot leaf stage and justified the large scale use of this fungicide for rice blast control.

Dubey (1997) reported that three sprays of tricyclazole (0.015%) in combination with mancozeb (0.125%) was most effective in minimizing disease intensity (6%), node (3%) and neck (2%) infections and maximizing grain yield (30.1 q/ha). Mancozeb (0.12%) in combination either with thiophanate methyl (0.025%) or carbendazim (0.025%) was also proved effective while tricyclazole (0.03%), carbendazim (0.5%) and hexaconazole (0.1%) alone were ranked next in order of superiority, respectively.

Seed treatment with systemic fungicide pyroquilon at the rate of 4.0 g a.i./kg seed suppressed rice blast severity by an average of 26.8% and significantly increased the average yield when compared with untreated control (Filippi and Prabhu, 1997). They also concluded that both leaf and panicle blast significantly accounted for variation in grain yield.

Condole *et al.* (1999) studied the effect of panicle blast on the physical properties and milling quality of rice and concluded that the occurrence of unfilled, chalky and fissured kernels in diseased samples were higher by 30, 21 and 7 per cent, respectively than in blast-free samples. Blast reduced kernel bulk density by 140 kg/m^3 and head rice yield by 12 per cent. The moisture content of rough rice in blast infected panicles was lower than the average moisture content of rough rice from blast free panicles.

Ram Singh *et al.* (1999) evaluated 219 rice cultivars for blast resistance in a screening nursery and identified 13 genotypes *viz.*, HKR 90-403, HKR 90-404, HKR 90-413, HKR 90-421, HKR 91-406, HKR 95-435, HKR 95-458, HKR 95-505, HKR 95-511, CRMS 30, RP 3228-44-12-9-7, RP 3228-96-14-9-5 and UPR 1071-21-1-1 that were consistently resistant to the rice blast disease during 1995, 1996 and 1997.

Dubey (2000) evaluated twelve fungicidal spray schedules against rice blast. The spray schedule tricyclazole (0.03%) – Mancozeb (0.2%)-tricyclazole (0.03%) proved most effective with minimum neck and node infection and disease intensity resulting in highest grain yield. The next best spray schedule was thiophanate methyl (0.05%)-mancozeb (0.2%)-thiophane methyl (0.05%), followed by thiophanate methyl (0.05%)-mancozeb (0.2%)-mancozeb (0.2%). All spray schedules were economical while thiophanate methyl-mancozeb-thiophanate methyl gave the highest per rupee return of Rs. 11.60.

Kim *et al.* (2000) evaluated three known melanin biosynthesis inhibitors namely KC 10017, tricyclazole and carpropamid against *P. grisea in vitro* and concluded that all three MBI fungicides did not show any inhibitory effect on fungal growth. Carpropamid (KTU 3616), an anti-blast fungicide exhibited a little effect on *in vitro* mycelial growth and no effect on spore germination and appressoria formation (Rashmi and Singh, 1999).

Tirmali and Patil (2000) compared five new fungicide formulations *viz.*, propineb, carpropamid, fluquinconazole, prochloraz and epoxiconazole with tricyclazole. They reported that all the new formulations reduced neck blast incidence by 16.27 to 29.23 % while epoxiconazole (12.5SC) being highly effective (29.23 %), whereas, tricyclazole (75WP) showed superiority over all the new formulations in reducing neck blast incidence by 50.06% and increasing grain yield by 89.13% followed by epoxiconazole with an yield increase of 41.30% over the unsprayed control.

Yashoda *et al.* (2000) reported that seed treatment with carbendazim (2 g/kg seed) in combination with 3 sprays of tricyclazole at 0.06% or 3 sprays of pyroquilon at 0.1% resulted in lowest leaf blast incidence whereas, chlorothalonil has the lowest efficacy in controlling leaf blast. They also said that 3 sprays with carbendazim resulted in a higher reduction of blast than 2 sprays.

Becerra and Tosquicy (2001) reported that the best control of rice blast disease and the highest grain yield (4.43 t/ha) were obtained when azoxystrobin was applied at 0.6 l/ha, although good control of rice blast was also attained at 0.2 and 0.4 l/ha on the susceptible rice cultivar Milagro Filipino Depurodo in Mexico.

Hossain and Kulkarni (2001b) tested the efficacy of 6 systemic fungicides *viz.*, carbendazim, difenconazole, hexaconazole, iprobenfos, propiconazole and tricyclazole at 500 and 1000 ppm *in vitro*. They said that iprobenfos, propiconazole and carbendazim were most effective followed by hexaconazole and tricyclazole. They also found that mancozeb was the best non-systemic fungicide among chlorothalonil, copper-oxychloride, mancozeb and iprodione at 1000 and 3000 ppm in inhibiting the growth of *Pyricularia grisea in vitro*.

Hossain and Kulkarni (2001a) reported that seed treatment with carbendazim 2 g/kg seed followed by 2 sprays of tricyclazole (0.06%) was the best in managing the disease and getting higher yields. The next best treatment was seed treatment with carbendazim (2 g/kg) followed by 2 sprays of propiconazole (0.1%) which was superior to hexaconazole.

Koizumi *et al.* (2001) evaluated two systemic fungicides carpropamid and azoxystrobin as seed treatment against rice blast on partially resistant rice cultivar Doman Naka and concluded that carpropamid at 5 g a.i./kg seed was more effective in reducing blast development than azoxystrobin at 6 g a.i./kg seed.

Sharma and Kapoor (2002) reported that three sprays each of tricyclazole, carbendazim, prochloraz, hexaconazole, chlorothalonil and cyproconazole at the tillering, booting and panicle emergence stages were effective in controlling rice blast. However, tricyclazole was superior over rest of the fungicides followed by carbendazim.

Vijaya (2002) evaluated the efficacy of tricyclazole (0.1%), thiophanate-methyl (0.1%), chlorothalonil (0.25%), mancozeb (0.25%) and carbendazim (0.1%) against rice blast caused by *Pyricularia grisea* and concluded that the per cent disease incidence (PDI) was lowest with tricyclazole (17.6%) followed by thiophanate-methyl (19.3%). The highest yields were obtained with tricyclazole (5.7 t/ha) followed by thiophanate-methyl (5.6 t/ha).

Cortesi and Giuditta (2003) compared the efficacy of azoxystrobin and trifloxystrobin to tricyclazole and tricyclazole + propiconazole applied at the beginning of stem elongation and at late booting. They concluded that two applications of azoxystrobin (250 g/ha) and trifloxystrobin (125 g/ha) were more effective than tricyclazole (225 g/ha). However, the difference of efficacy between two treatments of strobilurins and one treatment of tricyclazole at 450 g/ha was not significant. They said that both strobilurins and tricyclazole were highly effective against leaf blast and neck blast and reduced incidence and disease severity by 90-100% and 75-90%, respectively. There was no significant improvement in efficacy of tricyclazole (225 g/ha) in combination with propiconazole (125 g/ha) when compared to tricyclazole (225 g/ha) alone.

Cortesi and Guiditta (2003) suggested two sprays at late booting and at heading with azoxystrobin or trifloxystrobin at 250 and 125 g./ha, respectively or one treatment with tricyclazole at 450 g/ha followed by one treatment with strobilurin when severe rice blast epidemics are predicted.

Prabhu *et al.* (2003) evaluated the response of four upland rice cultivars to foliar fungicide application in relation to panicle blast control and obtained differential disease control and yield response of cultivars. The losses in grain yield of cultivars IAC 202, Caiapo, Rio Paraniba and Araguaia due to panicle blast were 44.8, 27.4, 24.4 and 18.2 per cent, respectively.

Cardenas *et al.* (2004) reported that chitosan and its hydrolysate at 100 mg/litre or their combination with copper had no significant effect on P. grisea mycelial growth; however, they inhibited pathogen sporulation by 70.85 per cent. The few sporulation of chitosan was dispersed to the colony periphery, while in the control it was grouped in the center. These results indicate the potential of using chitosans at low concentrations for initial disease inoculum reduction in the field.

Prajapati *et al.* (2004) tested cyproconazole 50 SC (0.1%), tricyclazole 75 WP (0.045%), carbendazim 50 WG (0.05%), bitertenol 25 WP (0.025%) and propiconazole 25 EC (0.025%) against carbendazim 50 WP (0.05%) as standard control using a highly susceptible rice cultivar, Pankhali 203. They concluded that tricyclazole was significantly superior in decreasing the leaf blast and neck blast by 62.9 and 64.1 per cent, respectively with corresponding increase of 72.3 per cent in grain yield over untreated control and was at par with carbendazim 50 WG.

Dubey (2005) studied the efficacy of carpropamid (0.1%), tricyclazole (0.03%), thiophanate methyl (0.05%), carbendazim (0.05%), chlorothalonil (0.15%), validamycin (0.05%) and copper oxychloride (0.2%) against rice blast in the main field using the susceptible rice cultivar Birsa Dhan 202. He concluded that carpropamid was the most effective fungicide for blast management with the minimum neck (1.1%) and node infections (1.7%), disease severity (3.8%) and the maximum grain yield (4.54 t/ha) followed by tricyclazole and thiophanate methyl.

Kumbhar (2005) stated that epoxiconazole 12.5 SC (2ml/l) was the most effective fungicide against rice blast followed by prochloraz 50 WP (1g/l), propineb 70 WP (5g/l), chlorothalonil 40 EC (2ml/l) and chlorothalonil 75 WP (1g/l). However, the efficacy of all the fungicides was next only to tricyclazole 75 WP (0.6%) which suppressed the neck blast incidence to 37.88 per cent over the control. A maximum increase of 60.99 per cent in grain yield was achieved with tricyclazole 75 WP followed by epoxiconazole 12.5 SC which recorded an increase of 34.85 per cent over the control.

Ali Anwar and Bhat (2005) studied the efficacy of isoprothiolane 40 EC (1ml/kg seed), tricyclazole 75 WP (0.6 g/kg seed), edifenphos 50 EC (1 ml/kg seed), hexaconazole 5 EC (0.5 ml/kg seed) and mancozeb 75 WP (3 g/kg seed) as seed treatment against rice blast in the variety K-39. They stated that isoprothiolane 40 EC and tricyclazole 75 WP were superior as seed treatment with good amount of protection against leaf blast upto 35 days, while leaf blast symptoms were observed in edifenphos 50 EC treated plots at 30 days. Mancozeb 75 WP was the least effective of all the fungicides tested.

Zhang Chuan Qing *et al.* (2005) evaluated a method to study the potential risk of *Pyricularia grisea* resistance to tricyclazole. Both EC_{50} of hypal melanization (EC_{50}-H) and minimal inhibitory concentration of melanization in appressoria (MIC-A) by tricyclazole showed positive correlation to the EC_{50} of tricyclazole against blast disease tested *in vivo*, with relative co-efficiency (R_2) of 0.8995 and 0.8244, respectively. However, the stability and reproducibility of EC_{50}-H were better than those of MIC-A, suggesting that it could be used to detect the sensitivity of *P. grisea* to tricyclazole *in vitro*.

Groth (2006) applied azoxystrobin as foliar spray to the naturally infected field plots at the rates of 0.11, 0.17 and 0.22 kg a.i. per ha at boot (B) and heading (H) or only at H growth stages and at 0.17 kg a.i. per ha at 5 (H+5), 10 (H+10) and 15 (H+15) days after H and B with low or high blast pressure. Azoxystrobin application made at H,

H+5 and B+H significantly reduced blast incidence with high and low disease pressure resulting in significantly higher grain and head rice milling yields compared to unsprayed plots with high blast pressure. With fungicide application made at B, H+10 and H+15 days post heading, rice had higher disease incidence resulting in lower grain and milling yields compared with rice receiving a heading application.

2.5 FUNGICIDE RESISTANCE

Benzimidazole fungicides were introduced for plant disease control in 1960s and early 1970s. Several benzimidazole products are currently used as foliar fungicides, post-harvest application and seed treatment. Benzimidazoles have made a great impact on effective control of many diseases which were not controlled by non-systemic fungicides. Their high activity against many pathogenic fungi of the class ascomycetes, and certain basidiomycetes and deuteromycetes resulted in their wide spread use in disease control. This group of fungicides is particularly vulnerable, since several fungal species have developed resistance to these fungicides under different environmental conditions with in few years of their intensive use (Dekker, 1976; Georgopoulos, 1977, Greenaway and Whatley, 1975). The first instance of resistant development was reported in powdery mildew of cucurbits in greenhouses after one year of application (Schroeder and Prevvidenti, 1969).

Shabi and Ben-Yephet (1976) obtained MBC resistant isolates of *Venturia pyrina* Aderh. 1896 from a pear orchard which could tolerate up to 2000 µg/ml concentration of MBC as against 0.5 µg/ml tolerated by the wild type. They reported that the isolate that was tolerant to three benzimidazole fungicides (benomyl, MBC and thiophanate-methyl) was sensitive to captan, dodine and imazalil.

Horsten and Fehrmann (1980) reported that strains of *Septoria nodorum* (Berk.) Berk. 1845 weakly resistant to carbendazim grew better on media with 1-3 µg/ml of carbendazim than sensitive strains. They found that growth and spore production of highly resistant strains at 1000 µg/ml was similar to that of sensitive strains in fungicide

free media. Growth reduction was accompanied by increased spore production. In pathogenicity tests on wheat, symptom development and spore production were similar in both sensitive and highly resistant strains.

Reddy *et al.* (1980) reported the carbendazim resistance in *Gloeosporium ampelophagum* (Pass.) Sacc. 1878 for the first time in India and stated that an isolate of *G. ampelophagum* developed 4-fold (500-2000 µg/ml) resistance after training *in vitro*.

Delen (1980) reported that the pathogen, *Endothia parasitica* (Murrill) P.J. Anderson & H.W. Anderson 1912 causing chestnut blight of apple, acquired persistent resistance to benomyl and carbendazim after continuous training to these systemic fungicides *in vitro*.

Pan and Sen (1981) reported that four isolates (S_1, S_{321}, S_2 and S_{15} according to their virulence) of *Macrophomina phaseolina* (Tassi) Goid. 1947 acquired 50, 60, 30 and 30 fold resistance to carbendazim, respectively after training to increasing concentrations of the fungicide. Further transfers did not increase tolerance any more. It was reported that several generations were required to reach maximum adaptability and the culturing and morphological characters were also differed among isolates.

Tripathi and Schlosser (1982) demonstrated the mechanism of resistance of *Botrytis cinerea* to carbendazim and found that the uptake of MBC (carbendazim) was 44 per cent less in resistant strains than susceptible strains. They suggested that reduced uptake and lesser binding of MBC to tubulin-like protein contributed towards MBC resistance.

Of 23 isolates tested, nine isolates of *Septoria tritici* were found to be resistant to MBC. The minimum inhibitory concentration for sensitive isolates was 0.2 – 0.4 ppm and for resistant ones it was more than 1000 ppm. It was also reported that the resistant isolates were cross-resistant to thiobendazole and thiophanate methyl (Fisher and Griffen, 1984).

Brown *et al.* (1984) reported that the growth of sensitive isolates of *Pseudocercosporella herpotrichoides* (Fron) Deighton 1973 was completely inhibited at 0.5 μg/ml of carbendazim while the tolerant isolates grew on agar containing 1000 μg/ml. They found that the tolerant isolates were cross tolerant to benomyl, thiophanate-methyl and to a lesser degree to thiobendazole, but, not to prochlaraz. No difference in pathogenicity to wheat was observed between tolerant and sensitive isolates.

Mohiotto (1985) reported that carbendazim and benomyl tolerant isolates of *Cercosporidium personatum* (Berk. & M.A. Curtis) Deighton 1967 could grow well in oat meal agar containing 100 μg/ml while sensitive isolates were inhibited at 1 μg/ml.

Bateman *et al.* (1985) reported sensitivity of eye spot pathogen *Pseudocercosporella herpotrichoides* from wheat fields to carbendazim type fungicides. Tolerant isolates were detected by their growth on agar containing 1 mg/ml of carbendazim. Carbendazim tolerant isolates were sensitive to methyl N-(3,5-dichlorophenyl) carbamate.

Benzimidazole tolerance in *Cercospora beticola* Sacc. 1876 in India was first reported by Pal and Mukhopadhyay (1985). In preliminary tests with 58 monosporic isolates of the sugarbeet pathogen, five showed strong tolerance and four weaker tolerance to carbendazim. Only one tolerant isolate was obtained from carbendazim treated plots.

Ziemba and Gerska-Poczopko (1986) tested isolates of *Cercosporella herpotrichoides* Fron. 1912 from wheat with benomyl, carbendazim and thiophanate-methyl and tested on solid agar containing carbendazim at 0.07-5 μg/ml. No induced tolerance was observed to benzimidazoles by the above isolates.

Kumar and Thind (1992) reported for the first time resistance development under field conditions in *G. ampelophagum* to carbendazim in India. Among the seven

isolates studied, five appeared to be highly sensitive to carbendazim as there was complete inhibition of their colony growth at 0.1 µg/ml and were comparable with the reference sensitive strain. However, two isolates collected from vineyards in Ludhiana exhibited high degree of tolerance to this fungicide as the colonies could grow at concentrations upto 50 and 100 µg/ml respectively.

Testing of carbendazim resistance under *in vitro* conditions against 26 isolates of *G. ampelophagum* collected from different grape growing areas in Punjab state revealed the presence of sensitive isolates. However, seven isolates originating from orchards with known history of intensive carbendazim use, exhibited highly reduced sensitivity to carbendazim with the minimum inhibitory concentration values between 86–200 µg/ml (Thind *et al.*, 1994).

Uday Bhan and Chatrath (1994) developed resistant strains of chickpea isolate of *Botrytis cinerea* against carbendazim by training the parent strain on fungicide-amended medium for 12 generations. The resistance thus acquired persisted even after growing it on fungicide from medium for six generations. *In vivo* studies showed that resistant strain was more virulent than the wild strain when inoculated on a susceptible chickpea variety.

Mohan and Thind (1995) tested 52 isolates of *G. ampelophagum* from different vineyards in Punjab. The results revealed the presence of 13 isolates with highly reduced sensitivity to carbendazim. Resistance factor of these isolates varied from 363 to more than 900 µg/ml. Isolates were categorized into these morphological groups *viz.*, GA-I, GA-II, GA-III. Most of the resistant isolates were equally pathogenic and produced symptoms on leaves treated with carbendazim upto 1000 µg/ml.

Gupta and Sharma (1996) reported that *Venturia inequalis* (Crooke) G. Winter 1875 developed resistance to carbendazim. ED_{50} value of highly resistant isolate was 8.7 µg/ml whereas ED_{50} values of other isolates ranged from 0.2 to 3.3 µg/ml. Similarly, ED_{90} values were ranging from 67.4 to 564.9 µg/ml.

Sunder and Satyavir (1998) tested sensitivity of 28 isolates of *Fusarium moniliformae* J. Sheld. 1904 from bakanae infected plants of rice to carbendazim. Growth inhibition of isolates varied between 0 to 20% and 77 to 99.5% at its lower and higher concentrations.

Mingguo and Xiu (2001) reported that *Fusarium graminearum* Schwabe 1838 developed resistance to carbendazim. Carbendazim resistant isolates showed positive cross resistance to benomyl, thiabendazole and thiophanatemethyl.

Osada and Imouye (1980) observed diminishing control of rice blast disease by IBP when compared to other blasticides. The minimal inhibitory concentration (MIC) value of isolates causing a diminishing effect of IBP in the field *vivo* were above 40 ppm. Most isolates of the fungus from that locality also showed the resistance to IBP *in vitro* suggesting the occurrence of IBP-resistant strains in that locality.

Taga *et al.* (1982) obtained mutants resistant to IBP from wild-type strains of *P. grisea* isolated from finger millet and were grouped into two classes, highly resistant (HR) and moderately resistant (MR). HR strains grew at 0.2 mM IBP and showed cross resistance to isoprothiolane. In constrast, MR strains did not grow at 0.2 mM IBP and were sensitive to isoprothiolane. Ramdom ascospore progenies from crosses with HR strains segregated 1:1 for resistance : sensitivity, indicating that a single major gene controls IBP resistance in HR strains, but not MR strains.

Osada (1983a) observed that disease severity in submerged application plots of isoprothiolane and IBP were not significantly different from that in no application plots under the prevalence, of resistant strains to KSM and/or IBP. While submerged application of probenazole was found effective to both leaf and panicle blast under the same condition. Isolation frequency of IBP-resistant strains were increased gradually by submerged application of IPT and IBP indicating the decline in control effect of IPT and IBP on rice blast was caused by prevalence of IBP-resistant strains.

Osada (1983b) examined the effect of rice cultivars differing in field resistance to blast on population change in resistant strains to Kasugamycin and/or IBP by inoculating the mixture of each strains on the three cultivars Mokoto (ss), Sasamishi ki (s) and Toyonishiki (r) and observed no difference among the three rice cultivars with regard to isolation frequency of resistant strains to KSM and IBP.

Fukaya and Kobayashi (1983) developed a simple and efficient method to detect resistant strains of *Pyricularia grisea* to Kasugamycin. Conidiospores on lesions were painted over the rice decoction agar medium (young rice plants 100g, saccharose 20g, agar 20g and de-conized water 2000 ml) containing Kasugamycin at 100 μg/ml. After incubation for 20 hours at 28°C, resistant strains were detected by formation of secondary branch from germ tube.

Rose Maria (1987) reported that *Cochliobolus meyabeanus* (Ito & Kurib.) Drechsler ex Dastur 1942 and *Pyricularia grisea* acquired tolerance to the fungicide edifenphos just by one exposure. *Cochliobolus miyabeanus* and *P. grisea* adopted 11 and 33 times the ED_{50} value of edifenphos, respectively. The acquired tolerance in *C. miyabeanus* was not of permanent nature and was lost on maintenance in fungicide-free medium, but the acquired tolerance of *P. oryzae* was totally retained. However, both fungi lost their sporulating capacity when transferred to fungicide-free media.

Yamaguchi (1988) reported kasugamycin and iprobenfos tolerant strains of *P. grisea* in various parts of Japan and also found that kasugamycin tolerant strains were cross tolerant to blasticidin S, IBP tolerant strains to edifenphos and isoprothiolane.

Yaoita and Goh (1990) investigated the effect blasticides on the development of tolerant strains of *P. grisea*. They opined that the blasticides which were related to the development of tolerant strains were iprobenfos, edifenphos, isoprothiolane, kasugamycin and blasticidin S. They suggested mixed application of fungicides and blasticides for effective control of blast and recommended blasticides with different modes of action and those without cross tolerance.

Viswanathan and Narayanasamy (1991) reported that *Pyricularia grisea* had developed increased tolerance towards mancozeb following repeated transfers in fungicide amended media which was lost after five successive generations on fungicide-free media. They also opined that no acquired resistance occurred in *P. grisea* when cultured on media amended with tricyclazole.

Peng *et al.* (1993) reported field isolates of *Pyricularia grisea* that developed tolerance to isoprothiolane with an average EC_{50} of 17.79 µg/ml compared to 5.71 µg/ml for sensitive isolates. They opined that most tolerant isolates occurred in double harvesting rice areas of Southern China with a frequency of 8.33 per cent.

Zhang Shuya *et al.* (2005) obtained an azoxystrobin resistant mutant *viz.* B9-R2 by exposing *P. grisea* to ultraviolet radiation. The amount of conidia and spore germination of B9-R2 was 65.8 and 12.9 per cent lower than the wild parent isolate B9, respectively. There was no significant difference between B9 and B9-R2 in terms of melanin production and pathogenicity. Azoxystrobin did not inhibit the melanization of B9-R2, but inhibited conidial production, which was reduced by 93.2% when the mutant was grown in AEA media amended with azoxystrobin at 100 µg/ml. Azoxystrobin failed to control blast disease in rice seedlings inoculated with B9-R2 *in vivo*.

2.6 CROSS RESISTANCE

Cross resistance among microorganisms occur when resistance to a chemical is conferred to another chemical by the same genetic factor (Delp, 1980). If a mutation affects sensitivity to one toxicant and not to another, the two compounds are said to be uncorrelated in terms of cross resistance, a positive correlation exists when the mutant is less sensitive or less resistant to both fungicides than the wild type strain (Georgopoulos, 1982). If the mutant is more resistant than the wild type to the one fungicide and more sensitive than wild type to the other, such a phenomenon is called negatively correlated cross resistance.

Brown *et al.* (1984) demonstrated that carbendazim tolerant isolates of *Pseudocercosporella herpotrichoides* were cross tolerant to benomyl, thiophanate-methyl and to a lesser degree to thiobendazole, but not to prochloraz.

Benzimidazole resistant isolates of *Septaria tritici* could tolerate upto 1000 µg/ml of MBC, whereas sensitive isolates could tolerate 0.2 – 0.5 µg/ml. Tolerant isolates were cross resistant to thoibendazole and thiophanate methyl (Fisher and Griffen, 1984).

Carbendazim tolerant isolate of *Gloeosporium ampelophagum* was positively correlated for its tolerance to the combination of carbendazim + captafol, copper oxychloride and mancozeb, while the tolerance to captafol was unaffected. Captafol trained isolate of *G. ampelophagum* exhibited positive cross tolerance to carbendazim+captafol, copper oxychloride and mancozeb (Swarnalatha, 1989).

Mohan and Thind (1995) reported that carbendazim resistant isolates of *G. ampelophagum* showed cross resistance to other formulations of carbendazim but the resistant isolates appear equally sensitive to multisite inhibitors like captan, mancozeb and Bordeaux mixture.

Carbendazim tolerant strain of *Botrytis cinerea* from chickpea showed positively correlated cross tolerance with mancozeb, thiram and tridimefon (Uday Bhan and Chatrath, 1994).

Keinath *et al.* (1996) demonstrated that benomyl resistant strains *Didymella bryoniae* (Auersw.) Rehm. 1881 were cross resistant to thiophanate-methyl. They found that 58 of 59 isolates resistant to benomyl, were also resistant to thiophanate-methyl. Benzimidazole group of fungicides showed negative cross-tolerance to N-(3-4-diethoxy phenyl) carbamate (NPC) fungicides (Elad *et al.*, 1988).

Zhigi *et al.* (2000) reported that carbendazim tolerant isolates of *Sclerotinia sclerotiorum* showed cross tolerance to benomyl, thiabendazole and thiophanatemethyl.

Hemantha Kumar *et al.* (2001a) reported that cross resistance of carbendazim resistant isolates of *G. ampelophagum* to hexaconazole, mancozeb, copper oxychloride, Bordeaux mixture, dithianon and chlorothalonil was unaltered.

Butters *et al.* (1984) reported that barley powdery mildew has become less sensitive to triadimenol throughout the United Kingdom during the 14 years of study. Fungicide tolerant strains were as pathogenic as sensitive strains and were cross-tolerant to other C-14 demethylation inhibitors but, not to morpholine fungicides. Sensitivity to triadimenol was negatively correlated with ethirimol.

Triadimefon resistant isolates of *Erysiphe graminis* f.sp. *hordei* from barley and *E. graminis* f.sp. *tritici* from wheat showed cross tolerance to other sterol inhibitors such as triadimenol, propiconazole, diclobutrazole, prochlaraz and nuarimol. No cross tolerance was detected to triademorph, fenpropimorph and pyrazophos (Buchenauer and Hellwald, 1985).

Erickson and Wilcox (1996) reported that triadimefon resistant isolate of *Uncinula necator* (Schwein.) Burrill 1892 was cross resistant to myclobutanil. Studies on detection and quantification of resistance of *Venturia inaequalis* population to sterol biosynthesis inhibitors (Koller *et al.*, 1997) revealed that a high degree of cross resistance between fenarimol and myclobutanil existed and the sensitivity tests with one of the demethylation inhibitors employed in the diagnostic tool can serve as a test for other DMIS.

2.7 RESISTANCE MANAGEMENT

Measures should be taken before a build up of resistance among isolates of the pathogen. Once the pathogen population has become resistant, the only possibility is to change over to other fungicides with different mechanisms of action or to non-chemical control measures. Different strategies have been proposed by Delp (1980), Gilpatrick (1983) and Dekker (1985) to overcome or delay the build up of fungicide resistance in the field. A comprehensive review of such anti resistance measure has been given by Staub (1991). Some of the more commonly recommended practices to prevent or delay the development of resistance were use of fungicides in mixture, alternation of fungicides and use of negatively correlated cross resistance fungicides.

2.7.1 Use of fungicides in mixture

Mixtures of resistance prone fungicides with other fungicides having a different mechanisms of action provide one of the best strategies that have proved highly effective to limit the build up of fungicide resistance in the field. Use of mixture of metalaxyl with mancozeb used against *Phytophthora infestans* on potato in UK compared to metalaxyl alone in Netherlands and Ireland serve as an ideal example for use of combinations to avoid build up of resistance (Staub and Sozzi, 1983, 1984; Davidse and

Dewaard, 1984). Delp (1981) reported that no resistance was encountered when metalaxyl was used with some companion fungicide against oomycetes foliar pathogens.

Tivoli *et al.* (1986) reported that thiabendazole in combination with imazalil was found to reduce the infection caused by thiabendazole resistant strains of *Fusarium roseum* var. *sambercinum*. *Phytophthora infestans* and *Pythium aphanidermatum* developed resistance to phenylamide fungicides. Application of mixtures of a phenylamide fungicide with mancozeb or mancozeb plus cymoxanil decreased the build up of phenylamide resistance, compared with phenylamide alone (Sanders *et al.*, 1985; Samoucha and Gisi, 1987; Cohen and Samoucha, 1990).

In studies on cereal powdery mildew (*Erysiphe-graminis* f.sp. *tritici* and *Erysiphe graminis* f.sp. *hordei*), field application of mixtures of triazoles with morpholine or amino pyrimidine fungicides was found to hinder the development of resistance (Bolton and Smith, 1988; Brent *et al.*, 1989; Heaney *et al.*, 1988; Lorenz *et al.*, 1992).

Benzimidazole fungicides are commonly used for the control of cercospora leaf spots of peanut in USA. In South Eastern states, where there was sole use of benomyl, practical resistance soon appeared. In Texas, where benzimidazole mancozeb mixtures were used from the start, no resistance developed except in trial plots where a benzimidazole alone was applied repeatedly (Smith, 1988).

Steva (1994) reported that reducing the rate of triadimenol to half enhanced development of resistance in grape powdery mildew fungus in France. Split application (lower doses but more frequent) of fenpropimorph and fenpropimorph-propiconazole mixtures led to significant decreases in fenpropimorph sensitivity of wheat powdery mildew fungus in Germany and Holland (Engels and Dewaard, 1994; Forster *et al.*, 1994).

Fusarium graminearum developed resistance to carbendazim in China. Application of mixtures of fungicides with different target sites or with different mechanisms of action appears to block or slow the development of resistance. Kangiunling, a new mixture of carbendazim, triadimeton and thiram, has been more effective than carbendazim alone in field trials at various sites in China (Shao *et al.*, 1998, Xu and Pan, 1998 and Wu, 1998).

Hemantha Kumar *et al.* (2001b) reported that *G. ampelophagum* developed resistance to carbendazim. So, use of hexaconazole alone or in combination with carbendazim was found to be most effective in managing the resistance development in grape anthracnose fungus.

G. ampelophagum developed resistance to carbendazim. When carbendazim was used in mixture with benomyl, fenarimol, thiophanate-methyl and tridemorph it reduced the growth of the pathogen (Deokate *et al.*, 2002b).

Huang Xing *et al.* (2005) evaluated the efficacy of tricyclazole, prochloraz and their mixture at 2:1, 4:1, 7:1 and 9:1 against rice blast *in vivo*. The 4:1 mixture of tricyclazole and prochloraz had the best control effect against P. grisea. The synergistic ration of the mixture was 2.28. The high control effect was due to inhibitory effect on spore formation and hypal growth with EC_{50} value of 0.238 and 0.346 µg/ml, respectively. The preventive and curative effects of the mixture could last for 7-10 days. The mixture also had control effect on wounded leaves and possessed a good residual performance.

2.7.2 Use of negatively correlated cross resistance fungicides

Negative cross resistance between two fungicides is a promising phenomenon for precluding resistance problems. Negative correlated cross resistance has

been observed for benomyl and phenyl carbamates in case of *Botrytis cinerea* in grapes and *Venturia nashicola* S. Tanaka & S. Yamam 1964 in pears (Kato *et al.*, 1984; Ishii *et al.*, 1985).

Dewaard and Vannistelrooy (1983) reported that majority of fenarimol-resistant isolates of *Aspergillus nidulans* (Eidam) G. Winter 1884, *Cladosperium cucumerium Penicellium expansum* Link 1809, *Penicillium italicum* Wehmer 1894, *Ustilago maydis* (DC.) Corda 1842 *in vitro* displayed a moderate degree of negatively correlated cross-resistance to dodine. A limited number of isolates also possessed an increased sensitivity to geuzatine. Rosenberger and Meyer (1985) reported that benomyl resistant *Penicillium expansum* showed negatively correlated cross resistance to diphenylamine.

One special and interesting approach to overcoming benzimidazole resistance has been the application of a mixture of the benzimidazole fungicide carbendazim with diethofencarbi to control *Botrytis* in grapes and other crops. Diethofencarb show negative cross-resistance with respect to benzimidazoles. Remarkebly, it inhibits only benzimidazole-resistant strains of the target pathogens and does not affect benzimidazole-sensitive strains (Elad *et al.*, 1992).

The effect of four new fungicidal formulations propineb (Antracol 70 WP), prochloraz (Octava 50WP), epoxiconazole (opus 12.5SC) and tricyclazole (Beam 75WP) against rice blast on the rice cultivar Taraori Basmati was investigated by Ram Singh *et al.* (1999). They said that among the fungicides, tricyclazole application resulted in the highest reduction in disease incidence (74.6%) followed by epoxiconazole.

Material & Methods

CHAPTER III

MATERIAL AND METHODS

3.1 LOCATION OF WORK

The experiments were conducted at the Directorate of Rice Research, Rajendranagar, Hyderabad, Andhra Pradesh which is located at a Longitude of 78° 23' E, Latitude of 17° 19' N and altitude of 542.3 m above mean sea level in tropical zone of South India during Kharif 2002-03 and 2003-04.

3.2 LABORATORY TECHNIQUES

The general laboratory techniques adopted in the present study were those described by Nene and Thapliyal (1979), Aneja (1993) and Dhingra and Sinclair (1994) with slight modification wherever necessary. Mention has been made for other special procedures and techniques used in the course of investigation.

3.2.1 Glassware

Corning/Borosil brand glassware were used in all the experiments. Petriplates (90 mm dia), test tubes, conical flasks of 100, 250, 500, 1000 and 2000 ml, micropipettes of 1-5 µl, 5-50 µl, 40-200 µl, macropipettes of 1-5 ml, measuring cylinders of 5, 10, 25, 50, 100, 500 and 1000 ml capacity were used. Glassware were thoroughly cleaned with detergent and washed with running tap water and later placed over night in cleaning solution [prepared by adding 60 g of potassium dichromate ($K_2Cr_2O_7$) into 300 ml of distilled water and then adding 60 ml of concentrated sulphuric acid (H_2SO_4) drop by drop and the final volume was made to 1000 ml with distilled water. The cleaned glassware were finally rinsed with distilled water and air dried.

3.3 BASAL MEDIUM

Oat meal agar (OMA) was used as the basal medium for isolation, maintenance and growth of *P. grisea*.

3.3.1 Composition:

Oats	:	20 g
Dextrose	:	20 g
Agar agar	:	20 g
Distilled water	:	1000 ml

3.3.2 Preparation of basal medium

Oat meal agar medium was prepared by boiling 20 g of oats in 500 ml of water for about 30 minutes in a conical flask. In another conical flask containing 500 ml of water, 20 g agar was melted. Both of these were filtered through a three layered muslin cloth into a third beaker and 20 g of dextrose was added to the filtrate. The final volume was made upto 1 litre by adding water. The pH of the medium was adjusted to 6.5 before sterilization by adding 1 N HCl or 1 N NaOH as the case may be. The medium was sterilized in an autoclave at 15 PSI (121.6^0 C) for 20 minutes.

3.4 WATER AGAR

Water agar medium (1.5%) was also used in addition to oat meal agar medium for the isolation of *P. grisea* from plant samples collected from different parts of India.

3.4.1 Composition

Agar agar	:	15 g
Distilled Water	:	1000 ml

3.4.2 Preparation of water agar

Water agar medium was prepared by melting 15 g of agar in 500 ml of water. The preparation was filtered through muslin cloth and final volume of the medium was made upto one litre by adding water. The medium was sterilized in an autoclave at 15 PSI (121.6^0 C) for 20 minutes.

3.5 CHEMICALS

Analytical or general grade chemicals of BDH or Sarabhai were used in all the investigations.

3.6 COLLECTION AND MAINTENANCE OF PYRICULARIA GRISEA ISOLATES

Leaf samples from blast infected paddy fields with known history of fungicide usage were collected from different parts of India (Table 1; Fig. 1 & Plates 1 to 5). Leaf samples were also collected from paddy fields where there was no fungicide usage. The leaf samples were surface sterilized in 0.1 per cent mercuric chloride and then washed three times in sterile distilled water and were kept in a moist chamber for 12 h under sterile conditions to induce sporulation. The spores thus obtained were gently tapped on to plain agar medium in 90 cm diameter petriplates. The petriplates were kept in the BOD incubator for 24 h at 28±1 °C. The germinated single spores were marked using stereobinocular microscope and were transferred on to oat meal agar medium (OMA) separately

Table 1: Details of *Pyricularia* grisea isolates collected from different parts of India

S. No	Name of the isolate	Location/Place of collection	State/Union Territory	Variety/Cultivar
1	Almora	VPKAS, Hawalbagh	Uttaranchal	VLK–39
2	Cuttak	CRRI, Cuttak	Orissa	IR-50
3	Gerua	RRLRRS, Gerua,	Assam	Tapaswini
4	Kadapa	Chennur, Kadapa	Andhra Pradesh	NDLR-8
5	Kapurthala	RRS, Kapurthala	Punjab	PR–116
6	Malan	RRS, Malan	Himachal Pradesh	T-23
7	Mandya	V.C.Farm, Mandya	Karnataka	Mandya Vijaya
8	Maruteru	ARS, Maruteru	Andhra Pradesh	Samba Masuri
9	Medchel	Dindigal	Andhra Pradesh	Samba Masuri
10	Naidupeta	Gottiprolu, Nellore	Andhra Pradesh	Samba Masuri
11	Nellore	ARS, Nellore	Andhra Pradesh	Samba Masuri
12	Pattambi	RARS, Pattambi	Kerala	IR-50
13	Pondicherry	KVK, Pondicherry	Union Territory	IR-50
14	Rajendranagar	DRR, Rajendranagar	Andhra Pradesh	HR-12
15	Tirupathi	Avilala, Chittoor	Andhra Pradesh	IET-1444

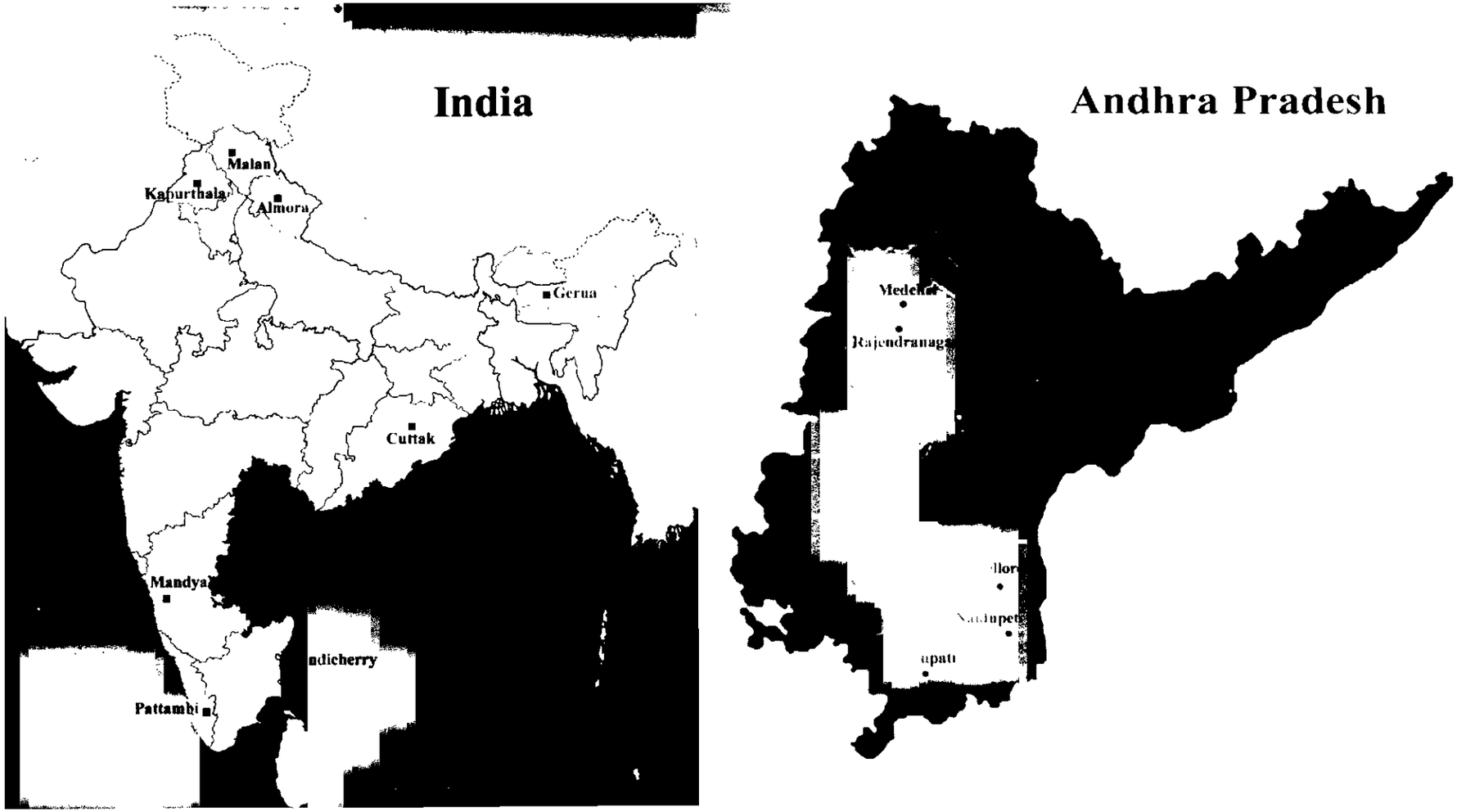


Fig.1: Map showing the areas from which rice blast disease samples were collected in India and Andhra Pradesh state



Plate 1: Leaf blast symptoms caused by *Pyricularia grisea* in the main field

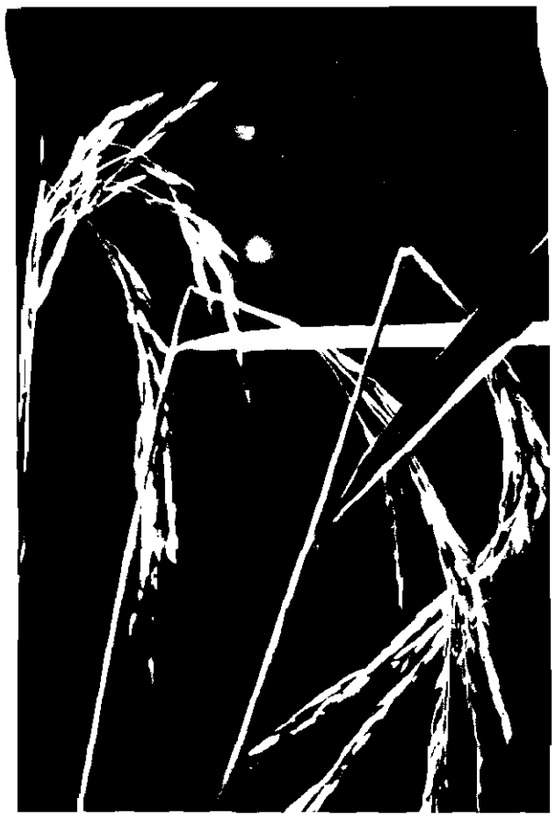


Plate 2: Neck blast symptoms (broken necks) caused by *Pyricularia grisea*



Plate 3: Neck blast symptoms (lesioned necks) caused by *Pyricularia grisea*

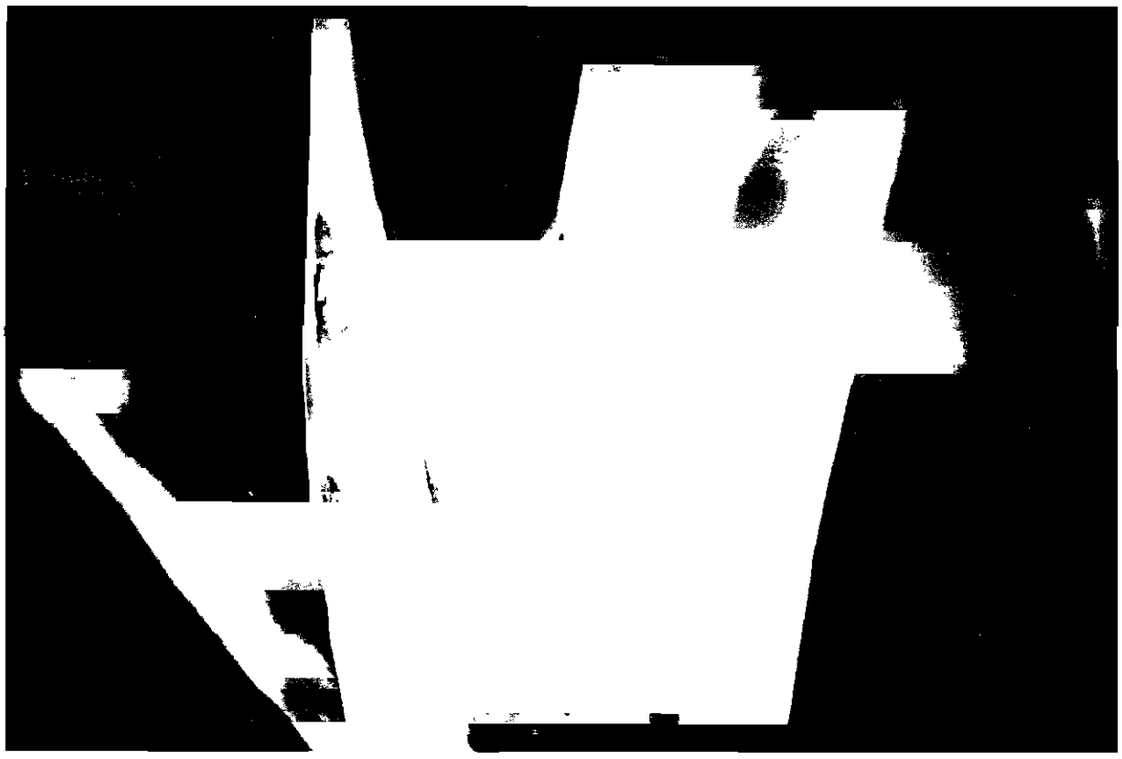


Plate 4: Leaf blast symptoms caused by *Pyricularia grisea* in the nursery

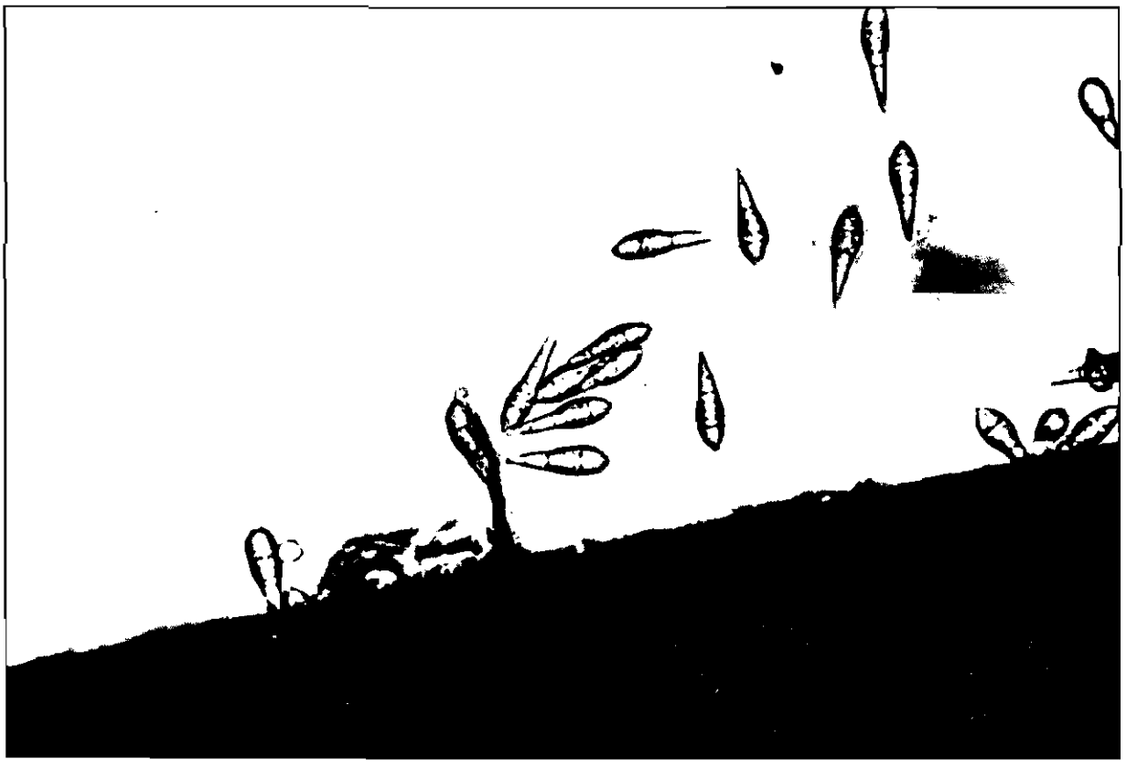


Plate 5: Conidia of *Pyricularia grisea* *(400X)*

and kept in the incubator at 28±1 °C. The pure cultures thus obtained were maintained on OMA slants (Dhingra and Sinclair, 1994).

3.7 PATHOGENICITY TEST

The pathogenicity of the isolates was tested by inoculating 15 day old seedlings of HR-12 rice cultivar with conidial suspensions (1 x 10^6 conidia/ml) of *P. grisea*. After one week, the plants were observed for the development of blast lesions.

3.8 SCREENING OF RICE CULTIVARS TO STUDY THEIR RELATIVE RESISTANCE TO PYRICULARIA GRISEA

One hundred forty four rice cultivars were screened using Uniform Blast Nursery (UBN) pattern (Plate 10). Each test cultivar was sown in a single row of 50 cm length at 10 cm apart. After every ten cultivars, the local susceptible variety, HR-12 was sown. The entire nursery was surrounded on all sides by two rows of HR-12. Rajendranagar isolate of *P. grisea* was cultured on OMA plates at 28°C. The plates fully covered with mycelia were placed under continuous illumination to induce sporulation. The sporulated plates were flooded with water and scraped to dislodge the conidia. Later, the suspension was filtered through a three layered cheese cloth and gelatin was added at 5 g/ l. The concentration of the inoculum was adjusted to 1 x 10^6 conidia/ml with the help of haemocytometer. Inoculation was done by spraying the inoculum on 15 days old seedlings. The observations were recorded based on leaf blast severity on 0-9 scale of standard evaluation system (SES, 2002) of rice developed by IRRI. Three cultivars *viz.*, one highly susceptible, one moderately resistant and one highly resistant were selected for further studies on induced systemic acquired resistance (Plate 8).

Score	Disease reaction
0	No lesions observed
1	Small brown specks of pin point size or larger brown specks without sporulating center
2	Small roundish to slightly elongated, necrotic gray spots, about 1-2 mm in diameter, with a distinct brown margin
3	Lesion type is the same as in scale 2, but a significant number of lesions are on the upper leaves
4	Typical susceptible blast lesions 3 mm or longer, infecting less than 4% of the leaf area
5	Typical blast lesions infecting 4-10% of the leaf area
6	Typical blast lesions infecting 11-25% of the leaf area
7	Typical blast lesions infecting 26-50% of the leaf area
8	Typical blast lesions infecting 51-75% of the leaf area and many leaves are dead
9	More than 75% of the leaf area affected

3.9 IN VITRO TOXICITY OF SYSTEMIC ACQUIRED RESISTANCE INDUCING CHEMICALS ON P. GRISEA

Inhibition zone technique (Thornberry, 1950) was employed with modification. Spore suspension from culture tube was collected by dispensing sterilized distilled water into it and shaking vigorously. The spore suspension was filtered through sterilized cheese cloth. Seeded agar was prepared by adding spore suspension to warm molten agar to provide 0.5 – 1.0 x 10^6 viable test fungus spores/ml of seeded agar. Twenty ml of seeded agar was dispensed into each 90 mm, flat bottomed petriplates and the plates were rotated horizontally to ensure even coverage of the agar before it solidifies. These plates with tops in place were stacked in a refrigerator (2°-4°C) upside down to prevent condensation of moisture.

Wells of 5 mm diameter were made at the centre of each petriplate in the chilled seeded agar and 90 µl of test chemical was poured in to each well. Different SAR inducing chemicals were tested at 1, 10^{-1}, 10^{-2}, 10^{-3}, 10^{-4} and 10^{-5} M concentrations and each treatment was replicated thrice in a completely randomized design (CRD). Controls were maintained for all the experiments adding 90 µl of sterile distilled water. The zones of inhibition of the treatments were compared with those of control.

3.10 INDUCTION OF SYSTEMIC ACQUIRED RESISTANCE IN RICE AGAINST P. GRISEA USING CHEMICALS

Three rice cultivars, one highly susceptible (HR-12), one moderately resistant (Swarnadhan) and one highly resistant (Rasi) were selected to study their response to the systemic acquired resistance inducing chemicals. Five systemic acquired resistance (SAR) inducing chemicals *viz.* salicylic acid, acibenzolar-S-methyl (BTH), ferric chloride, sodium selenite and zinc sulphate at 10^{-3}M, 10^{-3}M, 10^{-4}M, 10^{-5}M and 10^{-3}M concentrations, respectively were tested. The investigations were carried out in the plastic trays (30×20 cm) under glass house conditions by imposing the following treatments:

1. Seed treatment alone
2. Seed treatment + one spray at 15 days after sowing (DAS)
3. Seed treatment + one spray at 15 DAS + 2nd spray at 22 DAS

Bold and healthy seeds of Rasi, Swarnadhan and HR-12 varieties were soaked over night in salicylic acid 10^{-3}M, BTH 10^{-3}M, ferric chloride10^{-4}M, sodium selenite 10^{-5}M and zinc sulphate 10^{-3}M solutions, separately and sown in plastic trays containing sterilized soil with each individual treatment replicated four times. The first spray of SAR-inducing chemicals was given 15 days after

sowing and the next spray was given 22 days after sowing in the respective treatments. All the treatments were inoculated simultaneously at 30 DAS with spore suspension (1×10^6 conidia/ml) of *P. grisea*. Observations on seed germination, incubation period, lesion number, lesion size and sporulation/lesion were taken as follows –

3.10.1 Seed germination (%)

Before sowing the seed in the plastic trays, samples of treated seed from each individual chemical were drawn separately for each individual variety. The seed thus collected was tested for germination by following standard seed germination procedure developed by the International Seed Testing Association (ISTA, 1985). The observations were recorded as per cent seed germination.

3.10.2 Incubation period (days)

Incubation period was measured as the time taken from the date of inoculation with *P. grisea* to the expression of symptoms in the form of necrotic specks and was expressed in number of days.

3.10.3 Number of lesions/leaf

The number of lesions per leaf was recorded on the second leaf from the top at 10 DAI. Five random samples/tray were taken to represent the average number of lesions per leaf.

3.10.4 Lesion size

Lesion size was measured using a transparent graph sheet for the second leaf from the top at 10 DAI. Five leaves/tray with sporulating lesions were selected randomly to calculate the average lesion size in square millimeter.

3.10.5 Sporulation/lesion

Ten sporulating lesions per tray were excised and placed in a scintillation vial each containing 1 ml of 1 per cent (wt/vol) copper sulphate solution enriched with a small drop of 1 per cent Triton X-100 (vol/vol) solution to prevent spore germination (Seebold *et al.*, 2001). The vials were then stored at 3°C in the refrigerator until evaluation. The vials were agitated vigorously for two minutes before evaluation. Ten samples were drawn from each vial and the number of conidia were counted using haemocytometer and mathematically related to the number of conidia per millimeter. Since the lesion size varied for different treatments, sporulation was expressed as number of conidia/mm^2 rather than number conidia/lesion as follows. After counting conidia, excised lesions were removed from scintillation vials and fixed to a sheet of paper. The area of the lesion was measured with the help of a transparent graph sheet. The number of conidia from a given lesion was divided by the corresponding lesion size to obtain the number of conidia per square millimeter of lesion.

3.10.6 Leaf blast severity

The disease incidence was recorded by visual estimation of leaf blast severity in each treatment at 5, 10, 15 and 20 days after inoculation. Five

random seedlings were selected per tray and the diseased incidence was measured on top three leaves. Leaf blast severity was expressed as per cent leaf area infected.

3.11 TESTING THE EFFICACY OF FUNGICIDES ON P. GRISEA

The details of the fungicides used for investigation against *P. grisea* both *in vitro* and *in vivo* are furnished in Table 2.

3.11.1 In the laboratory

Each fungicide was tested at different concentrations *viz.* 0, 1, 5, 10, 25, 50, 100, 250, 500 and 1000 µg/ml by adopting inhibition zone technique as described earlier. Rajendranagar isolate of *P. grisea* was employed for investigation. The size of the inhibition zone was measure in millimeters and compared with the control plates.

3.11.2 In the nursery beds

The details of fungicides and their dosages for both seed treatment and foliar spray are furnished in Table 3.

3.11.2.1 Seed treatment

Seed dip method (Nene and Tapliyal, 1971) was employed for seed treatment. Fungicide solution/suspension was prepared in water. Bold and healthy seed of highly susceptible variety HR-12 was dipped in fungicide solution/suspension over night. Next day, seed was sown in the nursery beds and a sample of treated seed for each fungicide was drawn separately. The seed thus collected was used to study the seed germination (ISTA, 1985). Seed dipped in plain water was used as control. Treated seed was sown in rows of 100 cm length

Table 2: Details of fungicides employed in the investigation

S.No.	Common name	Trade name	Chemical name	Source of supply
1.	Carbendazim	Bavistin 50 WP	methyl benzimidazol-2-ylcarbamate	BASF
2.	Edifenphos	Hinosan 50 EC	O-ethyl S, S-diphenyl phosphorodithioate	Bayer
3.	Iprobenfos	Kitazin 48 EC	S-benzyl O,O-di-isopropyl phosphorothioate	Pesticides India
4.	Isoprothiolane	Fuji-One 40 EC	di-isopropyl 1,3-dithiolan-2-ylidenemelonate	Rallis
5.	Pyroquilon	Fungorene 50 WP	1,2,5,6-tetrahydropyrrolo-4H-Pyrrolo [3,2,1-ij] quinolin-4-one	Syngenta
6.	Kasugamycin	Kasu-B 2L	1L-1,3,4/2,5,6-1-deoxy-2,3,4,5,6-penta hydroxy cyclohexyl 2-amino-2,3,4,6-tetra deoxy-4-(α-iminoglycino)-α-D-arabino-hexopyranoside hydrochloride hydrate: [S-amino-2-methyl-6-(2,3,4,5,6-penta hydroxy cyclo=hexyloxyl_ tetra hydropyran-3-yl] amino-α-iminoacetic acid hydrochloride hydrate	Dhanuka
7.	Difenconazole	Score 25 EC	Cis,trans-3-chloro-4-[4-methyl-2-(1H-1,2,4-triazol-1-ylmethyl)-=1,3-dioxolan-2-yl] phenyl 4-chlorophenyl ether	Syngenta
8.	Diniconazole	Sumi-8 25 WP	(E)-(R)-1-(2,4-dichlorophenyl)-4,4-dimethyl-2-(1H-1,2,4-triazol-1-yl)Pent-1-en-3-ol	Sumitomo
9.	Epoxiconazole	Opal 7.5 EC	(2RS,3SR)-1-[3-(2-chlorophenyl)2,3-epoxy-2-(4-fluorophenyl)-Propyl]-1H-1,2,4-triazole	BASF
10.	Propiconazole	Tilt 25 EC	(±)-1-[2-(2,4-dichlorophenyl)-4-propyl-1,3-dioxalan-2-yl methyl]-1H-1,2,4-triazole	Syngenta
11.	Tricyclazole	Sivic 75 WP	S-methyl-2,3,4-triazole [3,4-b] [1,3] benzothiozole	NFCL
12.	Pyraclostrobin	F-500 25 EC	methyl N-{2-[2-(4-chlorophenyl)-1 H-pyrazole-3-yloxymethyl]phenyl}(N-methoxy) carbamate	BASF
13.	Fenoxanil	Fenoxanil 20 SC	mixture of 85% (R)-N-[(RS)-1-cyano-1,2-dimethylpropyl]-2-(2,4-dichlorophenoxy) propionamide and 15% (S)-N-[(RS)-1-cyano-1,2-dimethylpropyl]-2-(2,4-dichlorophenoxy)propionamide	BASF
14.	RIL 010 F_1 25 SC	-	Methoxyacrylate	Rallis
15.	Azoxystrobin	Amistar 25 SC	Methyl(E)-2-{2-[6-(2-cyanophenoxy)Pyrimidin-4-yloxy]Phenyl}-3-methoxy acrylate	Syngenta
16.	Carbendazim + Epoxicanozole	Swing 25 EC	As given above	BASF
17.	Difenconazole + Propiconazole	Armure 30 EC	As given above	Syngenta
18.	Mancozeb	Indofil M-45 75WP	[[1,2-ethanediylbis (carbamodithioato)](2-)] manganese mixture with [[1,2-ethanediylbis (carbamodithioato)](2-)]zine	Indofil
19.	BTH	Bion 50WG	benzo-(1,2,3)-thiadiazole-7-carbothioic acid-S-methyl ester	Syngenta

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Table 3: Formulation and dosage details of fungicides used in the investigation for seed treatment and foliar spray

S. No.	Common Name	Trade Name	Formulation	Dosage	
				Seed treatment (g/kg or ml/kg)	Foliar spray (g/l or ml/l)
1	Azoxystrobin	Amistar	25 SC	2.0	1.0
2	Carbendazim	Bavistin	50 WP	2.0	1.0
3	Difenconazole	Score	25 EC	2.0	2.0
4	Diniconazole	Sumi-8	25 WP	2.0	2.0
5	Edifenphos	Hinosan	50 EC	2.0	1.0
6	Epoxiconazole	Opal	7.5 EC	2.0	2.0
7	Fenoxanil	Fenoxanil	20 SC	2.0	1.0
8	Iprobenfos	Kitazin	48 EC	2.0	1.5
9	Isoprothiolane	Fuzi-One	40 EC	2.0	1.5
10	Kasugamycin	Kasu-B	2 L	2.0	2.0
11	RIL 010	-	25 SC	2.0	1.0
12	Propiconazole	Tilt	25 EC	2.0	2.0
13	Pyraclostrobin	F-500	25 EC	2.0	1.0
14	Pyroquilon	Fungorene	50 WP	2.0	1.0
15	Tricyclazole	Sivic	75 WP	2.0	0.6
16	Carbendazim + Epoxiconazole	Swing	25 EC	2.0	1.0
17	Difenconazole + Propiconazole	Armure	30 EC	2.0	2.0

at 10 cm apart in 1 x 1 m nursery beds. The beds were watered regularly using sprinklers. Fifteen day old seedlings were inoculated with *P. grisea* (1 x 10^6 conidia/ml). The nursery beds were covered with polythene sheet from evening to morning every day to build inoculum pressure. Sprinklers were employed for watering beds, increasing relative humidity and keeping leaf surfaces wet (Plate 9). Observations were recorded on seed germination (%), incubation period (days) and leaf blast severity (%). The observations on leaf blast severity were taken by visual estimation at 5 day interval starting from fifth day after inoculation until disease severity in the untreated control reached the maximum.

3.11.2.2 Foliar spray

Bold and healthy seed of highly susceptible cultivar HR-12 was soaked in water over night. Next day, the seed was sown in rows of 100 cm at length 10 cm apart in 1 x 1 m nursery beds. The beds were watered regularly using sprinklers. Fifteen day old seedlings were inoculated with Rajendranagar isolate of *P. grisea* (1 x 10^6 conidia/ml). The plots were sprayed with fungicides immediately after the appearance of initial disease symptoms. The observations on leaf blast severity were taken by visual estimation at 5 day interval starting from fifth day after inoculation until disease severity in the untreated control reached the maximum (Plate 11).

3.11.3 In the main field

Field trials were conducted for two consecutive seasons during *Kharif* 2002 and *Kharif*, 2003, respectively under artificial inoculation conditions at the Directorate of Rice Research, Rajendranagar, Hyderabad. Twenty five days old seedlings were transplanted at a spacing of 20 x 15 cm in 2.5 x 2.0 m size plots

under recommended fertilizer doses *viz.* N 100 kg/ha (Urea), P 60 kg/ha (SSP) and K 40 kg/ha (MOP) in the main field. Inoculation was done at maximum tillering stage.

3.11.3.1 Mass multiplication of *P. grisea* inoculum:

Sorghum grain of the variety CSH-11 was soaked in plain water for one hour. The grain was thoroughly washed with tap water and then boiled in water in an open vessel on a gas stove for 30 minutes. The excess water was drained out. Conical flasks (250 or 500 ml) were filled with boiled grain upto one third of their volume. Three to five ml of 0.2% yeast extract solution was added into each flask to enhance sporulation. The conical flasks were autoclaved at 15 PSI (121.6^0 C) for 20 minutes. The flasks were seeded with 7 mm mycelial disks of *P. grisea* (5 no. each) in an inoculation chamber when they were at lukewarm state. Care was taken to see that the grain was moist but not wet during inoculation. The flasks were gently shaken for thorough mixing of the inoculum. The seeded flasks were kept for incubation at 28 ± 1° C in a BOD incubator. After 7 days, the ramified sporulating mycelium in the conical flasks was raked using a glass rod by adding sufficient quantity of water and then filtered through a three layered cheese cloth. The spore concentration was adjusted to 1 x 10^6 conidia per ml with the help of a haemocytometer and 0.5% gelatin was added (Plates 6 & 7).

First spray of fungicides was done immediately after the appearance of initial disease symptoms at maximum tillering stage to control leaf blast. The second spray was done at flowering stage for panicle blast. Observations were recorded thrice at 10 day interval for leaf blast starting from 10 days after spray of fungicides as per cent leaf blast severity. Neck blast was recorded only once at

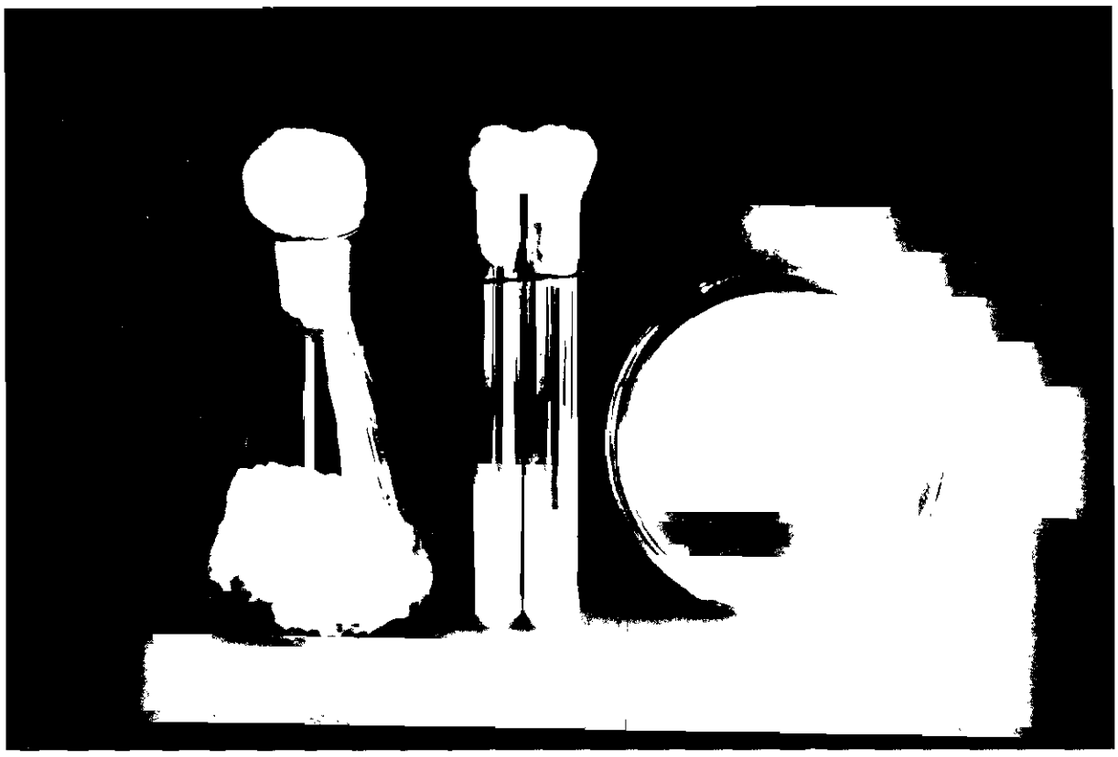


Plate 6: Laboratory culture of *Pyricularia grisea*



Plate 7: Mass multiplication of *P. grisea* on sorghum grain

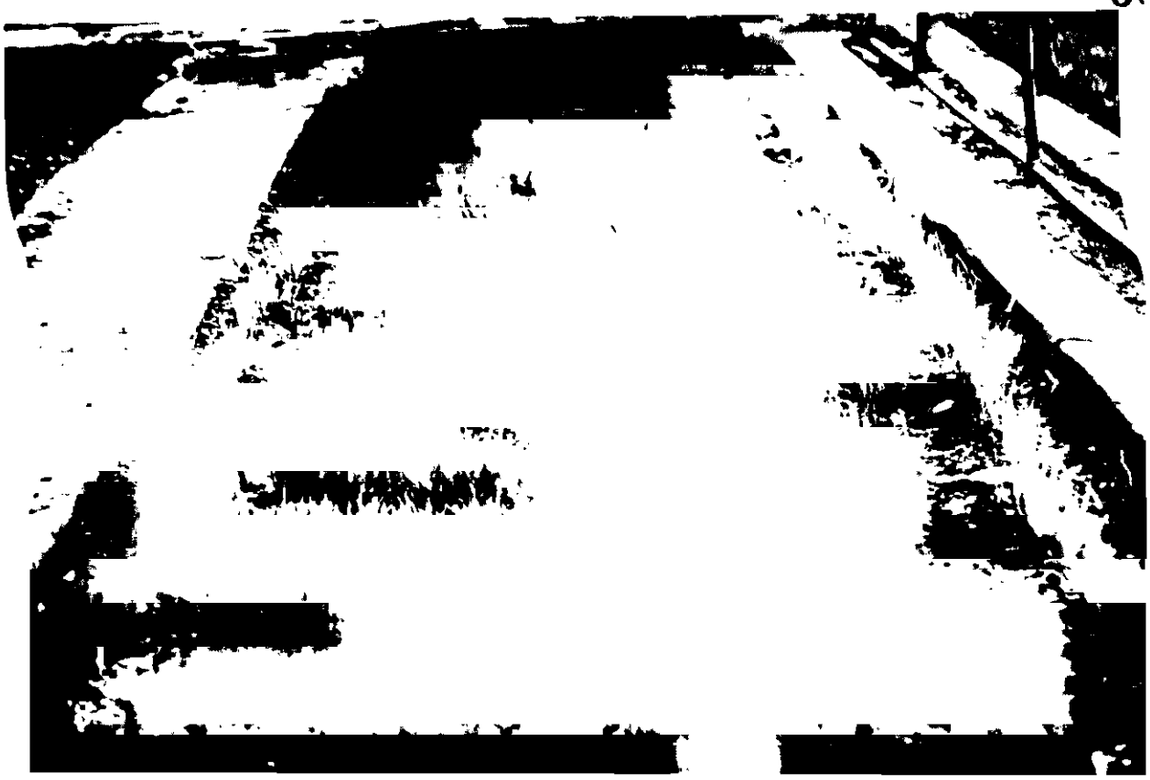


Plate 8: Uniform Blast Nursery (UBN) pattern



Plate 9: Sprinklers used to increase the relative humidity



Plate 10: Screening of rice cultivars for host plant resistance against rice blast

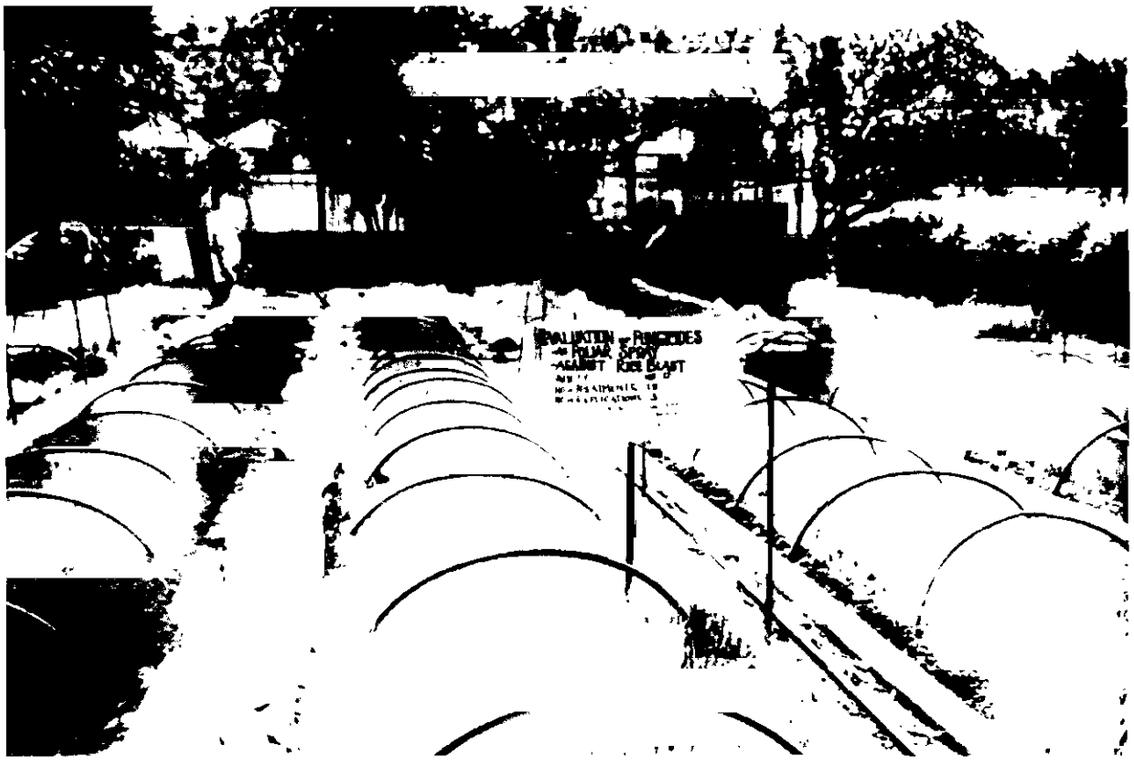


Plate 11: Evaluation of fungicides as foliar spray against rice blast in the nursery

grain maturity stage in terms of lesioned necks and broken necks. Observations on grain yield were also recorded as quantity of ill filled grain, filled grain and total grain yield.

All the nursery and field experiments were laid-out in randomized block design (RBD) with 18 treatments each replicated thrice while completely randomized block design (CRD) was employed for the *in vitro* studies.

3.12 TESTING FOR THE DEVELOPMENT OF RESISTANCE IN P. GRISEA TO CARBENDAZIM, EDIFENPHOS AND IPROBENFOS

3.12.1 Mycelial growth studies

The relative sensitivity of the isolates obtained from different parts of India was assayed individually at different concentrations (0-1000 µg/ml) on OMA in the laboratory by adopting poisoned food technique (Dhingra and Sinclair, 1994). Different concentrations of carbendazim, edifenphos and iprobenfos *viz.,* 1, 5, 10, 25, 50, 100, 250, 500 and 1000 µg/ml were prepared individually in sterilized oat meal agar (OMA). The required quantity of fungicide on active ingredient (a.i.) basis was added to OMA at lukewarm state and the poisoned medium was poured into culture plates. With the help of a cork borer, 5 mm agar discs of active growing fungus were cut and placed in the centre of fresh plates for seeding. Similarly, controls were maintained by placing 5 mm disk of test fungus at the centre of unpoisoned medium in the plates. Each treatment was replicated thrice and all the culture plates were incubated at 28±1 °C in BOD incubator.

Colony diameter was recorded after 9 days of incubation. The individual ED_{50} values were calculated by using probit analysis (Finny, 1952) while the minimum inhibitory concentration (MIC) values were calculated from the per cent growth inhibition data recorded at different concentrations of the fungicides studied. Per cent growth inhibition was calculated in each treatment by comparing with control plates (Vincent, 1969) using the formula

$$I = 100 (C - T)/C$$

Where, I = Incubation percentage
C = Growth in control
T = Growth in treatment

3.12.2 Spore germination studies

The spore germination technique developed by Montgomery and Moore (1938) was followed. On each clean, non-greasy glass slide, three circles of one cm diameter were drawn with the help of a marker pen at equal distance from each other. The test fungicidal solution/suspension of 50 µl was placed and spread evenly in the marked circle on the slide. Fungicide concentrations were dispensed with 50 µl micro pipette for each fungicide, proceeding from lower to higher concentration taking care to see that the concentrations were in no way affected during distribution. After drying of the solution/suspension 50 µl of conidial suspension was placed by means of 50 µl micro pipette and was spread in the marked circle. The seeded slides were incubated in the moist chamber at ambient room temperature (26-30°C). Spore germination counts were taken after 24 hours of incubation in the moist chambers and expressed as percentage.

The individual ED_{50} values were calculated by using probit analysis and minimum inhibitory concentration (MIC) values were calculated from per cent inhibition of spore germination.

3.13 TESTING THE CROSS RESISTANCE OF RESISTANT ISOLATES OF PYRICULARIA GRISEA TO OTHER BLASTICIDES

Cross resistance of carbendazim resistant isolate of *P. grisea* to edifenphos, iprobenfos, Kasugamycin, isoprothiolane and pyraclostrobin was determined by comparing its response to carbendazim sensitive isolate. Cross resistance of edifenphos resistant isolate of *P. grisea* to carbendazim, iprobenfos, Kasugamycin, isoprothiolane and pyraclostrobin was determined by comparing its response to edifenphos sensitive isolate. Cross resistance of iprobenfos resistant strain of *P. grisea* to carbendazim, edifenphos, kasugamycin, isoprothiolane and pyraclostrobin was determined by comparing its response to iprobenfos sensitive isolate. These isolates were grown in media containing the test fungicide at various concentrations *viz.*, 0, 1, 5, 10, 25, 50, 100, 250, 500 and 1000 µg/ml of their respective fungicide by adopting poisoned food technique. Each treatment was replicated thrice. ED_{50} and MIC values were calculated. Similarly, spore germination technique was adopted to determine ED_{50} and MIC values as described earlier.

3.14 TESTING THE PESTISTENCE OF ACQUIRED RESISTANCE OF THE RESISTANT ISOLATES

Carbendazim, edifenphos and iprobenfos resistant isolates of *P. grisea* were passed through fungicide free OMA for 12 successive generations. After 12 generations the isolates were tested at different concentrations of fungicide *viz.*, 0, 1, 5, 10, 25, 50, 100, 250, 500 and 1000 µg/ml of carbendazim,

edifenphos and iprobenfos, respectively. ED_{50} and MIC values were calculated and compared with the parent resistant isolates of carbendazim, edifenphos and iprobenfos, respectively. Similarly spore germination technique was adopted to determine ED_{50} and MIC values.

3.15 RESISTANCE MANAGEMENT STUDIES

Investigations were carried out on fungicide resistance management under both *in vitro* and *in vivo* conditions by employing those isolates that had developed highest resistance against carbendazim, edifenphos and iprobenfos, respectively. Five fungicides with different modes of action *viz.* kasugamycin, propiconazole, mancozeb, tricyclazole and BTH were used in combination with carbendazim, edifenphos and iprobenfos, respectively. The details regarding the mode of action of all the fungicides are presented in Table 4.

3.15.1 *In vitro* studies on resistance management

The carbendazim resistant isolate (Cuttak) was grown on fungicide amended oat meal agar medium containing carbendazim in combination with kasugamycin 2 L, propiconazole 25 EC, mancozeb 75 WP, tricyclazole 75 WP and BTH 50 WG while the edifenphos resistant isolate (Mandya) was grown on fungicide amended oat meal agar medium containing edifenphos in combination with kasugamycin 2 L, propiconazole 25 EC, mancozeb 75 WP, tricyclazole 75 WP and BTH 50 WG. Similarly, the iprobenfos resistant isolate (Maruteru) was grown on fungicide amended oat meal agar medium containing iprobenfos in combination with kasugamycin 2 L, propiconazole 25 EC, mancozeb 75 WP, tricyclazole 75 WP and BTH 50 WG. All the three resistant isolates were also grown on fungicide amended oat meal agar medium with kasugamycin 2 L,

Table 4: Mode of action and target sites of different fungicides employed in the Investigation

S. No.	Common Name	Group	Mode of Action	Target Site
1	Carbendazim	Benzimidazoles	Mitosis and cell division	β – tubulin assembly in mitosis
2	Azoxystrobin	Methoxyacrylates	Respiration	Complex III: cytochrome bc1 (ubiquinol oxidase) at Q_0 site
3	RIL 010	-do-	-do-	-do-
4	Pyraclostrobin	Methoxycarbamates	-do-	-do-
5	Kasugamycin	Hexopyranosyl antibiotic	Amino acids and protein synthesis	Protein syntheis
6	Edifenphos	Phosphorothiolates	Lipid and membrane synthesis	Phospholipid biosynthesis, methyl transferase
7	Iprobenfos	-do-	-do-	-do-
8	Isoprothiolane	Dithiolanes	-do-	-do-
9	Defenconazole	Triazoles	Sterol biosynthesis in membranes	C_{14} – demethylase in sterol biosynthesis
10	Diniconazole	-do-	-do-	-do-
11	Epoxiconazole	-do-	-do-	-do-
12	Propiconazole	-do-	-do-	-do-
13	Pyroquilon	Pyrroloquinolinone	Melanin biosynthesis in cell wall	Reductase in melanin biosynthesis
14	Tricyclazole	Triazolo-benzothiazole	-do-	-do-
15	Fenoxanil	Propionamide	-do-	Dehydratase in melanin biosynthesis
16	Mancozeb	Dithiocarbamates	Multisite contact activity	Multisite contact activity
17	Carbendazim + Epoxiconazole	Benzimidazoles + Triazoles	As given above	As given above
18	Difenconazole + Propiconazole	Triazoles + Triazoles	As given above	As given above
19	Acibenzolar-S-methyl	Benzo-thiadiazole BTH	Host plant defense induction	Salicylic acid path way

propiconazole 25 EC, mancozeb 75 WP, tricyclazole 75 WP and BTH 50 WG individually. These isolates were grown at various concentrations *viz.*, 0, 1, 5, 10, 25, 50, 100, 250, 500 and 1000 µg/ml of their respective fungicide by adopting poisoned food technique. Each treatment was replicated thrice. ED_{50} and MIC values were calculated. Similarly, spore germination technique was adopted to determine ED_{50} and MIC values as described earlier.

3.15.2 *In vivo* studies on resistance management

The fungicide combinations of kasugamycin 2 L, propiconazole 25 EC, mancozeb 75 WP, tricyclazole 75 WP and BTH 50 WG with carbendazim 50 WP, edifenphos 50 EC and iprobenfos 48 EC, respectively were used as foliar sprays in glass house. Bold and healthy seed of highly susceptible cultivar HR-12 was soaked in water over night. Next day, the seed was sown in the plastic trays containing sterilized soil. The trays were watered regularly using sprinklers. Fifteen day old seedlings were inoculated with Cuttak (carbendazim resistant), Mandya (edifenphos resistant) and Maruteru (iprobenfos resistant) isolates of *P. grisea* (1×10^6 conidia/ml), respectively. The plots were sprayed with fungicides immediately after the appearance of initial disease symptoms. Untreated controls were sprayed with sterile distilled water, simultaneously. The treatments were imposed in CRD replicating thrice. The per cent disease incidence was recorded when the leaf blast severity reached cent per cent in the untreated control.

Results

Chapter IV

RESULTS

The results of the investigations regarding evaluation of rice cultivars for resistance against blast disease; effect of systemic acquired disease resistance inducing chemicals on *P. grisea in vitro*; effect of systemic acquired resistance inducing chemicals on rice blast *in vivo*; efficacy of fungicides against *P. grisea in vitro*; efficacy of fungicides against rice blast *in vivo*; relative sensitivity of *P. grisea* isolates to different fungicides; Cross resistance of carbendazim, edifenphos and iprobenfos isolates to other fungicides; persistence of fungicide resistance in the resistant isolates and finally the resistance management are presented here under –

4.1 EVALUATION OF RICE CULTIVARS FOR RESISTANCE AGAINST BLAST DISEASE

Different rice cultivars were evaluated for blast disease resistance against *Pyricularia grisea* isolate of Rajendranagar during the years 2002 and 2003 by adapting Uniform Blast Nursery (UBN) pattern developed by the International Rice Research Institute (IRRI). The objective of this experiment is to know the level of resistance existing in rice cultivars against *P. grisea* and to utilize some of them for further investigation. The reaction of rice cultivars against disease challenge was recorded by employing 0-9 scale developed by IRRI. The disease score was recorded when the disease severity in the susceptible checks reached the maximum. The cultivars were categorized into 5 groups *viz.* highly resistant (0-1 score), resistant (2-3 score), moderately resistant/susceptible (4-5 score), susceptible (6-7 score) and highly susceptible (8-9 score) based on their reaction to the blast disease.

A total of 145 rice cultivars were evaluated and the results are presented in Table 5. Forty six cultivars were highly resistant, 32 were resistant, 15 were moderately resistant/susceptible, 19 were susceptible and 33 were highly susceptible. The cultivars HPR-1155, HPR-1160, HRI-126, IET-15391, Jeeragasala-2, Katuandum, PR-111, Rasi, VL-7066 and VL-97-3682 recorded 0 score with out any blast lesions. Small brown specks of pin point size or larger brown specks without sporulating center (1 scale) were observed in 36 rice cultivars *viz.* Aditya, ADT-44, Akutphon, Athira, CB-98094, Deepthi, Erenphon, GR-101, HKR-126, HPR-2002, HPR-2026, HPR-2036, IR-64, Jeeragasala-1, Jeeragasala-3, JGL-1738, Jyothi, KRH-2, Narmada, PA-6201, Phonoibi, PR-116, Punssi, PY-2, Sarloo-52, Simhapuri, Sriranga, Swarna Prabha, Tulasi, VL-3611, VL-3964, VL-4018, VL-4040, VL-7045, VL-7059 and VL-89-1190. All these varieties between 0-1 scale were classified as highly resistant to rice blast disease.

Small roundish to slightly elongated, necrotic gray spots about 1-2 mm in diameter with a distinct brown margin (2 scale) were observed in ADT-36, ASD-16, Bharani, CB-97048, CB-98004, CD-47, HPR-20086, HPR-2023, JGL-4147, KMB-81, Kothamologuku, Nagarjuna, NDR-106, NDR-80, PHB-71, Prasanna, Pusa Basumati -1, Swarnamukhi, VL-90-1656, VL-95-6389 and VSR-8. Small roundish to slightly elongated, necrotic gray spots about 1-2 mm in diameter with a distinct brown margin on the upper leaves (3 scale) were observed in Ajaya, CB-97053, DRRH-2, Govind, GR-5, HKR-120, HPR-2062, IR-62, Kanchana, Kudualian and PR-114. All these varieties with 2-3 scale were classified as resistant to rice blast disease (Fig. 2&3).

Table 5: Evaluation of rice cultivars for resistance against rice blast disease

S. No.	Name of cultivar	Disease score (0-9 scale)		
		Kharif, 2002*	*Kharif*, 2003*	Mean
Highly resistant rice cultivars (0 and 1)				
1	HPR - 1155	0.0	0.0	0.0
2	HPR - 1160	0.0	0.0	0.0
3	HRI - 126	0.0	0.0	0.0
4	IET - 15391	0.0	0.0	0.0
5	Jeeragasala - 2	0.0	0.0	0.0
6	Katuandum	0.0	0.0	0.0
7	PR - 111	0.0	0.0	0.0
8	Rasi	0.0	0.0	0.0
9	VL - 7066	0.0	0.0	0.0
10	VL - 97 - 3682	0.0	0.0	0.0
11	Aditya	1.0	1.0	1.0
12	ADT - 44	1.0	0.0	0.5
13	Akutphon	1.0	0.0	0.5
14	Athira	1.0	0.0	0.5
15	CB - 98094	2.0	0.0	1.0
16	Deepthi	1.0	0.0	0.5
17	Erenphon	0.0	1.0	0.5
18	GR - 101	1.0	0.0	0.5
19	HKR - 126	1.0	0.0	0.5
20	HPR - 2002	1.0	0.0	0.5
21	HPR - 2026	2.0	0.0	1.0
22	HPR - 2036	1.0	0.0	0.5
23	IR - 64	1.0	0.0	0.5
24	Jeeragasala - 1	1.0	0.0	0.5
25	Jeeragasala - 3	1.0	0.0	0.5
26	JGL 1738	1.0	0.0	0.5
27	Jyothi	1.0	0.0	0.5
28	KRH - 2	0.0	2.0	1.0
29	Narmada	1.0	0.0	0.5
30	PA - 6201	0.0	2.0	1.0
31	Phonoibi	2.0	0.0	1.0
32	PR - 116	1.0	1.0	1.0
33	Punssi	1.0	0.0	0.5
34	PY - 2	1.0	0.0	0.5
35	Sarloo - 52	2.0	0.0	1.0
36	Simhapuri	1.0	0.0	0.5
37	Sriranga	1.0	0.0	0.5

38	Swarna Prabha	0.0	1.0	0.5
39	Tulasi	0.0	2.0	1.0
40	VL - 3611	0.0	2.0	1.0
41	VL - 3964	0.0	1.0	0.5
42	VL - 4018	0.0	1.0	0.5
43	VL - 4040	1.0	1.0	1.0
44	VL - 7045	0.0	1.0	0.5
45	VL - 7059	1.0	1.0	1.0
46	VL - 89 - 1190	0.7	1.0	0.8
Resistant rice cultivars (2 and 3)				
47	ADT – 36	2.0	2.0	2.0
48	ASD – 16	2.3	1.0	1.7
49	Bharani	2.0	2.0	2.0
50	CB – 97048	2.0	1.0	1.5
51	CB – 98004	2.0	1.0	1.5
52	CD – 47	2.0	1.0	1.5
53	HPR – 20086	1.3	2.0	1.7
54	HPR – 2023	2.0	1.0	1.5
55	JGL – 4147	2.0	1.0	1.5
56	KMB – 81	1.0	2.0	1.5
57	Kothamologuku	2.0	2.0	2.0
58	Nagarjuna	1.0	2.0	1.5
59	NDR – 106	2.0	1.0	1.5
60	NDR - 80	2.0	2.0	2.0
61	PHB – 71	2.0	1.0	1.5
62	Prasanna	1.0	2.0	1.5
63	Pusa Basumati – 1	1.0	2.0	1.5
64	Swarnamukhi	1.0	2.0	1.5
65	VL - 90 - 1656	0.3	2.0	1.2
66	VL – 95 – 6389	1.0	1.3	1.2
67	VSR – 8	2.0	1.7	1.8
68	Ajaya	2.0	3.0	2.5
69	CB – 97053	2.0	3.0	2.5
70	DRRH – 2	2.0	3.0	2.5
71	Govind	3.0	2.0	2.5
72	GR – 5	2.0	3.0	2.5
73	HKR – 120	2.0	3.0	2.5
74	HPR – 2062	3.0	2.0	2.5
75	IR – 72	2.0	3.0	2.5
76	Kanchana	1.7	3.0	2.3
77	Kudualian	2.0	4.0	3.0
78	PR – 114	3.0	2.0	2.5

Moderately resistant/Moderately susceptible rice cultivars (4 and 5)				
79	HRI – 120	3.3	3.3	3.3
80	IET – 5656	3.0	3.3	3.2
81	KJT – 3	4.0	4.0	4.0
82	Maranel	5.0	3.0	4.0
83	Masuri	4.0	3.0	3.5
84	Phalguna	4.7	3.3	4.0
85	Swarnadhan	3.7	2.7	3.2
86	VSR - 28	5.0	3.0	4.0
87	Kharvela	6.0	4.0	5.0
88	KJT – 5	5.0	5.0	5.0
89	PR – 106	5.7	4.0	4.8
90	PR – 113	6.0	4.0	5.0
91	PR – 115	4.0	6.0	5.0
92	Triguna	5.0	5.0	5.0
93	VL - 4438	4.0	6.0	5.0
Susceptible rice cultivars (6 and 7)				
94	ADT – 41	4.3	6.0	5.2
95	ASP – 26	6.3	5.7	6.0
96	HKR – 46	5.0	5.7	5.3
97	HPR – 2040	6.0	5.0	5.5
98	IR – 36	6.0	6.0	6.0
99	JGL – 1799	6.0	6.0	6.0
100	JGL – 2671	5.3	6.0	5.7
101	JGL – 3932	6.0	6.0	6.0
102	MTU – 3226	6.0	6.0	6.0
103	ORS – 241 – 2	6.0	5.3	5.6
104	PY - 1	5.0	6.0	5.5
105	PY – 3	6.0	6.0	6.0
106	Sabita	6.0	5.0	5.5
107	Vibhava	5.0	6.0	5.5
108	VL – 90 - 1692	6.0	6.0	6.0
109	HPR – 1002	6.0	7.0	6.5
110	Nidhi	7.0	7.0	7.0
111	Sonalisa	6.0	6.7	6.3
112	Surendra	6.0	6.7	6.3
Highly susceptible rice cultivars (8 and 9)				
113	Amulya	7.7	6.7	7.2
114	BPT – 5204	7.3	7.7	7.5
115	CB – 96073	7.0	9.0	8.0

116	GEB – 24	8.0	8.0	8.0
117	HPR – 927	8.7	6.7	7.7
118	IET – 10750	8.0	7.0	7.5
119	Jaya	7.0	8.0	7.5
120	JGL – 1851	7.7	7.0	7.3
121	Lalat	8.0	7.0	7.5
122	Mandya Vijaya	7.7	7.7	7.7
123	NDLR – 8	7.0	7.3	7.2
124	Suraksha	7.0	7.7	7.3
125	SYE 14-9-8	7.0	9.0	8.0
126	Vajram	7.7	8.0	7.8
127	Vijetha – 1001	8.0	7.7	7.8
128	GR – 3	8.7	9.0	8.8
129	GR – 4	8.3	8.0	8.2
130	GR – 11	9.0	9.0	9.0
131	HR – 12	9.0	9.0	9.0
132	IR – 50	8.0	9.0	8.5
133	JGL – 3828	9.0	9.0	9.0
134	Khitish	9.0	9.0	9.0
135	KJT – 4	9.0	8.7	8.8
136	Krishnahamsa	9.0	9.0	9.0
137	ORS – 240 – 2	9.0	8.0	8.5
138	Pratibha	8.0	9.0	8.5
139	Ratna	8.0	9.0	8.5
140	RP – 3895	8.0	8.3	8.2
141	RTN – 1	8.3	8.0	8.2
142	Swarna	8.3	9.0	8.7
143	Tapaswini	9.0	7.7	8.3
144	Taraori Basmati	9.0	9.0	9.0
145	Vikramarya	8.7	7.7	8.2

* Mean of three replications

Fig. 2: Disease reaction of different rice cultivars towards *Pyricularia grisea*

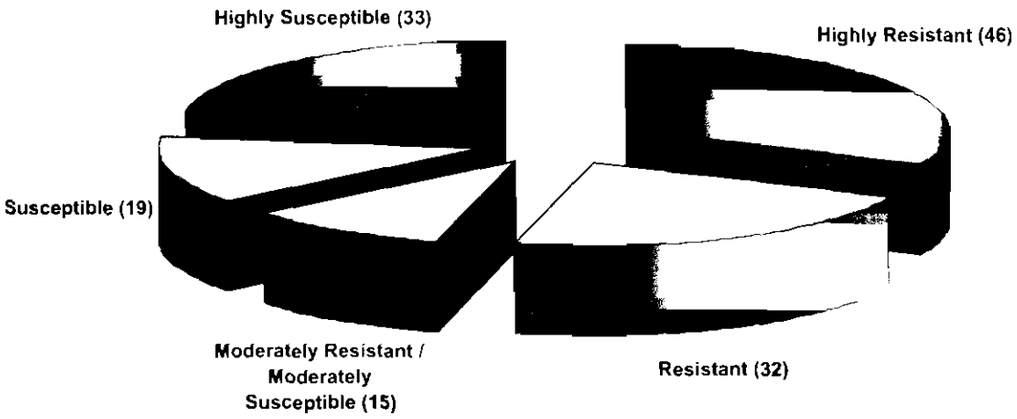
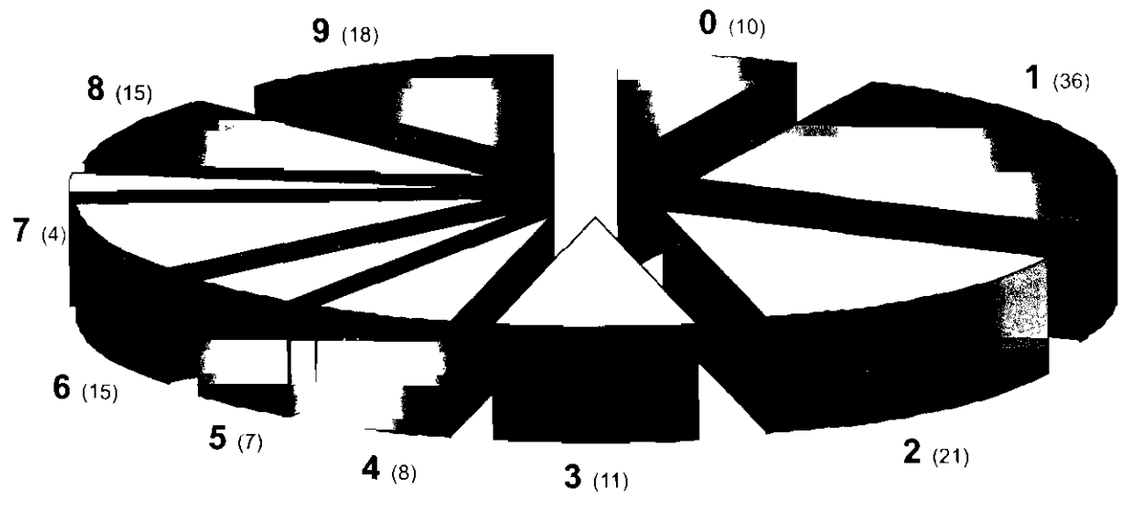


Fig. 3: Disease reaction (0-9 scale) of different rice cultivars towards *Pyricularia grisea*



Typical blast lesions of 3 mm diameter or longer infecting less than 4 per cent of leaf area (4 scale) were observed in HRI-120, IET-5656, KJT-3, Maranel, Masuri, Phalguna, Swarnadhan and VSR-28. Typical blast lesions infecting 4-10 per cent of leaf area (5 scale) were observed in Kharvela, KJT-5, PR-106, PR-113, PR-115, Triguna and VL-4438. All these varieties with 4-5 scale were classified as either moderately resistant/moderately susceptible to rice blast disease (Table 5).

Typical blast lesions infecting 11 – 25 per cent of the leaf area (6 scale) were observed in 15 rice cultivars *viz.* ADT-41, ASP-26, HKR-46, HPR-2040, IR-36, JGL-1799, JGL-2671, JGL-3932, MTU-3226, ORS-241-2, PY-1, PY-3, Sabita, Vibhava and VL-90-1692. Typical blast lesions infecting 26 – 50 per cent of the leaf area (7 scale) were observed in 4 rice cultivars *viz.* HPR-1002, Nidhi, Sonalisa and Surendra. All these varieties with 6-7 scale were classified as susceptible to rice blast disease (Fig. 2&3).

Typical blast lesions covering 51-75 per cent leaf area and leading to death of leaves (8 scale) were observed in 15 rice cultivars *viz.* Amulya, BPT-5204, CB-96073, GEB-24, HPR-927, IET-10750, Jaya, JGL-1851, Lalat, Mandya Vijaya, NDLR-8, Suraksha, SYE-14-9-8, Vajram and Vijetha-1001. Typical blast lesions infecting more than 75 per cent of the leaf area with most of the leaves dead (9 scale) were observed in GR-3, GR-4, GR-11, HR-12, IR-50, JGL-3828, Khitish, KJT-4, Krishnahamsa, ORS-240-2, Pratibha, Ratna, RP-3895, RTN-1, Swarna, Tapaswini, Taraori Basmati and Vikramarya. All these varieties with 8-9 scale were classified as highly susceptible to rice blast disease. The later group was used as susceptible check in the disease evaluation.

4.2 EFFECT OF SYSTEMIC ACQUIRED DISEASE RESISTANCE INDUCING CHEMICALS ON P. GRISEA IN VITRO

An experiment was conducted to know the inhibitory effect of systemic acquired resistance (SAR) inducing chemicals against *P. grisea* in the laboratory. Five such chemicals *viz.* salicylic acid, BTH, sodium selenite, ferric chloride and zinc sulphate were tested at different concentrations of 1 M, 10^{-1} M, 10^{-2} M, 10^{-3} M, 10^{-4} M and 10^{-5} M by employing Inhibition Zone Technique. All the five chemicals formed inhibition zones at 1 M, 10^{-1} M and 10^{-2} M while sodium selenite and ferric chloride formed inhibition zones at 10^{-3} M also. None of the chemicals formed inhibition zones at 10^{-5} M while sodium selenite alone formed inhibition zone at 10^{-4} M (Table 6).

The biggest inhibition zone was formed by sodium selenite (26.33 mm at 1 M) followed by ferric chloride (7.66 mm at 1 M) at any given concentration with an average of 8.61 and 2.33 mm, respectively. Salicylic acid and BTH formed the smallest inhibition zones with an average of 0.57 and 0.76 mm, respectively while zinc sulphate formed next smallest inhibition zone with an average of 0.81 mm. Salicylic acid, BTH and zinc sulphate were found non-toxic to *P. grisea* from 10^{-3} M, while zero inhibition of the fungus was observed from 10^{-4} M and 10^{-5} M with ferric chloride and sodium selenite, respectively (Plates 12, 13 & 14). Hence, salicylic acid, BTH and zinc sulphate were used at 10^{-3} M to induce systemic resistance, while ferric chloride and sodium selenite were used at 10^{-4} M and 10^{-5} M, respectively in the glass house experiment.

Table 6: *In vitro* efficacy of systemic acquired resistance inducing chemicals against *Pyricularia grisea*

Name of the chemical	Inhibition zone at different concentrations (mm)							Mean
	1M	10^{-1}M	10^{-2}M	10^{-3}M	10^{-4}M	10^{-5}M	Control	
Salicylic acid	2.33	1.33	0.33	0	0	0	0	0.57
BTH 50 WG	2.33	1.66	1.33	0	0	0	0	0.76
Sodium selenite	26.33	17.33	10.33	5.66	0.66	0	0	8.61
Ferric chloride	7.66	5.33	3.00	0.33	0	0	0	2.33
Zinc sulphate	3.00	2.33	0.33	0	0	0	0	0.81
Mean	8.33	5.59	3.06	1.12	0.13	0	0	

Fig. 4: *In vitro* efficacy of systemic acquired disease resistance inducing chemicals against *Pyricularia grisea*

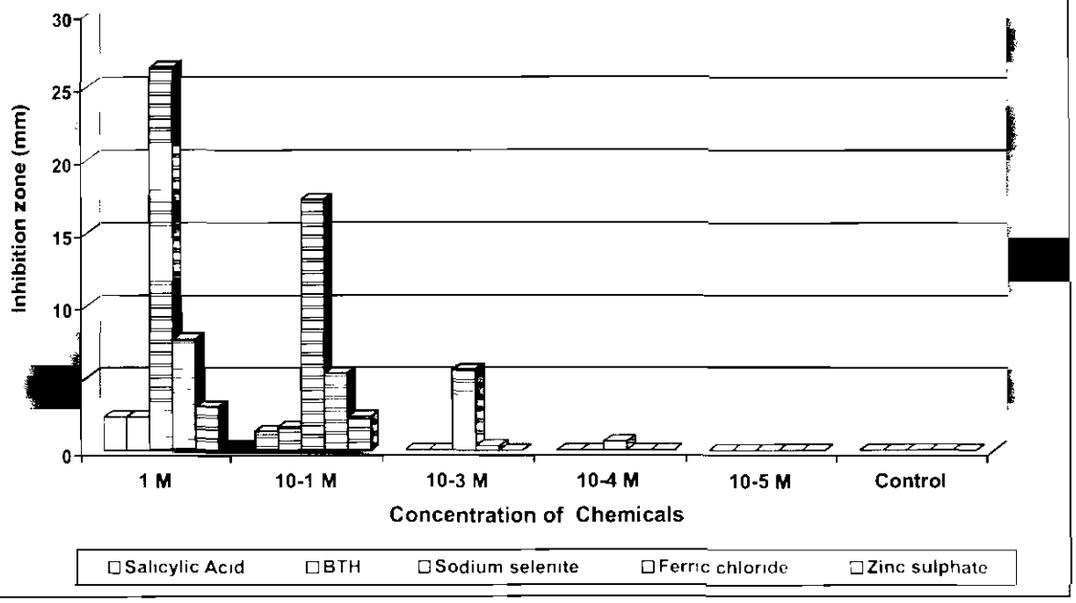
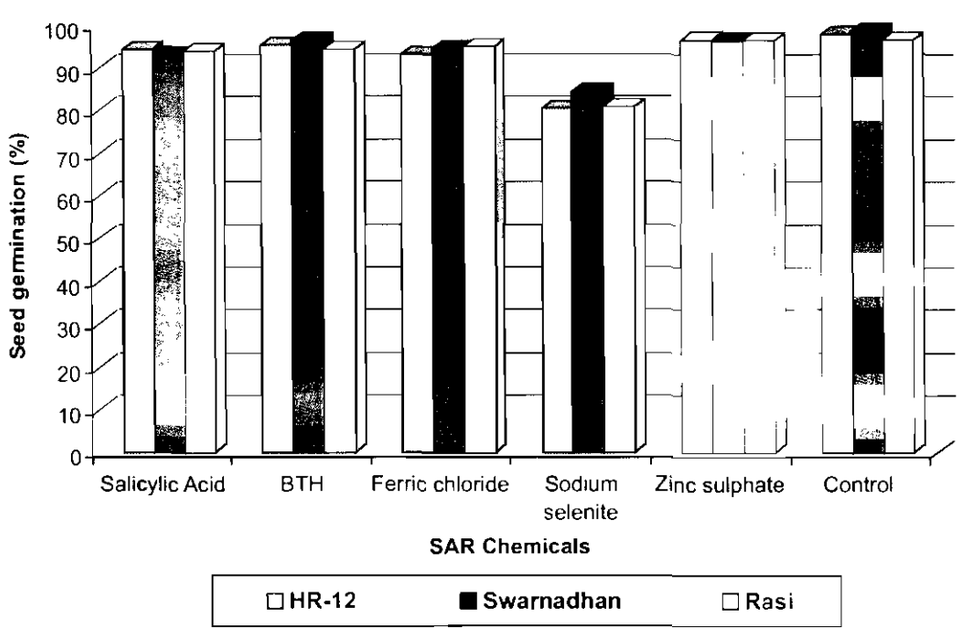


Fig. 5: Effect SAR chemicals on seed germination (%) in the varieties HR-12, Swarnadhan and Rasi (*Kharif*, 2002 & 03)



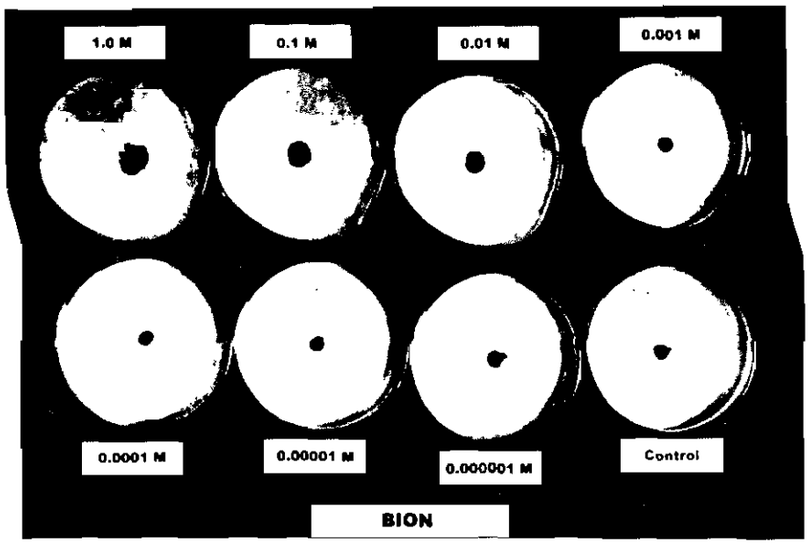


Plate 12: *In vitro* inhibition of mycelial growth of *P. grisea* by BTH

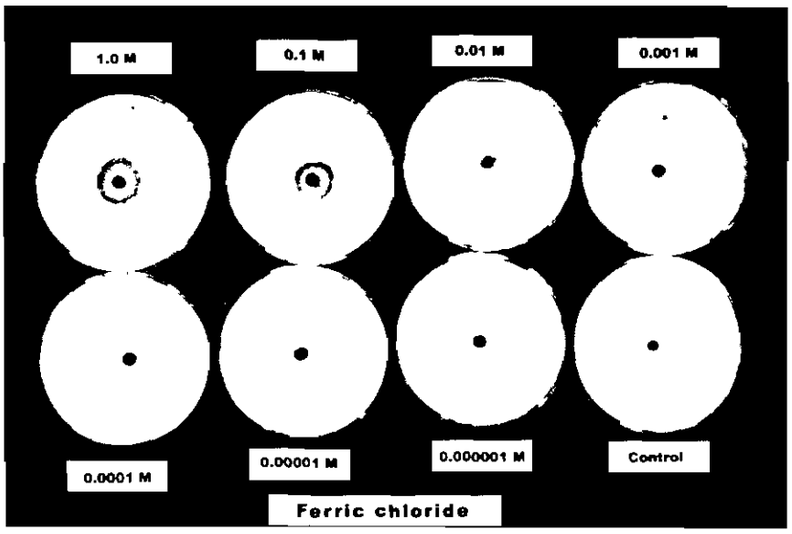


Plate 13: *In vitro* inhibition of mycelial growth of *P. grisea* by ferric chloride

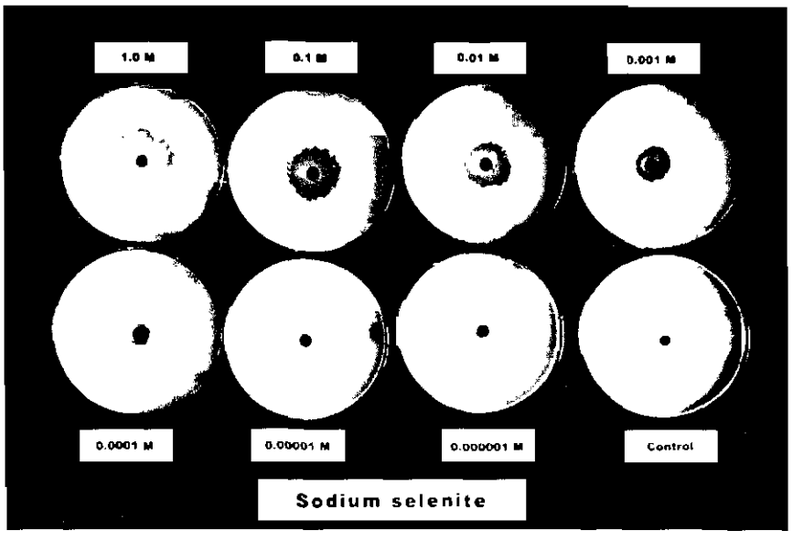


Plate 14: *In vitro* inhibition of mycelial growth of *P. grisea* by sodium selenite

4.3 EFFECT OF SYSTEMIC ACQUIRED RESISTANCE INDUCING CHEMICALS ON RICE BLAST DISEASE IN VIVO

Green house experiments were conducted to assess the effect of selected SAR inducing chemicals *viz.* salicylic acid (10^{-3} M), BTH (10^{-3} M), ferric chloride (10^{-4} M), sodium selenite (10^{-5} M) and zinc sulphate (10^{-3} M) for inducing systemic acquired resistance against blast disease in HR-12, Swarnadhan and Rasi cultivars during *Kharif*, 2002 and *Kharif*, 2003. The chemicals were applied as three kinds of treatments *viz.* seed treatment alone, seed treatment followed by one foliar spray and seed treatment followed by two foliar sprays at one week interval between the sprays. The investigations were carried out using the varieties HR-12 (susceptible), Swarnadhan (moderately resistant) and Rasi (highly resistant). Observations were recorded on seed germination, leaf blast severity, incubation period, lesion size, number of lesions/leaf and number of conidia/mm^2 of lesion. The results of *Kharif*, 2002, *Kharif*, 2003 and the pooled data of *Kharif* 2002 and 2003 are presented in Tables 7 to 15 (Plates 15 to 20).

4.3.1 Effect of SAR inducing chemicals on seed germination

The results presented in Tables 7, 8 and 9 show that except sodium selenite, none of the chemicals had given any significant reduction in seed germination. Similarly, none of the SAR inducing chemicals had shown any positive effect on seed germination as compared to the untreated control.

4.3.1.1 Effect on seed germination during *Kharif*, 2002

The results presented in Table 7 reveal that the per cent seed germination in HR-12, Swarnadhan and Rasi ranged between 77.50 & 94.08; 83.42 & 97.67 and 80.56

Table 7: Effect of systemic acquired resistance inducing chemicals on seed germination (*Kharif*, 2002)

Chemicals/Treatments	*Seed germination (%)		
	HR-12	Swarnadhan	Rasi
Salicylic acid 10^{-3}M	93.92 (76.61)	94.17 (76.19)	94.00 (76.08)
BTH 10^{-3}M	94.08 (77.21)	96.00 (80.30)	95.11 (77.57)
Ferric chloride 10^{-4}M	92.58 (75.03)	97.67 (83.42)	95.33 (77.70)
Sodium selenite 10^{-5}M	77.50 (61.81)	83.42 (66.07)	80.56 (63.95)
Zinc sulphate 10^{-3}M	93.75 (75.57)	96.25 (80.45)	96.56 (79.61)
Control	92.25 (74.14)	93.50 (75.42)	96.98 (80.23)
SEm±	1.55	1.52	1.05
CD (P=0.05)	4.39	4.30	3.00
CD (P=0.01)	5.71	5.59	3.90

*Figures in parentheses are arc sin transformed values

Table 8: Effect of systemic acquired resistance inducing chemicals on seed germination (*Kharif*, 2003)

Chemicals/Treatments	*Seed germination (%)		
	HR-12	Swarnadhan	Rasi
Salicylic acid 10^{-3}M	94.83 (77.62)	92.33 (74.04)	93.67 (75.75)
BTH 10^{-3}M	95.50 (77.99)	96.83 (80.83)	93.44 (75.25)
Ferric chloride 10^{-4}M	94.00 (76.70)	91.83 (73.46)	94.56 (76.71)
Sodium selenite 10^{-5}M	83.42 (66.07)	85.33 (67.53)	81.11 (64.28)
Zinc sulphate 10^{-3}M	94.17 (76.19)	93.50 (75.36)	96.56 (79.62)
Control	93.50 (75.42)	92.00 (73.63)	96.00 (78.55)
SEm±	1.30	0.89	0.76
CD (P=0.05)	3.69	2.54	2.18
CD (P=0.01)	4.79	3.30	2.83

*Figures in parentheses are arc sin transformed values

Table 9: Effect of systemic acquired resistance inducing chemicals on seed germination (*Kharif*, 2002 & 2003; pooled data)

Chemicals/Treatments	*Seed germination (%)		
	HR-12	Swarnadhan	Rasi
Salicylic acid 10^{-3}M	94.38 (76.70)	93.25 (74.97)	93.83 (75.79)
BTH 10^{-3}M	95.42 (77.83)	96.42 (79.37)	94.28 (76.18)
Ferric chloride 10^{-4}M	93.29 (75.27)	94.75 (76.84)	94.94 (77.09)
Sodium selenite 10^{-5}M	80.46 (63.81)	84.38 (66.75)	80.83 (64.09)
Zinc sulphate 10^{-3}M	96.71 (79.87)	96.46 (79.39)	96.56 (79.55)
Control	97.92 (82.21)	98.63 (84.28)	96.39 (79.27)
SEm±	0.79	0.64	0.69
CD (P=0.05)	2.25	1.83	1.98
CD (P=0.01)	2.92	2.38	2.57

*Figures in parentheses are arc sin transformed values

& 96.98, respectively. The per cent seed germination was significantly lower when treated with sodium selenite (10^{-5} M) i.e. 77.50, 83.42 and 80.56 in HR-12, Swarnadhan and Rasi cultivars, respectively. BTH 10^{-3} M had recorded the highest per cent seed germination (94.08%) in HR-12, but it was statistically on par with other chemicals except sodium selenite 10^{-5} M. Ferric chloride 10^{-4} M recorded the highest per cent of seed germination (97.67%) in Swarnadhan, but it was statistically on par with zinc sulphate 10^{-3} M (96.25%) and BTH 10^{-3} (96%). Untreated control had recorded the highest per cent seed germination (96.98%) in Rasi, but it was found statistically on par with zinc sulphate 10^{-3} M (96.56%), ferric chloride 10^{-4} M (95.33%) and BTH 10^{-3} M (95.11%).

4.3.1.2 Effect on seed germination during *Kharif*, 2003

The results presented in Table 8 reveal that the per cent seed germination in HR-12, Swarnadhan and Rasi ranged between 83.42 & 95.50; 85.33 & 96.83 and 81.11 & 96.56, respectively. The per cent seed germination was significantly lower when treated with sodium selenite (10^{-5} M) i.e. 83.42, 85.33 and 81.11 in HR-12, Swarnadhan and Rasi cultivars, respectively. BTH 10^{-3} M had recorded the highest per cent seed germination (95.50%) in HR-12, but it was statistically on par with other chemicals except sodium selenite 10^{-5} M. In Swarnadhan also, BTH 10^{-3} M had recorded the highest per cent seed germination (96.83%) that was significantly superior to rest of the chemicals. Zinc sulphate 10^{-3} M (93.50%), salicylic acid 10^{-3} M (92.33%), untreated control (92%), ferric chloride 10^{-4} M and BTH 10^{-3} (91.83%) were statistically on par with each other, but significantly superior to sodium selenite 10^{-5} M (85.33%) in Swarnadhan. Zinc sulphate 10^{-3} M recorded the highest per cent seed germination in Rasi

(96.56%) followed by untreated control (96%), ferric chloride 10^{-4} M (94.46%), salicylic acid 10^{-3} M (93.67%) and BTH 10^{-3} M (93.44%). Untreated control was statistically on par with both zinc sulphate as well as ferric chloride while salicylic acid and BTH were statistically on par with ferric chloride.

4.3.1.3 Effect on seed germination (*Kharif*, 2002 & 2003; pooled data)

The results presented in Table 9 reveal that the per cent seed germination in the cultivars *viz.* HR-12, Swarnadhan and Rasi ranged between 80.46 & 97.92; 84.38 & 98.63 and 80.83 & 96.56, respectively. The per cent seed germination was significantly lower when treated with sodium selenite (10^{-5} M) i.e. 80.46, 84.38 and 80.83 in HR-12, Swarnadhan and Rasi, respectively. Untreated control had recorded the highest per cent seed germination, 97.92 and 98.63 in HR-12 and Swarnadhan, respectively and it was significantly superior over rest of the chemicals. Zinc sulphate 10^{-3} M had recorded the highest per cent seed germination in Rasi (96.56%). However, it was statistically on par with untreated control (96.39%).

4.3.2 Effect of SAR inducing chemicals on leaf blast severity

Highly marked and varied responses were observed among the varieties, chemicals and treatments. The effect of SAR inducing chemicals was more pronounced in susceptible variety compared to the varieties with certain degree of resistance. The influence of SAR inducing chemicals was almost nil in the highly resistant variety, Rasi. Observations on leaf blast severity were recorded up to 20 days after inoculation with 5 day interval between each observation.

4.3.2.1 Effect of SAR inducing chemicals on leaf blast severity in HR-12

The data presented in Tables 10, 11 and 12 reveal that the chemicals in general were found significantly superior to the untreated control. BTH 10^{-3} M was superior in minimizing the leaf blast severity followed by salicylic acid 10^{-3} M. Sodium selenite 10^{-5} M and ferric chloride 10^{-4} M have shown some SAR inducing potential. However, the SAR inducing effect of zinc sulphate 10^{-3} M was negligible. The SAR inducing chemicals applied as seed treatment followed by either one foliar spray or two foliar sprays were found significantly superior to that of seed treatment alone. However, the 2nd spray did not show any significant effect in inducing any additional systemic resistance.

4.3.2.1.1 Effect on leaf blast severity in HR-12 during *Kharif*, 2002

At 5 days after inoculation (DAI), the chemicals were found significantly superior to the untreated control where the per cent leaf blast severity ranged between 2.42 and 11.92. BTH 10^{-3} M (2.42%) exhibited superiority in inducing SAR against rice blast followed by salicylic acid 10^{-3} M (3.58%), sodium selenite 10^{-5} M (4.17%), ferric chloride 10^{-4} M (5.42%) and zinc sulphate 10^{-3} M (7%) while the untreated control (11.92%) registered the highest per cent of leaf blast severity. The chemicals differed significantly with each other and also with the untreated control (Table 10).

The results presented in Table 10 reveal that the chemicals were significantly superior to the untreated control at 10 DAI where the per cent leaf blast severity varied between 13.75 and 32.42. BTH 10^{-3} M (13.75%) exhibited superiority in inducing SAR against rice blast followed by salicylic acid 10^{-3} M (18.67%), sodium

selenite 10^{-5} M (18.58%), ferric chloride 10^{-4} M (22%) and zinc sulphate 10^{-3} M (25.83%) while the untreated control (32.42%) registered the highest per cent of leaf blast severity. The chemicals differed significantly with each other and also with the untreated control except salicylic acid and sodium selenite.

At 15 DAI, the chemicals were found significantly superior to the untreated control where the per cent leaf blast severity varied between 29.00 and 67.92. BTH 10^{-3} M (29%) was the best among the chemicals in inducing SAR against rice blast followed by sodium selenite 10^{-5} M (37.17%), salicylic acid 10^{-3} M (37.92%), ferric chloride 10^{-4} M (43.83%) and zinc sulphate 10^{-3} M (57.67%) while the untreated control (67.92%) registered the highest per cent of leaf blast severity. The chemicals differed significantly with each other also with the untreated control except salicylic acid and sodium selenite (Table 10).

The chemicals were found significantly superior to the untreated control at 20 DAI where the per cent leaf blast severity ranged between 48.00 and 97.00. BTH 10^{-3} M (48%) exhibited superiority in inducing SAR against rice blast followed by salicylic acid 10^{-3} M (54.5%), sodium selenite 10^{-5} M (59.17%), ferric chloride 10^{-4} M (64.33%) and zinc sulphate 10^{-3} M (77.42%) while the untreated control (97%) registered the highest per cent of leaf blast severity. The chemicals differed significantly with each other also with the untreated control.

4.3.2.1.2 Effect on leaf blast severity in HR-12 during *Kharif*, 2003

The data presented in Table 11 show that the chemicals were significantly superior to the untreated control at 5 DAI, where the per cent leaf blast severity ranged

between 1.83 and 8.75. BTH 10^{-3} M (1.83%) exhibited superiority in inducing SAR against rice blast followed by salicylic acid 10^{-3} M (2.33%), ferric chloride 10^{-4} M (4.25%), sodium selenite 10^{-5} M (4.58%) and zinc sulphate 10^{-3} M (5.42%) while the untreated control registered the highest per cent of leaf blast severity (8.75%). However, there was no significant difference between BTH and salicylic acid while ferric chloride was statistically on par with sodium selenite.

At 10 DAI, the chemicals were significantly superior to the untreated control where the per cent leaf blast severity varied between 7.25 and 36.17. BTH 10^{-3} M (7.25%) exhibited superiority in inducing SAR against rice blast followed by salicylic acid 10^{-3} M (7.75%), ferric chloride 10^{-4} M (12.67%), sodium selenite 10^{-5} M (15.17%) and zinc sulphate 10^{-3} M (20.50%) while the untreated control (36.17%) registered the highest per cent of leaf blast severity. However, all the chemicals differed significantly with each other also with the untreated control except salicylic acid and sodium selenite.

The chemicals had shown marked and varied response at 15 days after inoculation where the per cent leaf blast severity ranged between 31.25 and 79.17. Salicylic acid 10^{-3} M (31.25%) recorded the lowest per cent leaf blast severity even though it was marginal when compared to BTH 10^{-3} M (31.5%). Ferric chloride 10^{-4} M (37.75%) was the next best chemical in the order of its efficacy followed by sodium selenite 10^{-5} M (42.5%) and zinc sulphate 10^{-3} M (45.83%). There was no significant difference between salicylic acid and BTH; ferric chloride and sodium selenite, respectively. Surprisingly, zinc sulphate was found statistically on par with sodium selenite for the first time (Table 11).

The results presented in Table 11 show that the chemicals were significantly superior to the untreated control at 20 DAI, where the per cent leaf blast severity ranged between 47.08 and 100. Salicylic acid 10^{-3} M (47.08%) exhibited superiority in inducing SAR against rice blast followed by BTH 10^{-3} M (49.17%), ferric chloride 10^{-4} M (58.33%), sodium selenite 10^{-5} M (59.58%) and zinc sulphate 10^{-3} M (67.08%) while the untreated control (100%) registered the highest per cent of leaf blast severity. However, there was no significant difference between salicylic acid and BTH while ferric chloride was statistically on par with sodium selenite.

4.3.2.1.3 Effect on leaf blast severity in HR-12 (*Kharif*, 2002 & 2003; pooled data)

At 5 days after inoculation, the chemicals were significantly superior to the untreated control where the per cent leaf blast severity ranged between 2.13 and 10.33 (Table 12). BTH 10^{-3} M (2.13%) had exhibited superiority in inducing SAR against rice blast followed by salicylic acid 10^{-3} M (2.96%), sodium selenite 10^{-5} M (4.38%), ferric chloride 10^{-4} M (4.83%) and zinc sulphate 10^{-3} M (6.21%) while the untreated control registered the highest per cent of leaf blast severity (10.33%). However, the SAR inducing chemicals differed significantly with each other and also with the control except ferric chloride and sodium selenite.

The chemicals were significantly superior to untreated control at 10 DAI, where the per cent leaf blast severity varied between 10.50 and 34.29. BTH 10^{-3} M (10.5%) exhibited superiority in inducing SAR against rice blast followed by salicylic acid 10^{-3} M (13.21%), sodium selenite 10^{-5} M (16.88%), ferric chloride 10^{-4} M (17.33%) and zinc sulphate 10^{-3} M (23.17%) while the untreated control registered the highest per

cent of leaf blast severity (34.29%). However, the chemicals differed significantly with each other also with the untreated control except ferric chloride and sodium selenite.

The results presented in Table 12 indicate same trend at 15 DAI also where the per cent leaf blast severity ranged between 30.25 and 73.54. BTH 10^{-3} M (30.25%) exhibited superiority in inducing SAR against rice blast followed by salicylic acid 10^{-3} M (34.58%), sodium selenite 10^{-5} M (39.83%), ferric chloride 10^{-4} M (40.79%) and zinc sulphate 10^{-3} M (51.75%) while the untreated control (73.54%) registered the highest per cent of leaf blast severity. The chemicals differed significantly with each other also with the untreated control except ferric chloride and sodium selenite.

At 20 DAI, the chemicals were found significantly superior to the untreated control where the per cent leaf blast severity ranged between 48.58 and 98.50. BTH 10^{-3} M (48.58%) exhibited superiority in inducing SAR against rice blast followed by salicylic acid 10^{-3} M (50.79%), sodium selenite 10^{-5} M (59.38%), ferric chloride 10^{-4} M (61.33%) and zinc sulphate 10^{-3} M (72.25%) while the untreated control (98.5%) registered the highest per cent of leaf blast severity. The chemicals differed significantly with each other and also with the untreated control except BTH and salicylic acid.

4.3.2.1.4 Effect of method and number of applications on leaf blast severity in HR-12 during *Kharif*, 2002

The per cent leaf blast severity in the treatment *viz.* seed treatment alone, seed treatment followed by one foliar spray and seed treatment followed by two foliar sprays ranged between 5.50 and 6.25; 20.63 and 23.58; 43.75 and 48.71; 64.96 and 69.79 at 5, 10, 15 and 20 days after inoculation, respectively (Table 10). The SAR chemicals applied as seed treatment alone recorded a leaf blast severity of 6.25, 20.63, 48.71 and

69.79 per cent at 5, 10, 15 and 20 days after inoculation, respectively while seed treatment followed by one foliar spray had registered a leaf blast severity of 5.50, 21.42, 44.29 and 64.96 per cent at 5, 10, 15 and 20 days after inoculation, respectively.

Seed treatment followed by two foliar sprays recorded a leaf blast severity of 5.50, 23.58, 43.75 and 65.46 per cent at 5, 10, 15 and 20 days after inoculation, respectively. However, the SAR chemicals applied as seed treatment followed by either one or two foliar sprays were significantly superior to those applied as seed treatment alone. It was clearly evident from the data presented in the Table 10 that seed treatment followed by two foliar sprays had not given any significant benefit over seed treatment followed by one foliar spray at any point of time upto 20 days after inoculation.

4.3.2.1.5 Effect of method and number of applications on leaf blast severity in HR-12 during *Kharif*, 2003

The per cent leaf blast severity in the treatment *viz.* seed treatment alone, seed treatment followed by one foliar spray and seed treatment followed by two foliar sprays ranged between 4.25 and 4.88; 14.96 and 18.00; 41.08 and 49.17; 59.38 and 68.75 at 5, 10, 15 and 20 days after inoculation, respectively (Table 11). The SAR chemicals applied as seed treatment alone recorded a leaf blast severity of 4.88, 18.00, 49.17 and 68.75 per cent at 5, 10, 15 and 20 days after inoculation, respectively while seed treatment followed by one foliar spray had registered a leaf blast severity of 4.46, 14.96, 41.08 and 62.50 per cent at 5, 10, 15 and 20 days after inoculation, respectively. Similarly, seed treatment followed by two foliar sprays recorded a leaf blast severity of 4.25, 16.79, 43.75 and 59.38 per cent at 5, 10, 15 and 20 days after inoculation, respectively. The SAR chemicals applied as seed treatment followed by either one foliar

spray or two foliar sprays were found significantly superior to those applied as seed treatment alone except at 5 DAI where seed treatment alone was found statistically on par with seed treatment followed by single foliar spray. It was clearly evident from the data presented in the Table 11 that seed treatment followed by two foliar sprays had not given any significant benefit over seed treatment followed by one foliar spray at any point of time upto 20 days after inoculation.

4.3.2.1.6 Effect of method and number of applications on leaf blast severity in HR-12 (*Kharif*, 2002 & 2003; pooled data)

The per cent leaf blast severity in the treatment *viz.* seed treatment alone, seed treatment followed by one foliar spray and seed treatment followed by two foliar sprays ranged between 4.88 and 5.56; 18.19 and 20.19; 42.69 and 48.94; 62.42 and 69.27 at 5, 10, 15 and 20 days after inoculation, respectively. The SAR chemicals applied as seed treatment alone recorded a leaf blast severity of 5.56, 19.31, 48.94 and 69.27 per cent at 5, 10, 15 and 20 days after inoculation, respectively while seed treatment followed by one foliar spray had registered a leaf blast severity of 4.98, 18.19, 42.69 and 63.73 per cent at 5, 10, 15 and 20 days after inoculation, respectively. Seed treatment followed by two foliar sprays registered a leaf blast severity of 4.88, 20.19, 43.75 and 62.42 per cent at 5, 10, 15 and 20 days after inoculation, respectively. However, the SAR chemicals applied as seed treatment followed by either one foliar spray or two foliar sprays were found significantly superior to those applied as seed treatment alone except at 10 DAI where seed treatment alone was found statistically on par with seed treatment followed by single foliar spray as well as seed treatment followed by two sprays. It was clearly evident from the data presented in Table 12 that seed treatment followed by two foliar sprays had not given any significant benefit over seed treatment followed by one foliar spray at any point of time up to 20 days after inoculation.

Table 10: Effect of systemic acquired resistance inducing chemicals on blast severity (%) in the varieties HR-12, Swarnadhan and Rasi during *Kharif*, 2002

Chemicals/Treatments	Disease severity (%) in HR-12				Disease severity (%) in Swarnadhan				Disease severity in Rasi
	5DAI	10DAI	15DAI	20DAI	5DAI	10DAI	15DAI	20DAI	
Chemicals									
Salicylic acid 10^{-3}M	3.58 (10.83)	18.67 (25.51)	37.92 (38.00)	54.50 (47.59)	0.00 (0.00)	0.83 (4.22)	1.67 (7.29)	2.17 (8.39)	
BTH 10^{-3}M	2.42 (8.84)	13.75 (21.74)	29.00 (32.53)	48.00 (43.86)	0.00 (0.00)	0.92 (4.70)	1.83 (7.69)	2.00 (8.04)	
Ferric chloride 10^{-4}M	5.42 (13.43)	22.00 (27.91)	43.83 (41.45)	64.33 (53.35)	0.00 (0.00)	1.42 (6.41)	2.25 (8.59)	2.42 (8.90)	No disease incidence
Sodium selenite 10^{-5}M	4.17 (11.71)	18.58 (25.52)	37.17 (37.53)	59.17 (50.30)	0.17 (0.96)	1.67 (7.05)	2.42 (8.90)	2.67 (9.34)	
Zinc sulphate 10^{-3}M	7.00 (15.31)	25.83 (30.47)	57.67 (49.43)	77.42 (61.67)	0.25 (1.43)	1.92 (7.89)	2.58 (9.21)	2.75 (9.51)	
Control	11.92 (20.17)	32.42 (34.69)	67.92 (55.58)	97.00 (82.21)	0.67 (3.83)	2.67 (9.36)	4.58 (12.32)	6.00 (14.18)	
SEm±	0.31	-0.54	0.71	0.92	0.56	0.68	0.32	0.29	-
CD (P=0.05)	0.87	1.53	2.01	2.62	1.60	1.93	0.92	0.84	-
Treatments									
ST alone	6.25 (14.09)	20.63 (26.70)	48.71 (44.32)	69.79 (58.03)	0.17 (0.96)	1.83 (7.48)	2.58 (9.05)	3.00 (9.71)	
ST + 1 spray	5.50 (13.56)	21.42 (27.38)	44.29 (41.65)	64.96 (55.68)	0.17 (0.96)	1.29 (5.83)	2.42 (8.78)	2.92 (9.58)	
ST + 2 sprays	5.50 (13.56)	23.58 (28.83)	43.75 (41.29)	65.46 (55.78)	0.21 (1.20)	1.58 (6.51)	2.67 (9.16)	3.08 (9.89)	
SEm±	0.22	0.38	0.50	0.65	0.40	0.48	0.23	0.21	-
CD (P=0.05)	0.62	1.08	1.42	1.85	ns	ns	ns	ns	-
Interaction									
SEm±	0.53	0.94	1.22	1.60	0.98	1.18	0.56	0.51	-
CD (P=0.05)	ns	ns	ns	ns	ns	ns	ns	ns	-

*Figures in parentheses are arc sin transformed values.

Table 11: Effect of systemic acquired resistance inducing chemicals on blast severity (%) in the varieties HR-12, Swarnadhan and Rasi during *Kharif*, 2003

Chemicals/Treatments	Disease severity (%) in HR-12				Disease severity (%) in Swarnadhan				Disease severity in Rasi
	5DAI	10DAI	15DAI	20DAI	5DAI	10DAI	15DAI	20DAI	
Chemicals									
Salicylic acid 10^{-3}M	2.33 (8.70)	7.75 (16.13)	31.25 (33.91)	47.08 (43.32)	0.00 (0.00)	0.67 (3.83)	1.67 (7.33)	1.92 (7.93)	
BTH 10^{-3}M	1.83 (7.69)	7.25 (15.56)	31.50 (34.01)	49.17 (44.60)	0.00 (0.00)	0.50 (2.87)	1.50 (6.93)	1.83 (7.73)	
Ferric chloride 10^{-4}M	4.25 (11.77)	12.67 (20.72)	37.75 (37.86)	58.33 (49.83)	0.00 (0.00)	1.08 (5.94)	2.25 (8.59)	3.08 (10.08)	No disease incidence
Sodium selenite 10^{-5}M	4.58 (12.20)	15.17 (22.72)	42.50 (40.65)	59.58 (50.58)	0.00 (0.00)	1.58 (7.17)	2.50 (9.05)	3.33 (10.47)	
Zinc sulphate 10^{-3}M	5.42 (13.42)	20.50 (26.86)	45.83 (42.58)	67.08 (55.04)	0.08 (0.48)	2.17 (8.44)	3.67 (10.96)	4.50 (12.20)	
Control	8.75 (17.16)	36.17 (36.91)	79.17 (64.15)	100.00 (90.00)	0.33 (1.91)	2.42 (8.90)	4.83 (12.65)	5.83 (13.97)	
SEm±	0.40	0.52	1.24	0.98	0.41	0.57	0.33	0.25	-
CD (P=0.05)	1.13	1.47	3.53	2.79	1.15	1.62	0.95	(0.72)	-
Treatments									
ST alone	4.88 (12.35)	18.00 (24.43)	49.17 (44.66)	68.75 (58.64)	0.08 (0.48)	1.42 (6.27)	2.58 (9.03)	3.33 (10.29)	
ST + 1 spray	4.46 (11.83)	14.96 (22.08)	41.08 (39.82)	62.50 (54.93)	0.08 (0.48)	1.29 (5.83)	2.75 (9.25)	3.33 (10.25)	
ST + 2 sprays	4.25 (11.30)	16.79 (22.96)	43.75 (42.10)	59.38 (53.11)	0.04 (0.24)	1.50 (6.45)	2.88 (9.49)	3.58 (10.65)	
SEm±	0.28	0.36	0.88	0.70	0.29	0.40	0.24	0.18	-
CD (P=0.05)	0.80	1.04	2.49	1.97	ns	ns	ns	ns	-
Interaction									-
SEm±	0.69	0.89	2.15	1.70	0.70	0.57	0.33	0.18	-
CD (P=0.05)	ns	ns	ns	ns	ns	ns	ns	ns	

*Figures in parentheses are arc sin transformed values.

Table 12: Effect of systemic acquired resistance inducing chemicals on blast severity (%) in the varieties HR-12, Swarnadhan and Rasi (*Kharif*, 2002 & 2003; pooled data)

Chemicals/Treatments	Disease severity (%) in HR-12				Disease severity (%) in Swarnadhan				Disease severity in Rasi
	5DAI	10DAI	15DAI	20DAI	5DAI	10DAI	15DAI	20DAI	
Chemicals									
Salicylic acid 10^{-3}M	2.96 (9.88)	13.21 (21.27)	34.58 (35.99)	50.79 (45.46)	0.00 (0.00)	0.75 (4.63)	1.67 (7.37)	2.04 (8.19)	No disease incidence
BTH 10^{-3}M	2.13 (8.33)	10.50 (18.90)	30.25 (33.29)	48.58 (44.20)	0.00 (0.00)	0.71 (4.33)	1.67 (7.37)	1.92 (7.94)	
Ferric chloride 10^{-4}M	4.83 (12.67)	17.33 (24.58)	40.79 (39.68)	61.33 (51.58)	0.00 (0.00)	1.25 (6.32)	2.25 (8.61)	2.75 (9.52)	
Sodium selenite 10^{-5}M	4.38 (12.00)	16.88 (24.21)	39.83 (39.10)	59.38 (50.43)	0.08 (0.68)	1.63 (7.23)	2.46 (8.99)	3.00 (9.93)	
Zinc sulphate 10^{-3}M	6.21 (14.40)	23.17 (28.71)	51.75 (46.00)	72.25 (58.23)	0.17 (1.35)	2.04 (8.20)	3.13 (10.15)	3.63 (10.96)	
Control	10.33 (18.74)	34.29 (35.82)	73.54 (59.15)	98.50 (84.52)	0.50 (3.66)	2.54 (9.15)	4.71 (12.51)	5.92 (14.08)	-
SEm±	0.23	0.39	0.69	0.76	0.38	0.41	0.24	0.20	-
CD (P=0.05)	0.65	1.12	1.95	2.16	1.08	1.17	0.68	0.58	
Treatments									
ST alone	5.56 (13.27)	19.31 (25.64)	48.94 (44.49)	69.27 (57.98)	0.13 (1.01)	1.63 (7.03)	2.58 (9.07)	3.17 (10.03)	
ST + 1 spray	4.98 (12.45)	18.19 (24.92)	42.69 (40.75)	63.73 (55.06)	0.13 (0.82)	1.29 (6.26)	2.58 (9.07)	3.13 (9.97)	
ST + 2 sprays	4.88 (12.29)	20.19 (26.19)	43.75 (41.38)	62.42 (54.16)	0.13 (1.01)	1.54 (6.64)	2.77 (9.35)	3.33 (10.31)	-
SEm±	0.16	0.28	0.49	0.54	0.27	0.29	0.17	0.14	-
CD (P=0.05)	0.46	0.79	1.38	1.53	ns	ns	ns	ns	
Interaction									-
SEm±	0.40	0.68	1.19	1.32	0.66	0.71	0.42	0.35	-
CD (P=0.05)	ns	ns	ns	ns	ns	ns	ns	ns	

*Figures in parentheses are arc sin transformed values.

Fig. 6: Effect of SAR inducing chemicals on blast severity (%) at 5 DAI in the varieties HR-12, Swarnadhan and Rasi (*Kharif*, 2002&03)

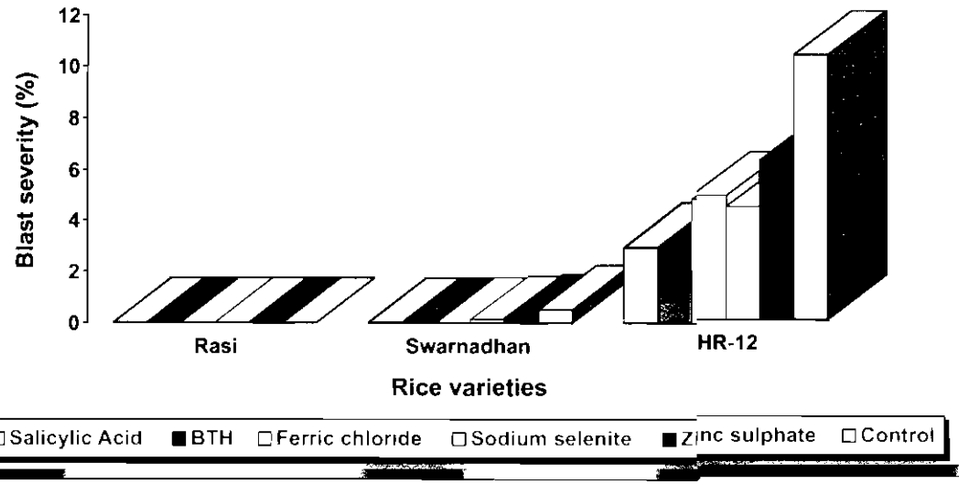


Fig. 7: Effect of SAR inducing chemicals on blast severity (%) at 10 DAI in the varieties HR-12, Swarnadhan and Rasi (*Kharif*, 2002&03)

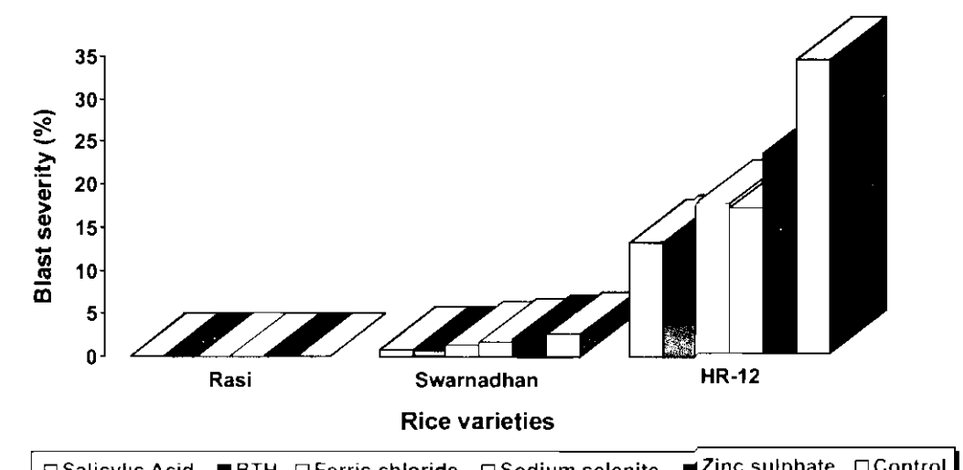


Fig. 8: Effect of SAR inducing chemicals on blast severity (%) at 15 DAI in the varieties HR-12, Swarnadhan and Rasi (*Kharif*, 2002&03)

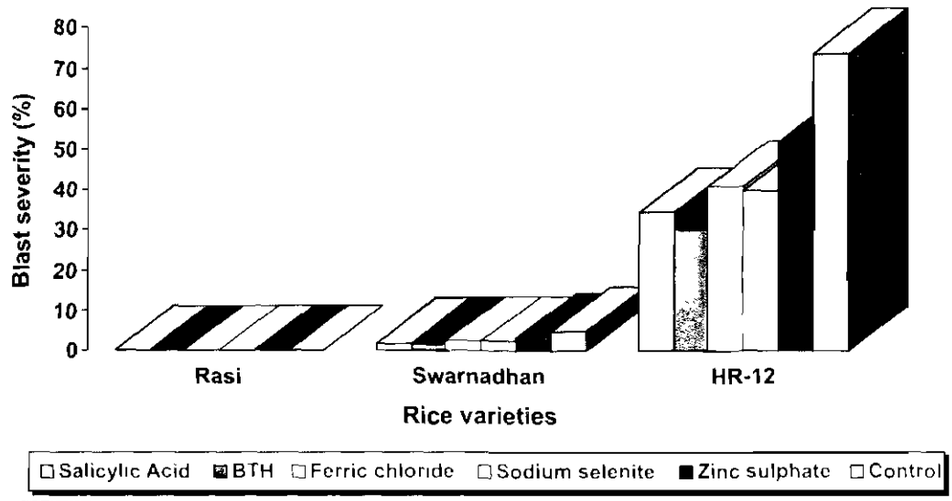


Fig. 9: Effect of SAR inducing chemicals on blast severity (%) at 20 DAI in the varieties HR-12, Swarnadhan and Rasi (*Kharif*, 2002&03)

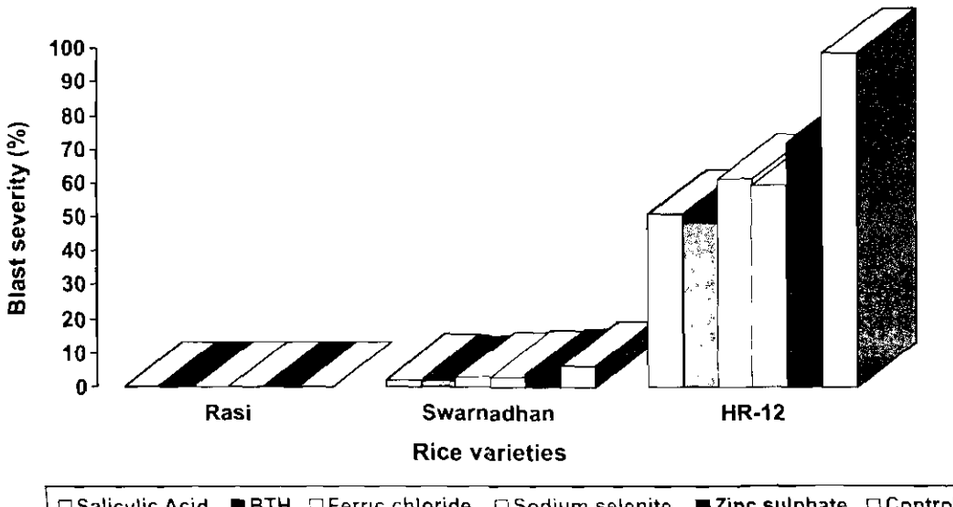




Plate 15: Evaluation of SAR inducing chemicals against rice blast in the glass house



Plate 16: Effect of BTH as seed treatment alone (1), seed treatment+one foliar spray(2) and seed treatment+two foliar sprays (3) in highly resistant variety, Rasi

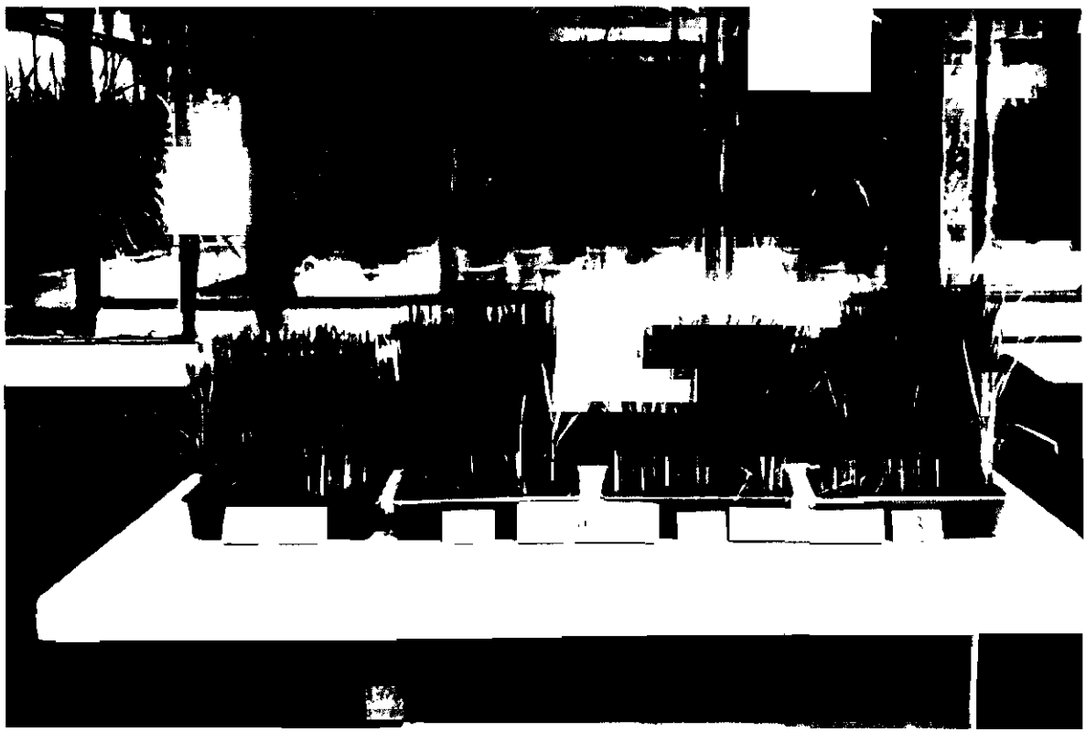


Plate 17: Effect of salicylic acid as seed treatment alone (1), seed treatment+one foliar spray (2) and seed treatment+two foliar sprays (3) in moderately resistant variety, Swarnadhan

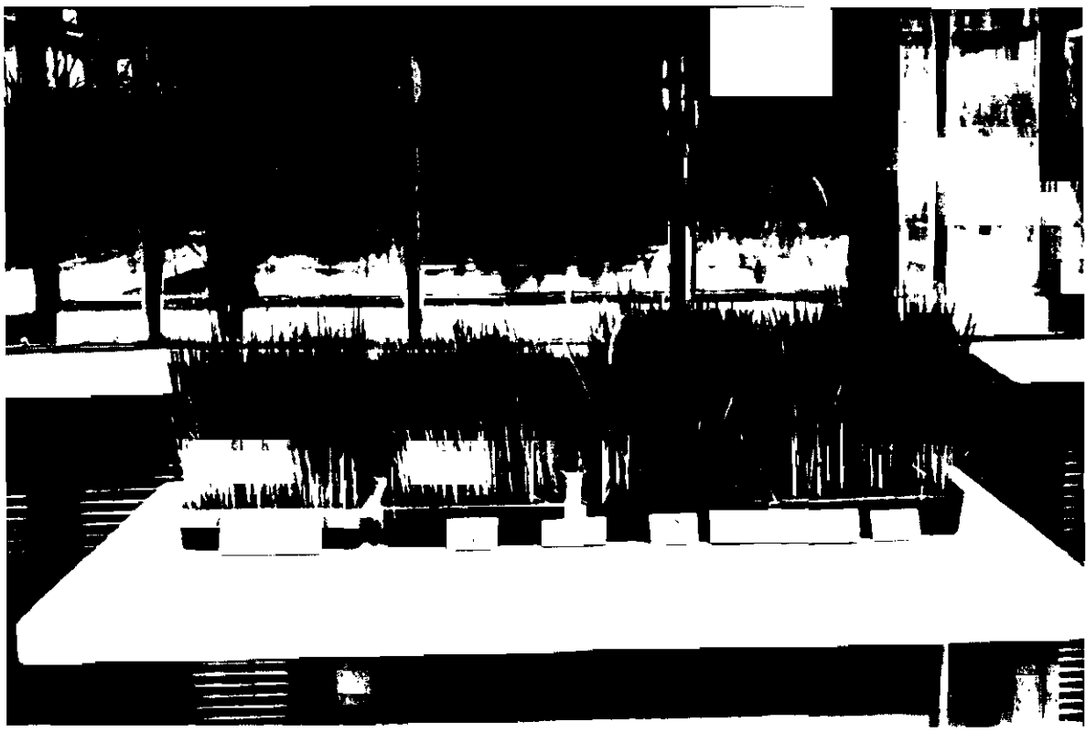


Plate 18: Effect of BTH as seed treatment alone (1), seed treatment+one foliar spray(2) and seed treatment+two foliar sprays (3) in moderately resistant variety, Swarnadhan

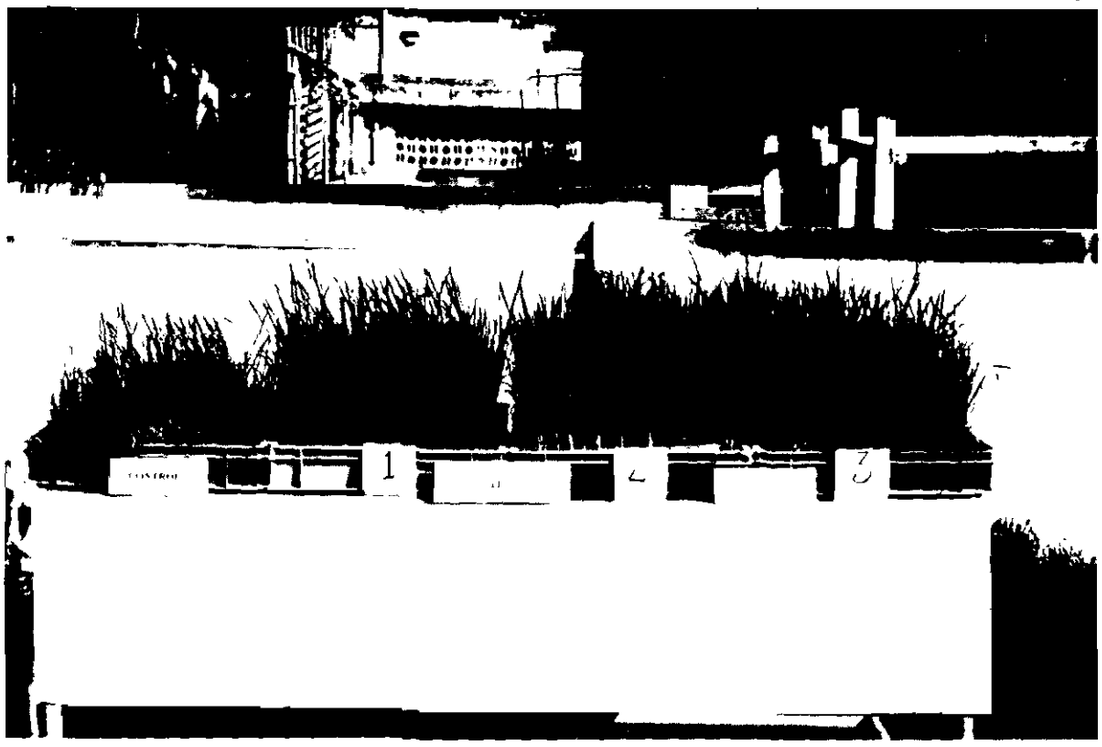


Plate 19: Effect of salicylic acid as seed treatment alone (1), seed treatment+one foliar spray (2) and seed treatment+two foliar sprays (3) in highly susceptible variety, HR-12



Plate 20: Effect of BTH as seed treatment alone (1), seed treatment+one foliar spray(2) and seed treatment+two foliar sprays (3) in highly susceptible variety, HR-12

4.3.2.1.7 Interaction effects on leaf blast severity in HR-12

The interaction effects between SAR inducing chemicals and their treatments *viz.* seed treatment alone, seed treatment followed by one spray and seed treatment followed by two foliar sprays were found non-significant at all situations.

4.3.2.2 Effect of SAR inducing chemicals on leaf blast severity in Swarnadhan

The data presented in the table 10, 11 and 12 reveal that the chemicals were significantly superior to the untreated control in inducing the systemic acquired resistance against rice blast. BTH 10^{-3} M and salicylic acid 10^{-3} M were more successful in reducing the leaf blast severity in Swarnadhan when compared to other chemicals. The effect of ferric chloride 10^{-4} M, sodium selenite 10^{-5} M and zinc sulphate 10^{-3} M was found negligible since Swarnadhan had already possessed certain degree of resistance against rice blast. The SAR inducing capabilities of the chemicals did not differ with the method and number of applications *viz.* seed treatment alone, seed treatment followed by either one or two foliar sprays.

4.3.2.2.1 Effect on leaf blast severity in Swarnadhan during *Kharif*, 2002

The data presented in Table 10 reveal that the chemicals were significantly superior to the untreated control at 5 DAI, where the per cent leaf blast severity varied between zero and 0.67. Leaf blast incidence was not observed in the plots treated with salicylic acid 10^{-3} M, BTH 10^{-3} M and ferric chloride 10^{-4} M while the untreated control recorded 0.67 per cent of leaf blast severity. Sodium selenite 10^{-5} M recorded a leaf blast severity of 0.17 per cent while zinc sulphate 10^{-3} M registered 0.25 per cent. However, all the SAR inducing chemicals were statistically on par with each other, but significantly superior to the untreated control.

The per cent leaf blast severity ranged between 0.83 and 2.67 at 10 DAI where the SAR inducing chemicals were found significantly superior to the untreated control except zinc sulphate (Table 10). Salicylic acid 10^{-3} M (0.83%) recorded the lowest leaf blast severity followed by BTH 10^{-3} M (0.92%), ferric chloride 10^{-4} M (1.42%), sodium selenite 10^{-5} M (1.67%) and zinc sulphate 10^{-3} M (1.92%) while the untreated control registered the highest per cent of leaf blast severity (2.67%). However, there was no significant difference between salicylic acid and BTH; BTH and ferric chloride, respectively. Ferric chloride, sodium selenite and zinc sulphate were statistically on par with each other, but were found significantly superior to the untreated control. There was no significant difference between zinc sulphate and untreated control.

The results presented in Table 10 show that the SAR inducing chemicals were found significantly superior to the untreated control at 15 DAI where the per cent leaf blast severity ranged between 1.67 and 4.58. Salicylic acid 10^{-3} M (1.67%) recorded the lowest per cent of leaf blast severity followed by BTH 10^{-3} M (1.83%), ferric chloride 10^{-4} M (2.25%), sodium selenite 10^{-5} M (2.42%) and zinc sulphate 10^{-3} M (2.58%) while the untreated control registered the highest per cent leaf blast severity (4.58%). However, salicylic acid was found statistically on par with BTH, but it was significantly superior to ferric chloride. Sodium selenite was found statistically on par with zinc sulphate, but it was significantly inferior to ferric chloride.

At 20 DAI, the SAR inducing chemicals were found significantly superior to the untreated control where the per cent leaf blast severity ranged between 2.17 and 6 (Table 10). BTH 10^{-3} M (2%) had recorded the lowest per cent of leaf blast severity

followed by salicylic acid 10^{-3} M (2.17%), ferric chloride 10^{-4} M (2.42%), sodium selenite 10^{-5} M (2.67%) and zinc sulphate 10^{-3} M (2.75%) while the untreated control registered the highest per cent leaf blast severity (6%). However, there was no significant difference between salicylic acid and BTH; BTH and ferric chloride, respectively. Ferric chloride, sodium selenite and zinc sulphate were statistically on par with each other, but were found significantly superior to the untreated control (Table 10).

4.3.2.2.2 Effect on leaf blast severity in Swarnadhan during *Kharif*, 2003

The data presented in Table 11 reveal that the chemicals were significantly superior to the untreated control at 5 DAI, where the per cent leaf blast severity varied between zero and 0.33. Leaf blast incidence was observed in none of the plots treated with salicylic acid 10^{-3} M, BTH 10^{-3} M, ferric chloride 10^{-4} M and sodium selenite 10^{-5} M except zinc sulphate 10^{-3} M (0.08%). But, zinc sulphate was found on par with other chemicals.

At 10 DAI, the SAR inducing chemicals were found significantly superior to the untreated control except zinc sulphate where the per cent leaf blast severity ranged between 0.50 and 2.42 (Table 11). BTH 10^{-3} M (0.5%) recorded the lowest per cent of leaf blast severity followed by salicylic acid 10^{-3} M (0.67%), ferric chloride 10^{-3} M (1.08%), sodium selenite 10^{-5} M (1.58%) and zinc sulphate 10^{-3} M (2.17%) while the untreated control registered the highest per cent of leaf blast severity (2.42%). There was no significant difference between salicylic acid and BTH while sodium selenite was statistically on par with zinc sulphate. However, zinc sulphate was statistically on par with the untreated control.

The chemicals were found significantly superior to the untreated control at 15 DAI where the per cent leaf blast severity ranged between 1.50 and 4.83. BTH 10^{-3} M (1.5%) recorded the lowest per cent of leaf blast severity followed by salicylic acid 10^{-3} M (1.67%), ferric chloride 10^{-4} M (2.25%), sodium selenite 10^{-5} M (2.5%) and zinc sulphate 10^{-3} M (3.67%) while the untreated control registered the highest per cent leaf blast severity (4.83%). Salicylic acid was statistically on par with BTH while there was no significant difference between ferric chloride and sodium selenite. Zinc sulphate was significantly superior to the untreated control, but inferior to sodium selenite and ferric chloride.

At 20 DAI, the chemicals followed the same trend as at 15 DAI where the per cent leaf blast severity ranged between 1.83 and 5.83. BTH 10^{-3} M (1.83%) recorded the lowest per cent leaf blast severity followed by salicylic acid 10^{-3} M (1.92%), ferric chloride 10^{-4} M (3.08%), sodium selenite 10^{-5} M (3.33%) and zinc sulphate 10^{-3} M (4.5%) while the untreated control registered the highest per cent of leaf blast severity (5.83%). However, salicylic acid was statistically on par with BTH while there was no significant difference between ferric chloride and sodium selenite. Zinc sulphate was significantly superior to the untreated control, but inferior to sodium selenite and ferric chloride (Table 11).

4.3.2.2.3 Effect on leaf blast severity in Swarnadhan (*Kharif*, 2002 & 2003; pooled data)

The data presented in Table 12 reveal that the chemicals were significantly superior to the untreated control at 5 DAI, where the per cent leaf blast severity varied between zero and 0.67. Leaf blast incidence was not observed in the plots treated with salicylic acid 10^{-3} M, BTH 10^{-3} M and ferric chloride 10^{-4} M while the untreated control

recorded 0.50 per cent leaf blast severity. Sodium selenite 10^{-5} M recoded a leaf blast severity of 0.08 per cent while zinc sulphate 10^{-3} M registered 0.17 per cent. The SAR inducing chemicals were statistically on par with each other except zinc sulphate.

At 10 DAI, the SAR inducing chemicals were found significantly superior to the untreated control except zinc sulphate where the per cent leaf blast severity ranged between 0.71 and 2.54 (Table 12). BTH 10^{-3} M (0.71%) recorded the lowest per cent of leaf blast severity followed by salicylic acid 10^{-3} M (0.75%), ferric chloride 10^{-4} M (1.25%), sodium selenite 10^{-5} M (1.63%) and zinc sulphate 10^{-3} M (2.04%) while the untreated control registered the highest per cent of leaf blast severity (2.54%). However, there was no significant difference between BTH and salicylic acid while sodium selenite was statistically on par with ferric chloride. Zinc sulphate was statistically on par with untreated control as well as sodium selenite.

The results presented in Table 12 show that the SAR inducing chemicals were significantly superior to the untreated control at 15 DAI, where the per cent leaf blast severity ranged between 1.67 and 4.71. BTH 10^{-3} M (1.67%) recorded the lowest per cent of leaf blast severity followed by salicylic acid 10^{-3} M (1.67%), ferric chloride 10^{-4} M (2.25%), sodium selenite 10^{-5} M (2.46%) and zinc sulphate 10^{-3} M (3.13%) while the untreated control registered the highest per cent of leaf blast severity (4.71%). Salicylic acid was statistically on par with BTH while there was no significant difference between ferric chloride and sodium selenite. Zinc sulphate was significantly superior to the untreated control, but it was inferior to sodium selenite and ferric chloride.

At 20 DAI, the SAR inducing chemicals followed the same trend as at 15 DAI where the per cent leaf blast severity ranged between 1.92 and 5.92. BTH 10^{-3} M

(1.92%) recorded the lowest per cent of leaf blast severity followed by salicylic acid 10^{-3} M (2.04%), ferric chloride 10^{-4} M (2.75%), sodium selenite 10^{-5} M (3%) and zinc sulphate 10^{-3} M (3.63%) while the untreated control registered the highest per cent of leaf blast severity (5.92%). However, salicylic acid was statistically on par with BTH while there was no significant difference between ferric chloride and sodium selenite. Zinc sulphate was significantly superior to the untreated control, but was inferior to sodium selenite and ferric chloride.

4.3.2.2.4 Effect of method and number of applications on leaf blast severity in Swarnadhan during *Kharif*, 2002

The SAR inducing capabilities of the chemicals did not differ with method and number of applications *viz.* seed treatment alone, seed treatment followed by either one or two foliar sprays. The interaction effects between SAR inducing chemicals and their treatments were found non-significant at all situations (Table 10).

4.3.2.2.5 Effect of method and number of applications on leaf blast severity in Swarnadhan during *Kharif*, 2003

The SAR inducing capabilities of the chemicals did not differ with the method and number of applications *viz.* seed treatment alone, seed treatment followed by either one or two foliar sprays. The interaction effects between SAR inducing chemicals and their treatments were found non-significant at all situations (Table 11).

4.3.2.2.6 Effect of method and number of applications on leaf blast severity in Swarnadhan (*Kharif*, 2002 & 2003; pooled data)

The SAR inducing capabilities of the chemicals did not differ with the method and number of applications *viz.* seed treatment alone, seed treatment followed by

Fig. 10: Effect of SAR inducing chemicals as different treatments at 5 DAI on the varieties HR-12, Swarnadhan and Rasi (*Kharif*, 2002&03)

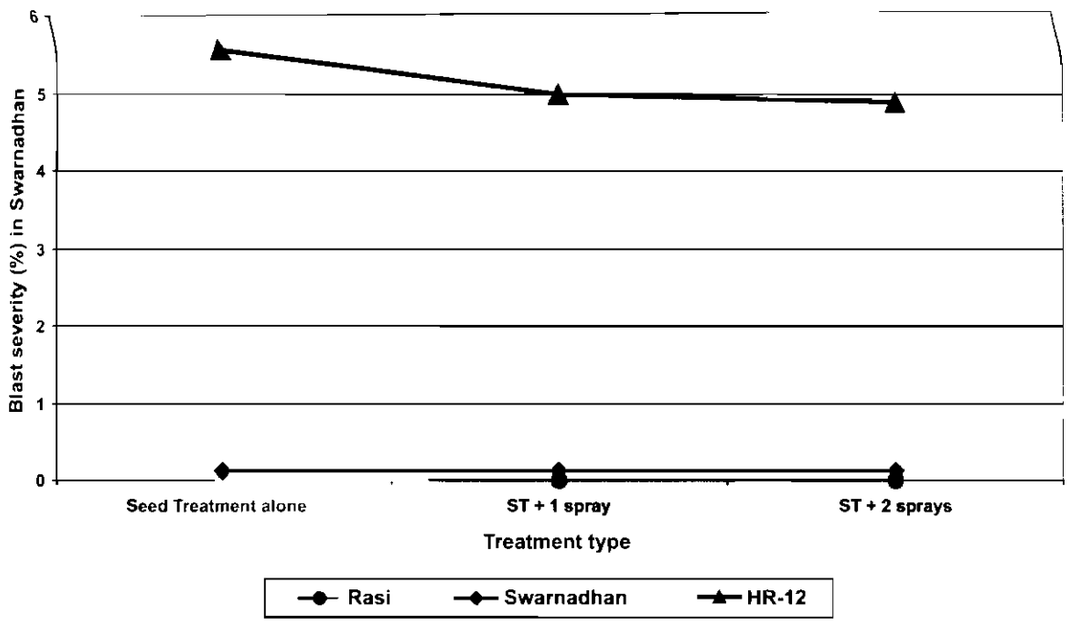


Fig. 11: Effect of SAR inducing chemicals as different treatments at 10 DAI on the varieties HR-12, Swarnadhan and Rasi (*Kharif*, 2002&03)

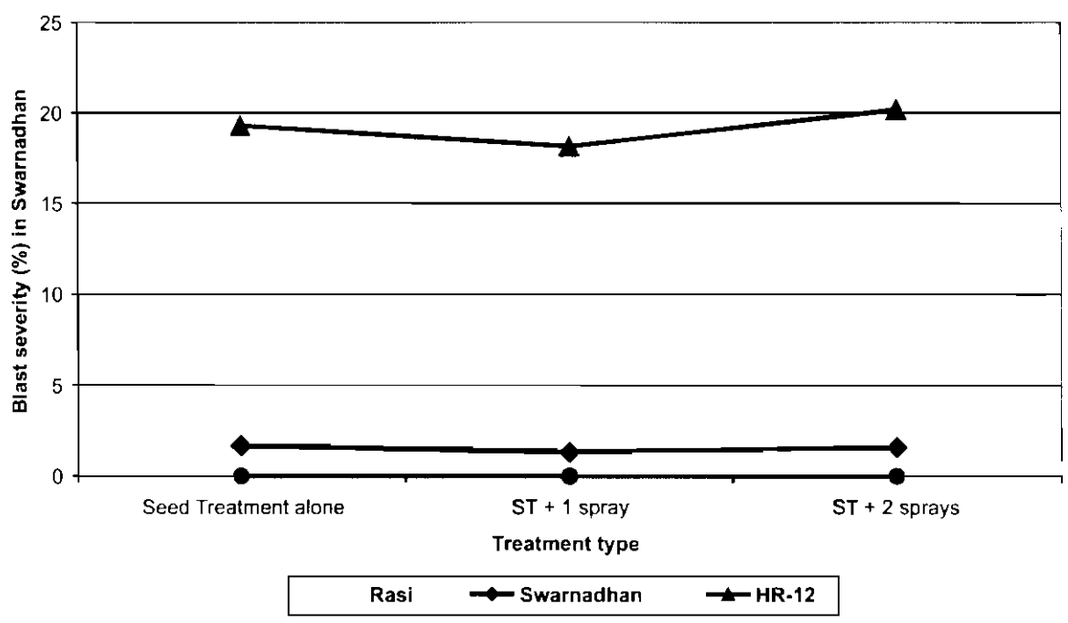


Fig. 12: Effect of SAR inducing chemicals as different treatments at 15 DAI on the varieties HR-12, Swarnadhan and Rasi (*Kharif*, 2002&03)

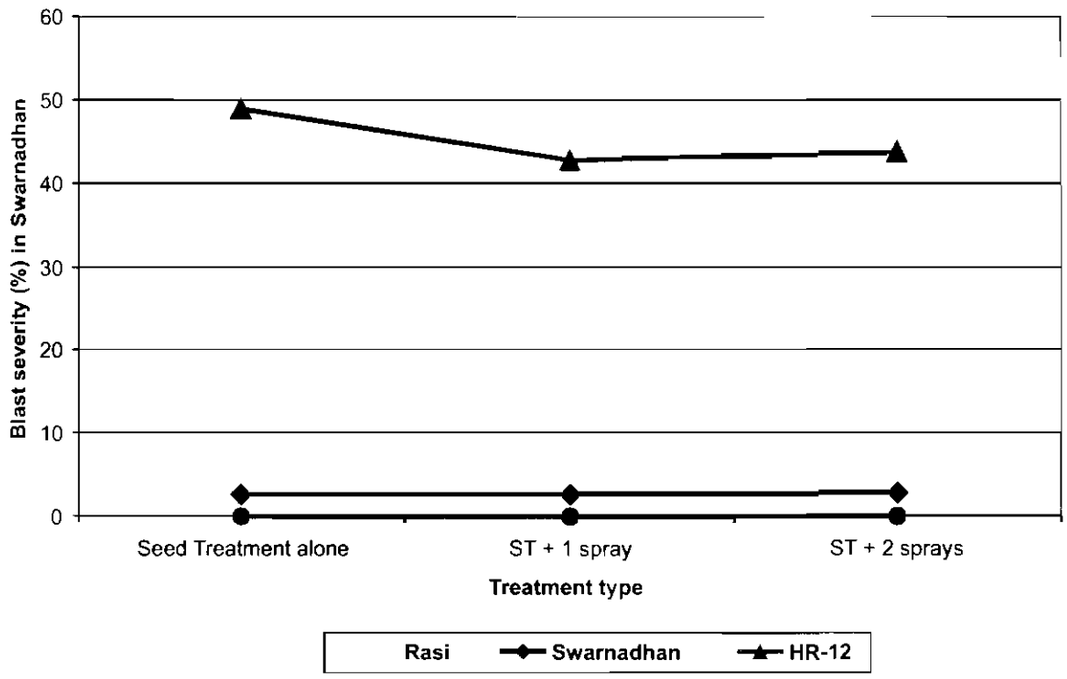
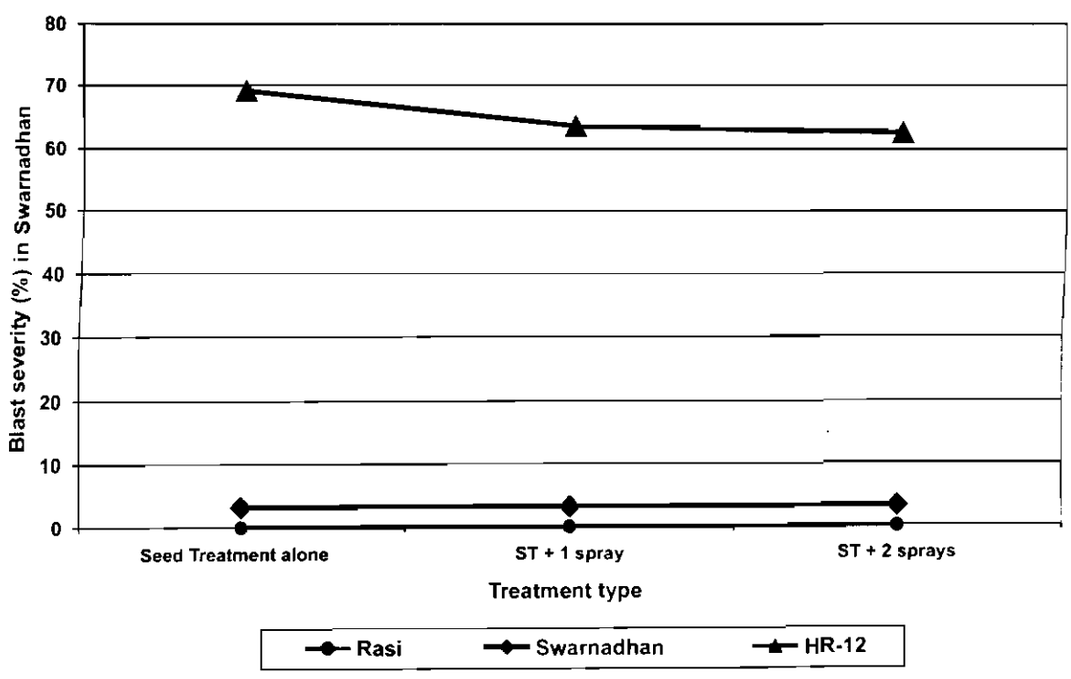


Fig. 13: Effect of SAR inducing chemicals as different treatments at 20 DAI on the varieties HR-12, Swarnadhan and Rasi (*Kharif*, 2002&03)



either one or two foliar spray, while the interaction effects between SAR inducing chemicals and their treatments were found non-significant at all situations (Table 12).

4.3.2.3 Effect of SAR inducing chemicals in the highly resistant variety Rasi

Disease incidence was observed neither in untreated control nor in chemical treatments as for as the Rasi variety was concerned (Tables 10, 11 & 12).

4.3.3 Effect of SAR inducing chemicals on different disease parameters

The effect of SAR inducing chemicals on different disease parameters *viz.* incubation period, lesion size, number of lesions per leaf and number of conidia per square millimeter was evaluated in HR-12, Swarnadhan and Rasi during *Kharif,* 2002 and 2003. The results are presented in Tables 13, 14 and 15.

4.3.3.1 Effect on incubation period during *Kharif*, 2002

The results presented in Table 13 show that the minimum number of days required for the appearance of initial disease symptoms were 3.08 days in HR-12 and 4.92 days in Swarnadhan, while there was no disease incidence in Rasi. The incubation period ranged between 3.08 days (untreated control) and 3.50 days (salicylic acid 10^{-3} M) in HR-12. There was no significant difference between untreated control and the SAR chemicals in HR-12. The incubation period ranged between 4.92 days (untreated control) and 9.17 days (salicylic acid 10^{-3} M) in Swarnadhan. The SAR inducing chemicals significantly delayed the appearance of initial disease symptoms when compared to untreated control in Swarnadhan. Salicylic acid 10^{-3} M (9.17 days) and BTH 10^{-3} M (8.92 days) have shown significantly superior performance over rest of the chemicals *viz.* ferric chloride 10^{-4} M (7.83 days), sodium selenite 10^{-5} M (7.17 days) and zinc sulphate 10^{-3} M

(6.25 days). There was no significant difference between BTH 10^{-3} M and salicylic acid 10^{-3} M while sodium selenite 10^{-5} M was statistically on par with both ferric chloride10^{-4} M and zinc sulphate 10^{-3} M.

Seed treatment alone with SAR inducing chemicals (3.54 days) was found significantly superior when compared to seed treatment followed by either one foliar spray (3.13 days) or two foliar sprays (3.13 days) in HR-12. Conversely, there was no significant difference between seed treatment alone (6.92 days) and seed treatment followed by either single foliar spray (7.79 days) or two foliar sprays (7.42 days) in Swarnadhan. The interaction effects of different SAR inducing chemicals and the number of applications were found insignificant.

4.3.3.2 Effect on lesion size during *Kharif*, 2002

The results presented in Table 13 reveal that the SAR inducing chemicals significantly reduced the lesion size over the untreated control in both HR-12 and Swarnadhan while there was no disease incidence in Rasi. In HR-12, the lesion size ranged between 3.43 mm^2 (salicylic acid 10^{-3} M) and 6.10 mm^2 (untreated control). BTH 10^{-3} M (3.77 mm^2) was found on par with both salicylic acid 10^{-3} M (3.43 mm^2) as well as ferric chloride 10^{-4} M (3.95 mm^2), but it was significantly superior to sodium selenite 10^{-5} M (5.13 mm^2) and zinc sulphate 10^{-3} M (5.45 mm^2). In Swarnadhan, the lesion size ranged between 1.81 mm^2 (BTH 10^{-3} M) and 3.79 mm^2 (untreated control). Salicylic acid 10^{-3} M (2.02 mm^2) was found on par with both BTH 10^{-3} M (1.81 mm^2) and ferric chloride 10^{-4} M (2.25 mm^2), but it was significantly superior to sodium selenite 10^{-5} M (3.04 mm^2) and zinc sulphate 10^{-3} M (3.15 mm^2).

Table 13: Effect of systemic acquired resistance inducing chemicals in the varieties HR-12, Swarnadhan and Rasi on different disease parameters during *Kharif*, 2002

Chemicals/Treatments	Incubation period (days)			Lesion size (mm^2)			No. of lesions per leaf*			No. of conidia/mm^2 of lesion*		
	HR-12	Swarna-dhan	Rasi	HR-12	Swarna-dhan	Rasi	HR-12	Swarna-dhan	Rasi	HR-12	Swarna-dhan	Rasi
Chemicals												
Salicylic acid 10^{-3}M	3.50	9.17	No incidence	3.43	2.02	No incidence	7.42 (2.89)	2.92 (1.97)	No incidence	1364.42 (36.91)	852.42 (29.19)	No incidence
BTH 10^{-3}M	3.42	8.92	-do-	3.77	1.81	-do-	7.58 (2.92)	3.00 (1.98)	-do-	1352.67 (36.73)	817.25 (28.59)	-do-
Ferric chloride 10^{-4}M	3.08	7.83	-do-	3.95	2.25	-do-	9.25 (3.19)	3.17 (2.01)	-do-	1356.17 (36.77)	910.42 (30.18)	-do-
Sodium selenite 10^{-5}M	3.25	7.17	-do-	5.13	3.04	-do-	8.25 (3.03)	6.25 (2.69)	-do-	1700.50 (41.22)	1004.58 (31.70)	-do-
Zinc sulphate 10^{-3}M	3.25	6.25	-do-	5.45	3.15	-do-	10.00 (3.31)	6.92 (2.81)	-do-	1737.33 (41.68)	1012.17 (31.82)	-do-
Control	3.08	4.92	-do-	6.10	3.79	-do-	12.17 (3.63)	8.75 (3.11)	-do-	1894.67 (43.52)	1229.67 (35.07)	
SEm±	0.13	0.35		0.15	0.15		0.06	0.07		0.49	0.28	
CD (P=0.05)	ns	1.00		0.43	0.42		0.18	0.20		1.40	0.78	
Treatments												
ST alone	3.54	6.92	No incidence	4.99	2.74	No incidence	9.71 (3.26)	6.21 (2.62)	No incidence	1626.46 (40.25)	1006.46 (31.67)	No incidence
ST + 1 spray	3.13	7.79	-do-	4.46	2.60	-do-	8.88 (3.12)	5.00 (2.36)	-do-	1561.38 (39.36)	948.88 (30.73)	-do-
ST + 2 sprays	3.13	7.42	-do-	4.47	2.69	-do-	8.75 (3.11)	5.58 (2.50)	-do-	1515.04 (38.79)	957.92 (30.87)	-do-
SEm±	0.10	0.25		0.11	0.10		0.04	0.05		0.35	0.20	
CD (P=0.05)	0.27	ns		0.30	ns		0.13	0.14		0.99	0.55	
Interaction												
SEm±	0.23	0.61		0.26	0.25		0.11	0.12		0.85	0.48	
CD (P=0.05)	ns	ns		ns	ns		ns	ns		ns	ns	

*Figures in parentheses are square route transformed values

Seed treatment with SAR inducing chemicals followed by either one foliar spray (4.46 mm^2) or two foliar sprays (4.47 mm^2) produced significantly smaller lesions as compared to seed treatment alone (4.99 mm^2) in HR-12. Conversely, there was no significant difference between seed treatment alone (2.74 mm^2) and seed treatment followed by either one foliar spray (2.60 mm^2) or two foliar sprays (2.69 mm^2). The interaction effects of different SAR inducing chemicals and the number of applications were found insignificant (Table 13).

4.3.3.3 Effect on number of lesions/leaf during *Kharif*, 2002

The results presented in Table 13 show that the SAR inducing chemicals significantly reduced the number of lesions per leaf over the untreated control in both HR-12 and Swarnadhan while there was no disease incidence in Rasi. In HR-12, the number of lesions ranged between 7.42 (salicylic acid 10^{-3} M) and 12.17 (untreated control). Salicylic acid 10^{-3} M (7.42 lesions/leaf) was found statistically on par with BTH 10^{-3} M (7.58 lesions/leaf), but has shown significantly superior effect over rest of the chemicals *viz.* sodium selenite 10^{-5} M (8.25 lesions/leaf), ferric chloride 10^{-4} M (9.25 lesions/leaf) and zinc sulphate 10^{-3} M (10 lesions/leaf). Zinc sulphate was statistically on par with ferric chloride, but it was significantly inferior to sodium selenite. In Swarnadhan, the number of lesions per leaf ranged between 2.92 (salicylic acid 10^{-3} M) and 8.75 (untreated control). Salicylic acid 10^{-3} M (2.92 lesions/leaf) was found statistically on par with BTH 10^{-3} M (3 lesions/leaf) and ferric chloride 10^{-4} M (3.17 lesions/leaf), but it was significantly superior to sodium selenite 10^{-5} M (6.25 lesions/leaf) and zinc sulphate 10^{-3} M (6.92 lesions/leaf).

Seed treatment followed two foliar sprays produced the lowest number of lesions per leaf (8.75) in HR-12, but it was found statistically on par with seed treatment followed by one foliar spray (8.88 lesions/leaf) and significantly superior to seed treatment alone (9.71 lesions/leaf). Conversely, seed treatment alone (6.21 lesions/leaf) was found statistically on par with seed treatment followed by two foliar sprays (5.58 lesions/leaf), but it was significantly inferior to seed treatment followed one foliar spray (5 lesions/leaf). However, the interaction effects of SAR inducing chemicals and the number of applications were found insignificant (Table 13).

4.3.3.4 Effect on number of conidia/mm^2 of lesion during *Kharif*, 2002

The results presented in Table 13 reveal that the SAR inducing chemicals were effective in reducing the number of conidia per square mm of lesion when compared to the untreated control in both HR-12 and Swarnadhan while there was no disease incidence in Rasi. The number of conidia per mm^2 of lesion ranged between 1352.67 (BTH 10^{-3} M) and 1894.67 (untreated control) in HR-12. Salicylic acid 10^{-3} M (1364.42 conidia/mm^2), BTH 10^{-3} M (1352.67 conidia/mm^2) and ferric chloride 10^{-4} M (1356.17 conidia/mm^2) were found statistically on par with each other, but were found significantly superior to sodium selenite 10^{-5} M (1700.50 conidia/mm^2) and zinc sulphate 10^{-3} M (1737.33 conidia/mm^2) in reducing the number of conidia per mm^2 of lesion in HR-12. The number of conidia per mm^2 of lesion ranged between 817.25 (BTH 10^{-3} M) and 1229.67 (untreated control) in Swarnadhan. There was no significant difference between Salicylic acid 10^{-3} M (817.25 conidia/mm^2) and BTH 10^{-3} M (852.42 conidia/mm^2), but these two were significantly superior to rest of the chemicals. Sodium selenite 10^{-5} M (1004.58 conidia/mm^2) and zinc sulphate 10^{-3} M (1229.67 conidia/mm^2) were statistically on par with each other but significantly inferior to ferric chloride 10^{-4} M (910.42 conidia/mm^2).

Seed treatment followed by one foliar spray produced 1515.04 conidia/mm^2 of lesion and seed treatment followed by two foliar sprays produced 1561.38 conidia/mm^2 of lesion while seed treatment alone had produced 1626.46 conidia/mm^2 in HR-12. Seed treatment followed by either one foliar spray or two foliar sprays was found significantly superior to seed treatment alone. The same trend was reflected in the variety Swarnadhan also where seed treatment alone and seed treatment followed with either one or two sprays produced 1006.46, 948.88 and 957.92 conidia/mm^2, respectively. The interaction effects of SAR inducing chemicals and the number of applications were found insignificant.

4.3.3.5 Effect on incubation period during *Kharif*, 2003

The results presented in Table 14 show that the minimum number of days required for the appearance of initial disease symptoms were 3.17 days in HR-12 and 5.67 days in Swarnadhan, while there was no disease incidence in Rasi. The incubation period ranged between 3.17 days (untreated control) and 3.67 days (both salicylic acid 10^{-3} M and BTH 10^{-3} M) in HR-12. Salicylic acid and BTH were significantly superior to other chemicals as well as the untreated control, though they were statistically on par with one another. The SAR inducing chemicals significantly delayed the appearance of initial disease symptoms in Swarnadhan when compared to untreated control. The incubation period ranged between 5.67 days (untreated control) and 10 days (BTH 10^{-3} M) in Swarnadhan. BTH 10^{-3} M (10 days) has shown significantly superior performance followed by salicylic acid 10^{-3} M (9.25 days), ferric chloride 10^{-4} M (7.92 days), sodium selenite 10^{-5} M (7.42 days) and zinc sulphate 10^{-3} M (6.58 days) over untreated control (5.67 days). There was no significant difference between sodium selenite 10^{-5} M and ferric chloride10^{-4} M while other chemicals significantly differed with one another and also with untreated control.

Seed treatment alone with SAR inducing chemicals (3.63 days) was significantly superior when compared to seed treatment followed by either one foliar spray (3.21 days) or two foliar sprays (3.17 days) in HR-12. Conversely, there was no significant difference between seed treatment alone (7.75 days) and seed treatment followed by either single foliar spray (7.96 days) or two foliar sprays (7.71 days) in Swarnadhan. The interaction effects of different SAR inducing chemicals and the number of applications were found insignificant (Table 14).

4.3.3.6 Effect on lesion size during *Kharif*, 2003

The results presented in Table 14 reveal that the SAR inducing chemicals significantly reduced the lesion size over the untreated control in both HR-12 and Swarnadhan while there was no disease incidence in Rasi. In HR-12, the lesion size ranged between 2.98 mm^2 (BTH 10^{-3} M) and 5.65 mm^2 (untreated control). BTH 10^{-3} M (2.98 mm^2) was found statistically on par with salicylic acid 10^{-3} M (3.28 mm^2), but it was significantly superior to sodium selenite 10^{-5} M (3.83 mm^2), ferric chloride 10^{-4} M (3.88 mm^2) and zinc sulphate 10^{-3} M (4.40 mm^2). In Swarnadhan, the lesion size ranged between 2.08 mm^2 (BTH 10^{-3} M) and 5 mm^2 (untreated control). Salicylic acid 10^{-3} M (2.38 mm^2) was statistically on par with BTH 10^{-3} M (2.08 mm^2), but it was significantly superior to ferric chloride 10^{-4} M (2.83 mm^2), sodium selenite 10^{-5} M (2.88 mm^2) and zinc sulphate 10^{-3} M (4.04 mm^2).

Seed treatment along with two foliar sprays had produced the smallest lesions (3.80 mm^2) followed by seed treatment with one foliar spray (4.03 mm^2) when compared to seed treatment alone (4.19 mm^2) in HR-12. However, Seed treatment followed by two foliar sprays was found statistically on par with seed treatment followed by one foliar spray, but was found significantly superior to seed treatment alone in HR-12. Conversely, there was no significant difference between seed treatment alone (3.23

Table 14: Effect of systemic acquired resistance inducing chemicals in the varieties HR-12, Swarnadhan and Rasi on different disease parameters during *Kharif*, 2003

Chemicals/Treatments	Incubation period (days)			Lesion size (mm^2)			No. of lesions per leaf*			No. of conidia/mm^2 of lesion*		
	HR-12	Swarna-dhan	Rasi	HR-12	Swarna-dhan	Rasi	HR-12	Swarna-dhan	Rasi	HR-12	Swarna-dhan	Rasi
Chemicals												
Salicylic acid 10^{-3}M	3.67	9.25	No incidence	3.28	2.38	No incidence	6.00 (2.64)	2.75 (1.93)	No incidence	1370.00 (37.00)	838.83 (28.97)	No incidence
BTH 10^{-3}M	3.67	10.00	-do-	2.98	2.08	-do-	5.75 (2.59)	2.58 (1.89)	-do-	1321.67 (36.35)	794.50 (28.19)	-do-
Ferric chloride 10^{-4}M	3.25	7.92	-do-	3.88	2.83	-do-	8.17 (3.02)	3.75 (2.17)	-do-	1581.00 (39.76)	874.33 (29.58)	-do-
Sodium selenite 10^{-5}M	3.17	7.42	-do-	3.83	2.88	-do-	9.08 (3.17)	3.75 (2.17)	-do-	1665.67 (40.80)	904.17 (30.08)	-do-
Zinc sulphate 10^{-3}M	3.08	6.58	-do-	4.40	4.04	-do-	10.92 (3.45)	5.75 (2.60)	-do-	1777.25 (42.04)	987.67 (31.42)	-do-
Control	3.17	5.67	-do-	5.65	5.00	-do-	12.42 (3.66)	8.67 (3.10)	-do-	2168.92 (46.55)	1208.33 (34.76)	-do-
SEm±	0.14	0.21		0.13	0.17		0.05	0.04		0.47	0.27	
CD (P=0.05)	0.39	0.60		0.36	0.48		0.14	0.12		1.34	0.78	
Treatments												
ST alone	3.63	7.75	No incidence	4.19	3.23	No incidence	9.33 (3.19)	4.92 (2.40)	No incidence	1744.04 (41.61)	963.33 (30.97)	No incidence
ST + 1 spray	3.21	7.96	-do-	4.03	3.23	-do-	8.38 (3.02)	4.33 (2.26)	-do-	1610.50 (39.95)	924.75 (30.33)	-do-
ST + 2 sprays	3.17	7.71	-do-	3.80	3.15	-do-	8.46 (3.05)	4.38 (2.27)	-do-	1587.71 (39.69)	915.83 (30.20)	-do-
SEm±	0.10	0.15		0.09	0.12		0.04	0.03		0.33	0.19	
CD (P=0.05)	0.28	ns		0.26	ns		0.10	0.09		0.95	0.55	
Interaction												
SEm±	0.24	0.37		0.22	0.23		0.09	0.07		0.82	0.47	
CD (P=0.05)	ns	ns		ns	ns		ns	ns		ns	ns	

*Figures in parentheses are square route transformed values

mm^2) and seed treatment followed by either one foliar spray (3.23 mm^2) or two foliar sprays (3.15 mm^2) in Swarnadhan. The interaction effects of different SAR inducing chemicals and the number of applications were found insignificant.

4.3.3.7 Effect on number of lesions/leaf during *Kharif*, 2003

The results presented in Table 14 show that the SAR inducing chemicals significantly reduced the number of lesions per leaf over the untreated control in both HR-12 and Swarnadhan, while there was no disease incidence in Rasi. In HR-12, the number of lesions per leaf ranged between 5.75 (BTH 10^{-3} M) and 12.42 (untreated control). Salicylic acid 10^{-3} M (6 lesions/leaf) found statistically on par with BTH 10^{-3} M (5.75 lesions/leaf), but had shown significantly superior effect over rest of the chemicals *viz.* ferric chloride 10^{-4} M (8.17 lesions/leaf), sodium selenite 10^{-5} M (9.08 lesions/leaf) and zinc sulphate 10^{-3} M (10.92 lesions/leaf). Ferric chloride, sodium selenite and zinc sulphate differed significantly with one another. In Swarnadhan, the number of lesions per leaf ranged between 2.58 (BTH 10^{-3} M) and 8.67 (untreated control). Salicylic acid 10^{-3} M (2.75 lesions/leaf) was found statistically on par with BTH 10^{-3} M (2.58 lesions/leaf), but it was found significantly superior to ferric chloride 10^{-4} M (3.75 lesions/leaf), sodium selenite 10^{-5} M (3.75 lesions/leaf) and zinc sulphate 10^{-3} M (5.75 lesions/leaf).

Seed treatment followed by one foliar spray produced the lowest number of lesions per leaf (8.38) in HR-12, but it was found statistically on par with seed treatment followed by two foliar sprays (8.46 lesions/leaf) and significantly superior to seed treatment alone (9.33 lesions/leaf). Similarly in Swarnadhan also, seed treatment followed by one foliar spray produced the lowest number of lesions per leaf (4.33), but it was found statistically on par with seed treatment followed by two foliar sprays (4.38

lesions/leaf) and significantly superior to seed treatment alone (4.92 lesions/leaf). However, the interaction effects of SAR inducing chemicals and the number of applications were found insignificant.

4.3.3.8 Effect on number of conidia/mm^2 of lesion during *Kharif*, 2003

The results presented in Table 14 reveal that the SAR inducing chemicals were effective in reducing the number of conidia per square mm of lesion when compared to the untreated control in both HR-12 and Swarnadhan while there was no disease incidence in Rasi. The number of conidia per mm^2 of lesion ranged between 1321.67 (BTH 10^{-3} M) and 2168.92 (untreated control) in HR-12. Salicylic acid 10^{-3} M (1370 conidia/mm^2) and BTH 10^{-3} M (1321.67 conidia/mm^2) were statistically on par with each other, but were significantly superior to ferric chloride 10^{-4} M (1581 conidia/mm^2), sodium selenite 10^{-5} M (1665.67 conidia/mm^2) and zinc sulphate 10^{-3} M (1777.25 conidia/mm^2) in reducing the number of conidia per mm^2 of lesion in HR-12. Ferric chloride was statistically on par with sodium selenite, but it was significantly superior to zinc sulphate in HR-12. The number of conidia per mm^2 of lesion ranged between 794.5 (BTH 10^{-3} M) and 1208.33 (untreated control) in Swarnadhan. Salicylic acid 10^{-3} M (838.83 conidia/mm^2) was statistically on par with both BTH 10^{-3} M (794.5 conidia/mm^2) as well as ferric chloride (874.33 conidia/mm^2), but these three were significantly superior to sodium selenite 10^{-5} M (904.17/mm^2) and zinc sulphate 10^{-3} M (987.67/mm^2) in Swarnadhan.

Seed treatment with two foliar sprays had produced the lowest number of conidia per mm^2 lesion (1587.71) followed by seed treatment with one foliar spray (1610.50 conidia/mm^2 per lesion) while seed treatment alone had produced 1744.04 conidia/mm^2 in HR-12. However, seed treatment followed by either one foliar spray or two foliar sprays was significantly superior to seed treatment alone. The same trend was

reflected in Swarnadhan also where seed treatment alone and seed treatment followed with either one or two sprays produced 963.33, 924.75 and 915.83 conidia/mm^2, respectively. The interaction effects of SAR inducing chemicals and the number of applications were found insignificant.

4.3.3.9 Effect on incubation period (*Kharif*, 2002 & 2003; pooled data)

The results presented in Table 15 show that the minimum number of days required for the appearance of initial disease symptoms were 3.13 days in HR-12 and 5.29 days in Swarnadhan, while there was no disease incidence in Rasi. The incubation period ranged between 3.13 days (untreated control) and 3.58 days (salicylic acid 10^{-3} M) in HR-12. Salicylic acid (3.58 days) and BTH (3.54 days) were significantly superior to other chemicals as well as untreated control though they were statistically on par with one another. The SAR inducing chemicals significantly delayed the appearance of initial disease symptoms as compared to untreated control in Swarnadhan. The incubation period ranged between 5.29 days (untreated control) and 9.46 days (BTH 10^{-3} M) in Swarnadhan. BTH 10^{-3} M (9.46 days) was statistically on par with salicylic acid 10^{-3} M (9.21 days). However, these two chemicals had shown significantly superior performance over ferric chloride 10^{-4} M (7.88 days), sodium selenite 10^{-5} M (7.29 days) and zinc sulphate 10^{-3} M (6.42 days). But ferric chloride, sodium selenite and zinc sulphate significantly differ with one another and with the untreated control.

Seed treatment alone with SAR inducing chemicals (3.58 days) was significantly superior when compared to seed treatment followed by either one foliar spray (3.17 days) or two foliar sprays (3.15 days) in HR-12. Conversely, seed treatment

alone (7.33 days) was found statistically on par with seed treatment followed by two foliar sprays (7.56 days), but significantly superior to seed treatment followed by one foliar spray (7.88 days) in Swarnadhan. The interaction effects of different SAR inducing chemicals and the number of applications were found insignificant.

4.3.3.10 Effect on lesion size (*Kharif*, 2002 & 2003; pooled data)

The results presented in Table 15 reveal that the SAR inducing chemicals significantly reduced the lesion size over the untreated control in both HR-12 and Swarnadhan, while there was no disease incidence in Rasi. In HR-12, the lesion size ranged between 3.36 mm^2 (salicylic acid 10^{-3} M) and 5.88 mm^2 (untreated control). BTH 10^{-3} M (3.38 mm^2) was statistically on par with salicylic acid 10^{-3} M (3.36 mm^2), but it was significantly superior to ferric chloride 10^{-4} M (3.92 mm^2), sodium selenite 10^{-5} M (4.48 mm^2) and zinc sulphate 10^{-3} M (4.93 mm^2). In Swarnadhan, the lesion size ranged between 1.95 mm^2 (BTH 10^{-3} M) and 4.40 mm^2 (untreated control). Salicylic acid 10^{-3} M (2.20 mm^2) was statistically on par with both BTH 10^{-3} M (1.95 mm^2) and ferric chloride 10^{-4} M (2.54 mm^2), but significantly superior to sodium selenite 10^{-5} M (2.96 mm^2) and zinc sulphate 10^{-3} M (3.59 mm^2).

Seed treatment along with two foliar sprays had produced the smallest lesions (4.13 mm^2) followed by seed treatment with one foliar spray (4.24 mm^2) when compared to seed treatment alone (4.59 mm^2) in HR-12. However, Seed treatment followed by two foliar sprays was statistically on par with seed treatment followed by one foliar spray, but was found significantly superior to seed treatment alone in HR-12. Conversely, there was no significant difference between seed treatment alone (2.98 mm^2)

Table 15: Effect of systemic acquired resistance inducing chemicals in the varieties HR-12, Swarnadhan and Rasi on different disease parameters (*Kharif*, 2002 & 2003; pooled data)

Chemicals/Treatments	Incubation period (days)			Lesion size (mm^2)			No. of lesions per leaf*			No. of conidia/mm^2 of lesion*		
	HR-12	Swarna-dhan	Rasi	HR-12	Swarna-dhan	Rasi	HR-12	Swarna-dhan	Rasi	HR-12	Swarna-dhan	Rasi
Chemicals												
Salicylic acid 10^{-3}M	3.58	9.21	No incidence	3.36	2.20	No incidence	6.71 (2.77)	2.83 (1.95)	No incidence	1367.21 (36.97)	845.63 (29.09)	No incidence
BTH 10^{-3}M	3.54	9.46	-do-	3.38	1.95	-do-	6.67 (2.76)	2.79 (1.94)	-do-	1337.17 (36.55)	805.88 (28.40)	-do-
Ferric chloride 10^{-4}M	3.17	7.88	-do-	3.92	2.54	-do-	8.71 (3.11)	3.46 (2.10)	-do-	1468.58 (38.31)	892.38 (29.88)	-do-
Sodium selenite 10^{-5}M	3.21	7.29	-do-	4.48	2.96	-do-	8.67 (3.10)	5.00 (2.45)	-do-	1683.08 (41.02)	954.38 (30.90)	-do-
Zinc sulphate 10^{-3}M	3.17	6.42	-do-	4.93	3.59	-do-	10.46 (3.38)	6.33 (2.71)	-do-	1757.29 (41.90)	999.92 (31.63)	-do-
Control	3.13	5.29	-do-	5.88	4.40	-do-	12.29 (3.64)	8.71 (3.11)	-do-	2031.79 (45.08)	1219.00 (34.92)	-do-
SEm±	0.10	0.19		0.11	0.12		0.04	0.04		0.32	0.20	
CD (P=0.05)	0.29	0.53		0.31	0.34		0.11	0.11		0.91	0.56	
Treatments												
ST alone	3.58	7.33	No incidence	4.59	2.98	No incidence	9.52 (3.23)	5.56 (2.52)	No incidence	1685.25 (40.97)	984.90 (31.33)	No incidence
ST + 1 spray	3.17	7.88	-do-	4.24	2.92	-do-	8.63 (3.08)	4.67 (2.32)	-do-	1585.94 (39.67)	936.81 (30.54)	-do-
ST + 2 sprays	3.15	7.56	-do-	4.13	2.92	-do-	8.60 (3.08)	4.98 (2.39)	-do-	1551.38 (39.28)	936.88 (30.54)	-do-
SEm±	0.07	0.13		0.08	0.08		0.03	0.03		0.23	0.14	
CD (P=0.05)	0.20	0.37		0.22	ns		0.08	0.08		0.65	0.40	
Interaction												
SEm±	0.17	0.32		0.19	0.21		0.07	0.07		0.56	0.34	
CD (P=0.05)	ns	ns		ns	ns		ns	ns		ns	ns	

*Figures in parentheses are square route transformed values

Fig. 14: Effect of SAR inducing chemicals in the variety HR-12 on different disease parameters(*Kharif*, 2002&03)

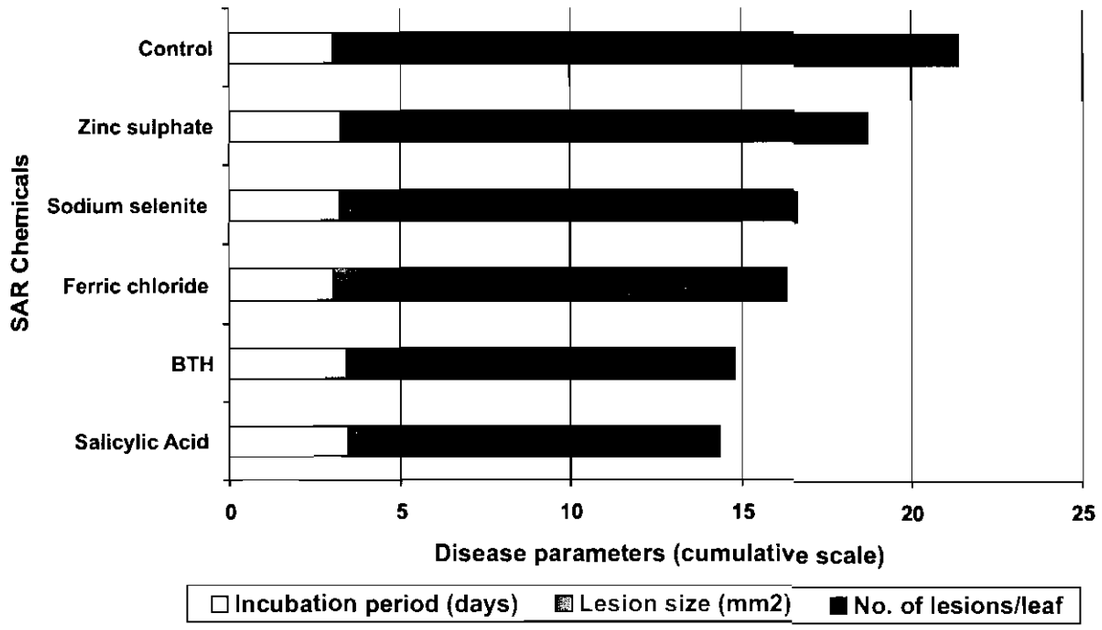


Fig. 15: Effect of SAR inducing chemicals as different kinds of treatments in the variety HR-12(*Kharif*, 2002&03)

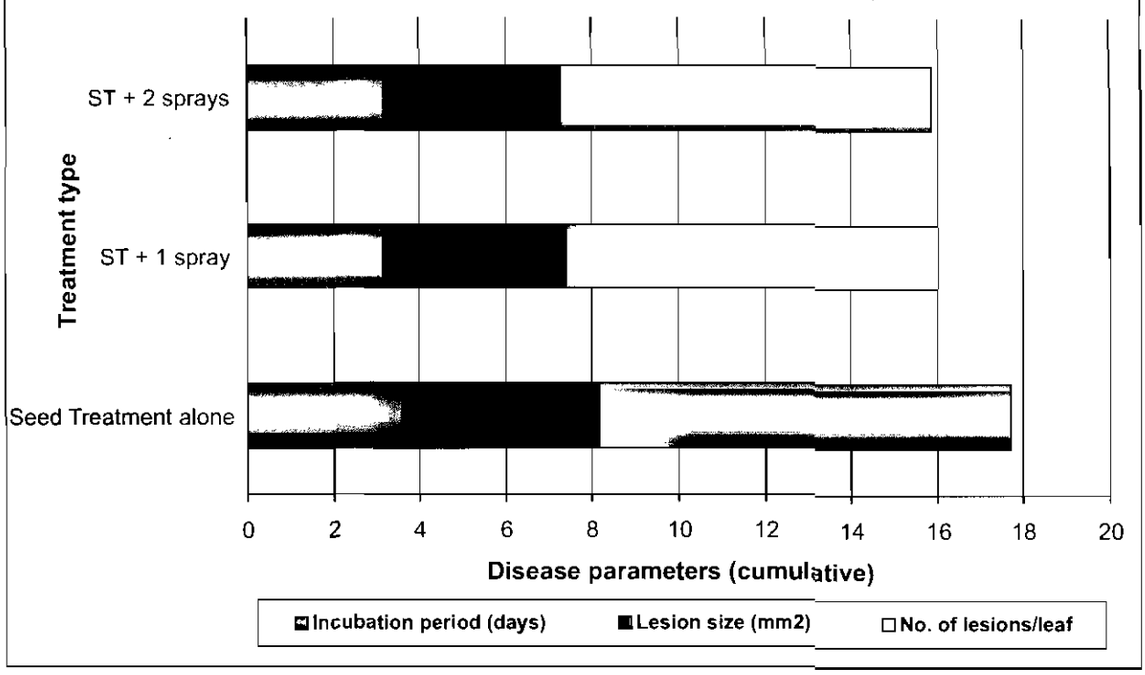


Fig. 16: Effect of SAR inducing chemicals in the variety Swarnadhan on different disease parameters (*Kharif*, 2002&03)

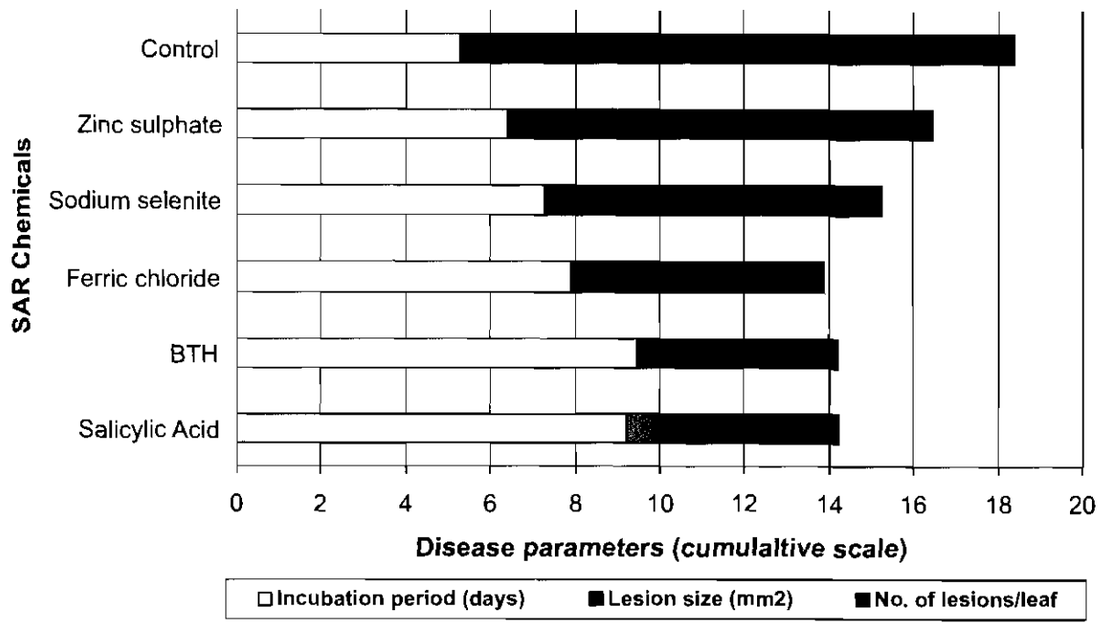
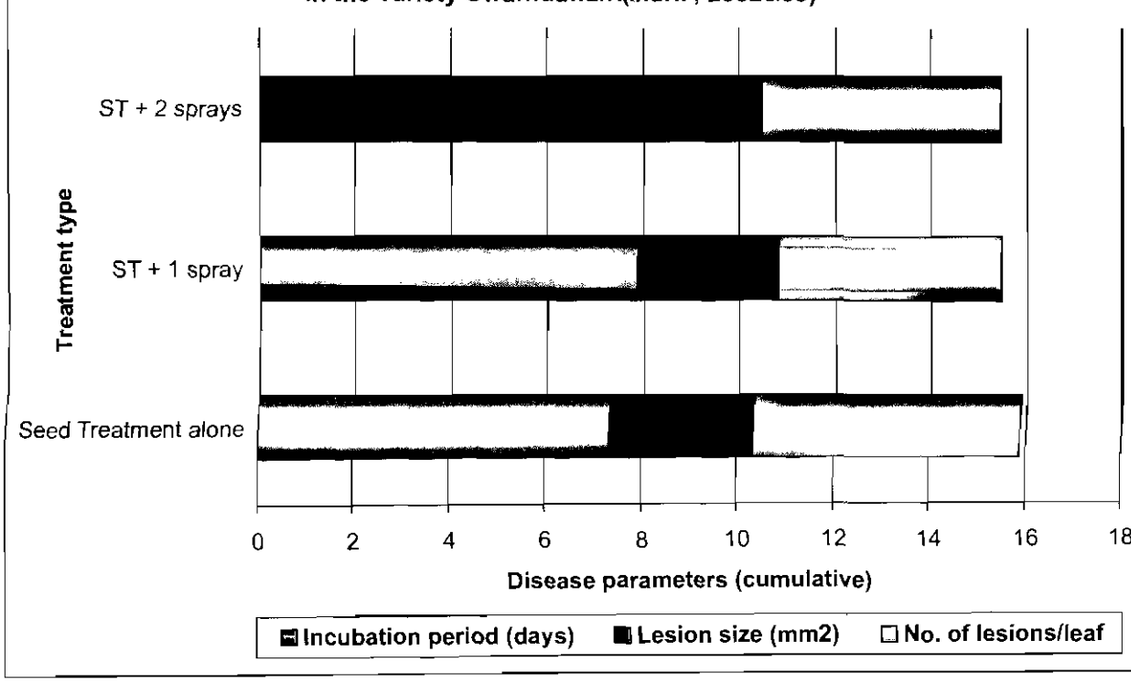


Fig. 17: Effect of SAR chemicals as different kinds of treatments in the variety Swarnadhan (*Kharif*, 2002&03)



and seed treatment followed by either one foliar spray or two foliar sprays (2.92 mm^2) in Swarnadhan. The interaction effects of different SAR inducing chemicals and the number of applications were insignificant.

4.3.3.11 Effect on number of lesions/leaf (*Kharif*, 2002 & 2003; pooled data)

The results presented in Table 15 show that the SAR inducing chemicals significantly reduced the number of lesions per leaf over the untreated control in both HR-12 and Swarnadhan while there was no disease incidence in Rasi. In HR-12, the number of lesions ranged between 6.67 (BTH 10^{-3} M) and 12.29 (untreated control). Salicylic acid 10^{-3} M (6.71 lesions/leaf) was statistically on par with BTH 10^{-3} M (6.67 lesions/leaf), but has shown significantly superior effect over rest of the chemicals *viz.* sodium selenite 10^{-5} M (8.67 lesions/leaf), ferric chloride 10^{-4} M (8.71 lesions/leaf) and zinc sulphate 10^{-3} M (10.46 lesions/leaf). Ferric chloride and sodium selenite were statistically on par with each other but significantly superior to zinc sulphate. In Swarnadhan, the number of lesions per leaf ranged between 2.79 (BTH 10^{-3} M) and 8.71 (untreated control). Salicylic acid 10^{-3} M (2.83 lesions/leaf) was statistically on par with BTH 10^{-3} M (2.79 lesions/leaf), but it was significantly superior to ferric chloride 10^{-4} M (3.46 lesions/leaf), sodium selenite 10^{-5} M (5 lesions/leaf) and zinc sulphate 10^{-3} M (6.33 lesions/leaf).

Seed treatment followed by two foliar sprays produced the lowest number of lesions per leaf (8.60) in HR-12, but it was statistically on par with seed treatment followed by one foliar spray (8.63 lesions per leaf) and significantly superior to seed treatment alone (9.52 lesions per leaf). In Swarnadhan, seed treatment followed by one foliar spray produced the lowest number of lesions per leaf (4.67), but it was statistically

on par with seed treatment followed by two foliar sprays (4.98 lesions/leaf) and significantly superior to seed treatment alone (5.56 lesions/leaf). The interaction effects of SAR inducing chemicals and the number of applications were found insignificant.

4.3.3.12 Effect on number of conidia/mm^2 of lesion (*Kharif*, 2002 & 2003; pooled data)

The results presented in Table 15 reveal that the SAR inducing chemicals were found effective in reducing the number of conidia per square mm of lesion when compared to the untreated control in both HR-12 and Swarnadhan, while there was no disease incidence in Rasi. The number of conidia per mm^2 of lesion ranged between 1337.17 (BTH 10^{-3} M) and 2031.79 (untreated control) in HR-12. Salicylic acid 10^{-3} M (1367.21 conidia/mm^2) and BTH 10^{-3} M (1337.17 conidia/mm^2) were statistically on par with each other, but were found significantly superior to ferric chloride 10^{-4} M (1468.58 conidia/mm^2), sodium selenite 10^{-5} M (1683.08 conidia/mm^2) and zinc sulphate 10^{-3} M (1757.29 conidia/mm^2) in reducing the number of conidia per mm^2 of lesion in HR-12. Sodium selenite was statistically on par with zinc sulphate, but significantly inferior to ferric chloride in HR-12. The number of conidia per mm^2 of lesion ranged between 805.88 (BTH 10^{-3} M) and 1219 (untreated control) in Swarnadhan. The SAR inducing chemicals significantly differ with one another and also with untreated control in the following order, BTH 10^{-3} M (805.88 conidia/mm^2), salicylic acid 10^{-3} M (845.63 conidia/mm^2), sodium selenite 10^{-5} M (954.38 conidia/mm^2), ferric chloride (892.38 conidia/mm^2) and zinc sulphate 10^{-3} M (999.92 conidia/mm^2).

Seed treatment with two foliar sprays had produced the lowest number of conidia per mm^2 lesion (1551.38) followed by seed treatment with one foliar spray (1585.94 conidia/mm^2 per lesion) while seed treatment alone had produced 1685.25

conidia/mm^2 in HR-12. Seed treatment followed by either one foliar spray or two foliar sprays was significantly superior to seed treatment alone. The same trend was reflected in Swarnadhan also where seed treatment alone and seed treatment followed with either one or two sprays produced 984.90, 936.81 and 936.88 conidia/mm^2, respectively. The interaction effects of SAR inducing chemicals and the number of applications were found insignificant.

4.4 EFFICACY OF FUNGICIDES AGAINST P. GRISEA IN VITRO:

An *in vitro* experiment was conducted to know the efficacy of 17 fungicides at 1, 5, 10, 25, 50, 100, 250, 500 and 1000 µg/ml concentrations by employing 'Inhibition Zone Technique'. The fungicides exhibited wide and varied response against *P. grisea* in the laboratory. The combination of carbendazim + epoxiconazole 25 EC had given best *in vitro* control of *P. grisea* while pyroquilon 50 WP, tricyclazole 75 WP and fenoxanil 20 SC were found completely ineffective in inhibiting the mycelial growth of the fungus. The zone of inhibition progressively increased with increasing concentrations of all the fungicides except pyroquilon 50 WP, tricyclazole 75 WP and fenoxanil 20 SC (Table 16).

The fungicides *viz.* carbendazim + epoxiconazole 25 EC (8.66 mm), azoxystrobin 25 SC (5.66 mm), carbendazim 50 WP (4.66 mm), pyraclostrobin 25 EC (4.66 mm) and RIL 010 25 SC (2.66 mm) inhibited mycelial growth at 1 µg/ml concentration while some other fungicides *viz.* difenconazole + propiconazole 30 EC (3.66 mm), edifenphos 50 EC (3.33 mm), isoprothiolane 40 EC (3 mm), propiconazole 25 EC (2.66 mm) and iprobenfos 48 EC (1.66 mm) started inhibiting mycelial growth at 5 µg/ml concentration. The remaining fungicides *viz.* epoxiconazole 7.5 EC (1.33 mm),

Table 16: *In vitro* efficacy of different fungicides against *Pyricularia grisea*

S. No	Name of the fungicide	Inhibition zone at different concentrations of the fungicide (mm)										Mean
		0 (µg/ml)	1 (µg/ml)	5 (µg/ml)	10 (µg/ml)	25 (µg/ml)	50 (µg/ml)	100 (µg/ml)	250 (µg/ml)	500 (µg/ml)	1000 (µg/ml)	
1	Carbendazim + Epoxiconazole 25 EC	0	8.66	11.33	15.00	18.33	25.66	36.00	39.33	44.66	54.33	25.33
2	Carbendazim 50 WP	0	4.66	8.66	14.33	17.00	20.00	25.66	31.00	40.00	45.00	20.63
3	Azoxystrobin 25 SC	0	5.66	8.00	12.33	17.33	19.00	21.66	23.33	25.33	26.33	15.91
4	Pyraclostrobin 25 EC	0	4.66	8.00	11.66	16.00	18.33	20.66	23.00	24.33	26.33	15.31
5	RIL 010 25 SC	0	2.66	8.33	11.00	13.33	15.33	18.00	21.00	22.66	24.00	13.63
6	Difenconazole + Propiconazole 30 EC	0	0	3.66	9.00	12.33	14.33	17.33	20.33	23.33	24.00	12.43
7	Edifenphos 50 EC	0	0	3.33	8.00	11.33	14.00	17.00	21.33	24.66	25.33	9.36
8	Isoprothiolane 40 EC	0	0	3.00	6.00	8.00	12.66	14.00	15.66	16.66	17.66	12.51
9	Propiconazole 25 EC	0	0	2.66	7.00	10.66	13.66	16.00	17.33	19.66	21.00	10.81
10	Iprobenfos 48 EC	0	0	1.66	4.66	8.66	12.00	17.00	18.00	22.33	24.33	10.86
11	Epoxiconazole 7.5 EC	0	0	0	1.33	2.66	6.66	8.33	9.66	11.66	12.33	5.26
12	Kasugamycin 2L	0	0	0	0.66	2.33	5.33	10.66	13.33	18.66	20.00	7.11
13	Difenconazole 25 EC	0	0	0	0.33	3.33	6.00	10.33	12.66	14.33	15.33	6.23
14	Diniconazole 25 WP	0	0	0	0.33	1.33	4.66	6.66	8.33	10.00	10.66	4.16
15	Pyroquilon 50 WP	0	0	0	0	0	0	0	0	0	0	0
16	Tricyclazole 75 WP	0	0	0	0	0	0	0	0	0	0	0
17	Fenoxanil 20 SC	0	0	0	0	0	0	0	0	0	0	0
Mean		0	1.55	3.45	6.01	8.41	11.04	14.11	16.13	18.72	20.39	

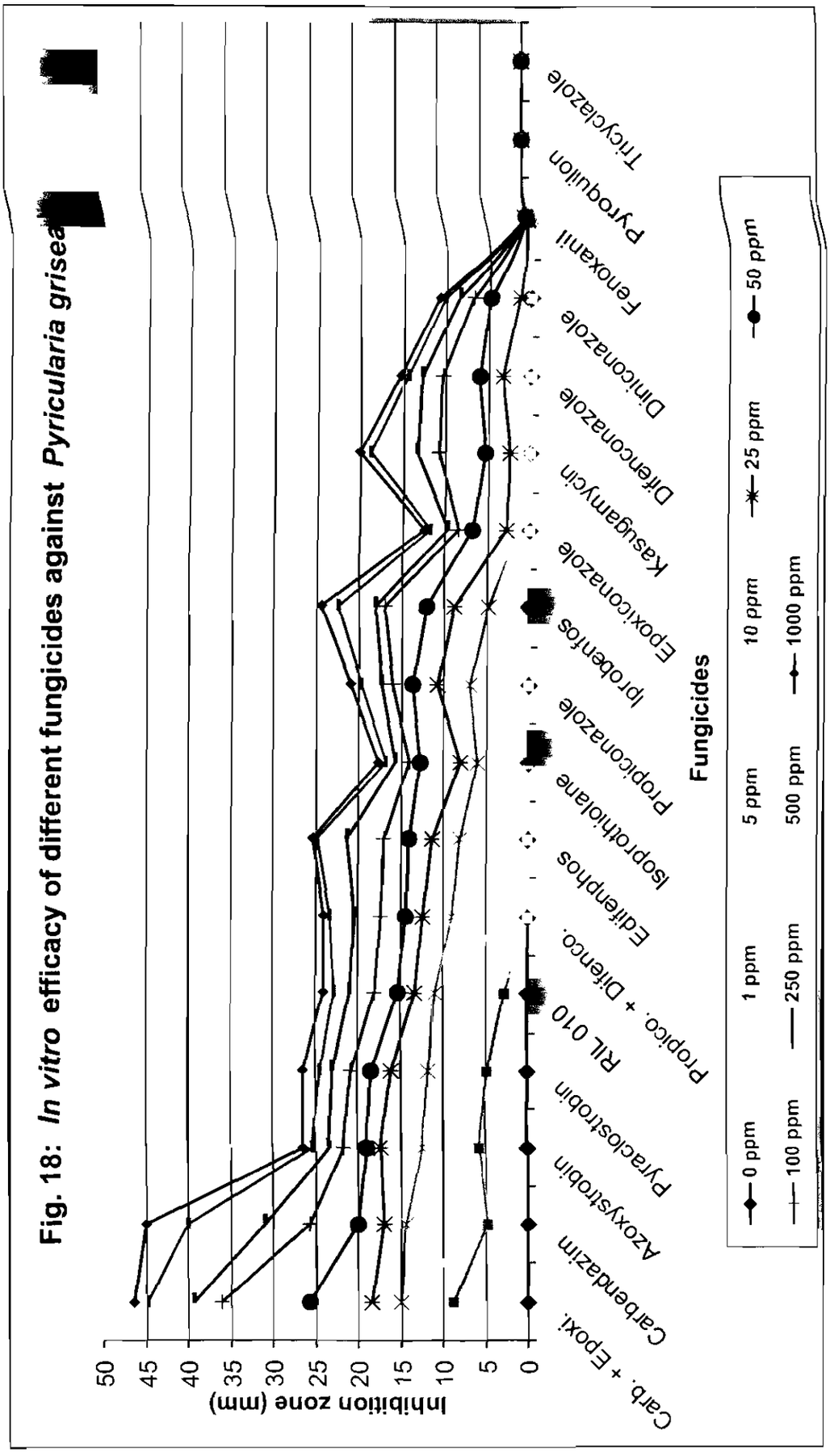


Fig. 18: *In vitro* efficacy of different fungicides against *Pyricularia grisea*

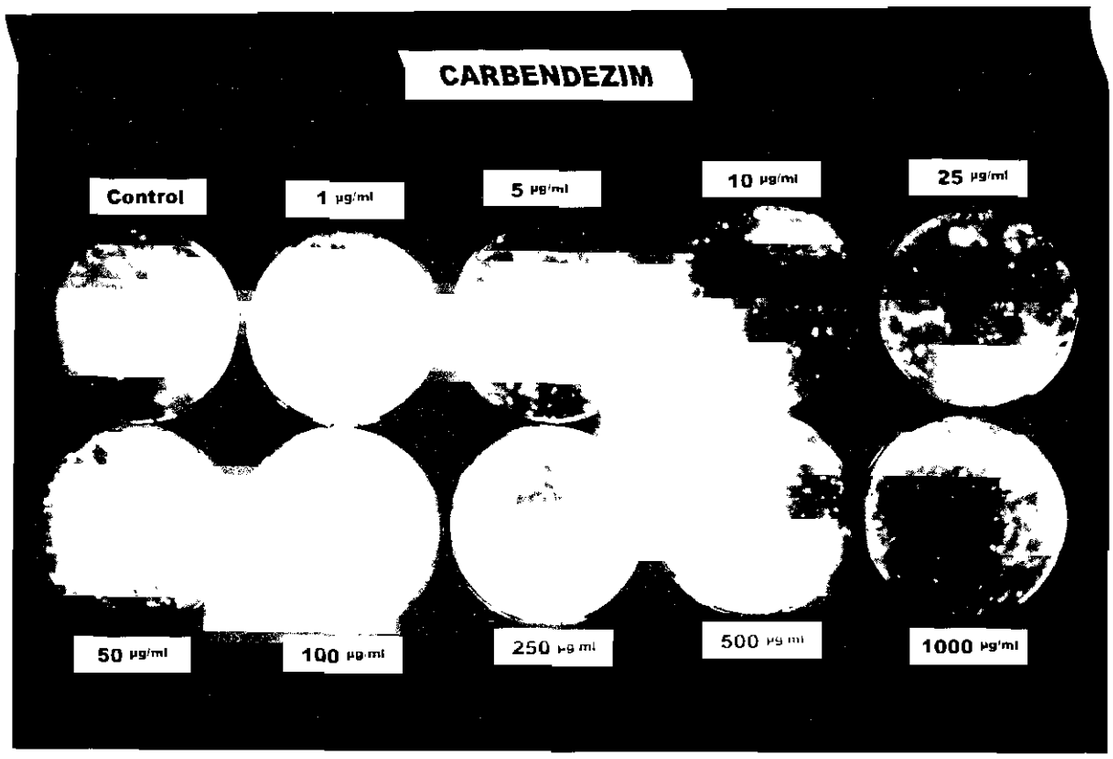


Plate 21: *In vitro* inhibition of mycelial growth of *P. grisea* at different concentrations of carbendazim 50 WP

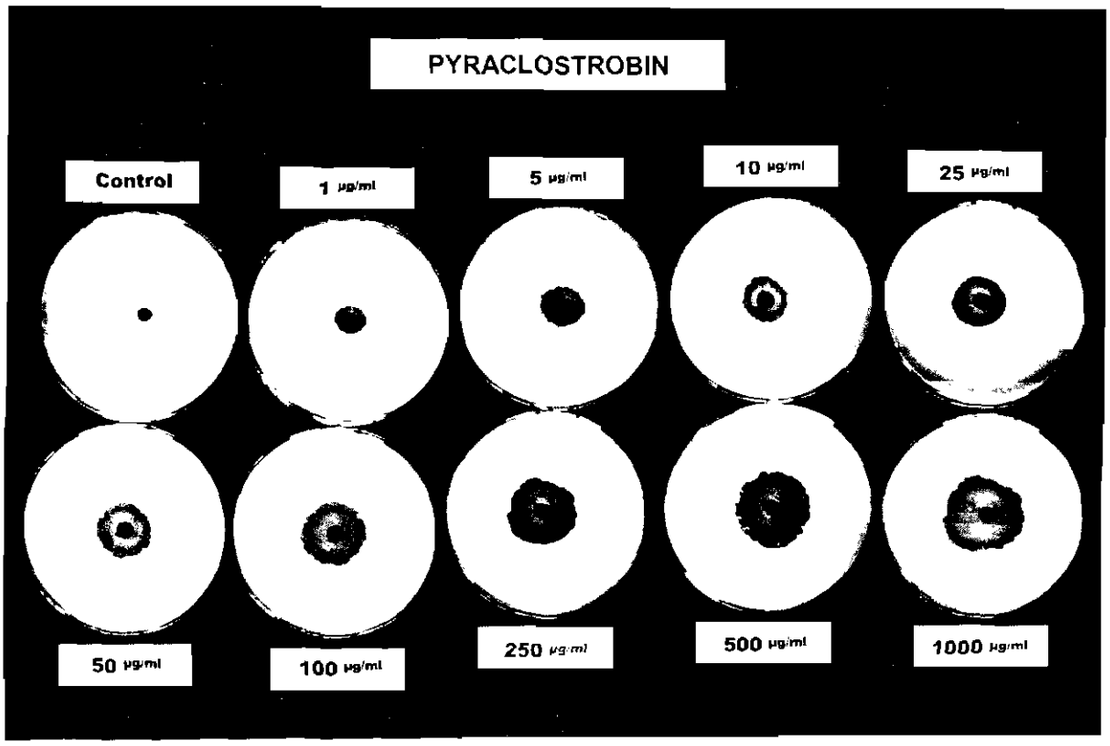


Plate22: *In vitro* inhibition of mycelial growth of *P. grisea* at different concentrations of pyraclostrobin 25 EC

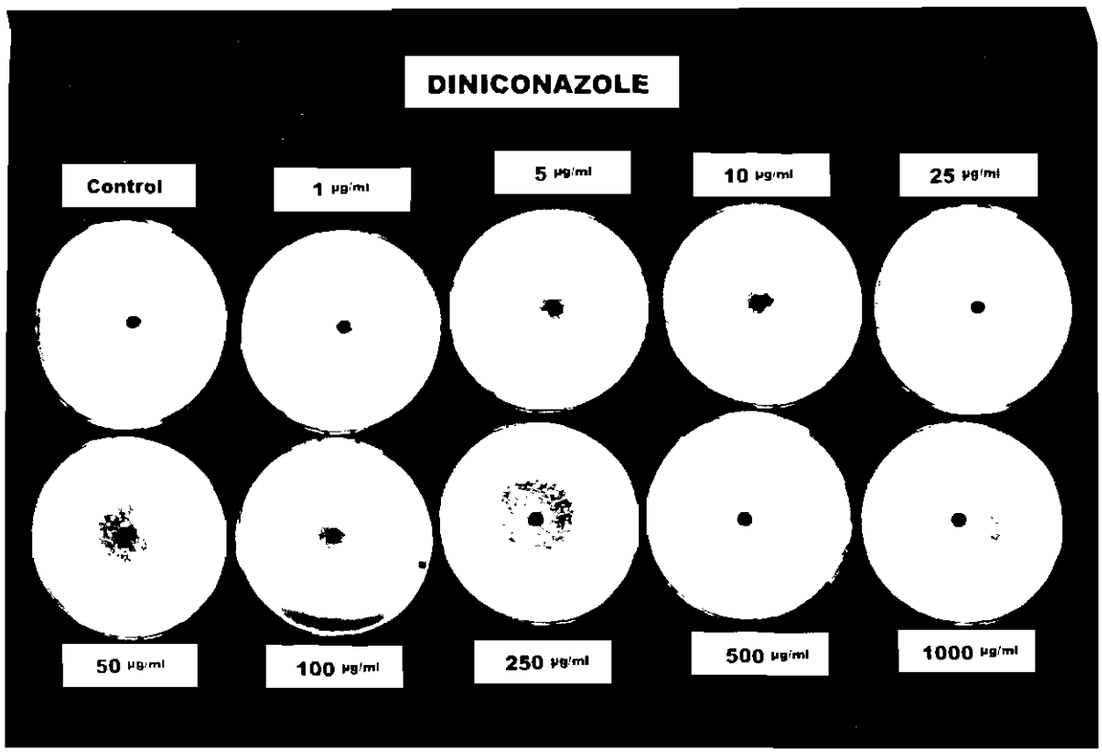


Plate 23: *In vitro* inhibition of mycelial growth of *P. grisea* at different concentrations of diniconazole 25 WP

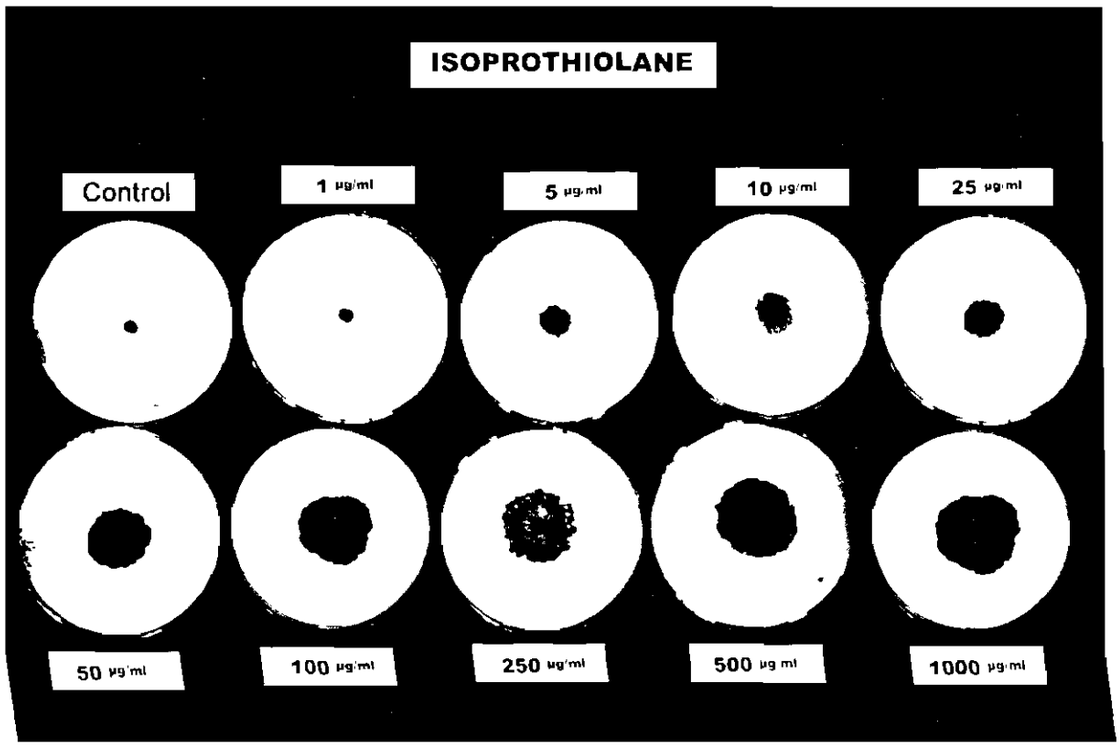


Plate 24: *In vitro* inhibition of mycelial growth of *P. grisea* at different concentrations of isoprothiolane 40 EC

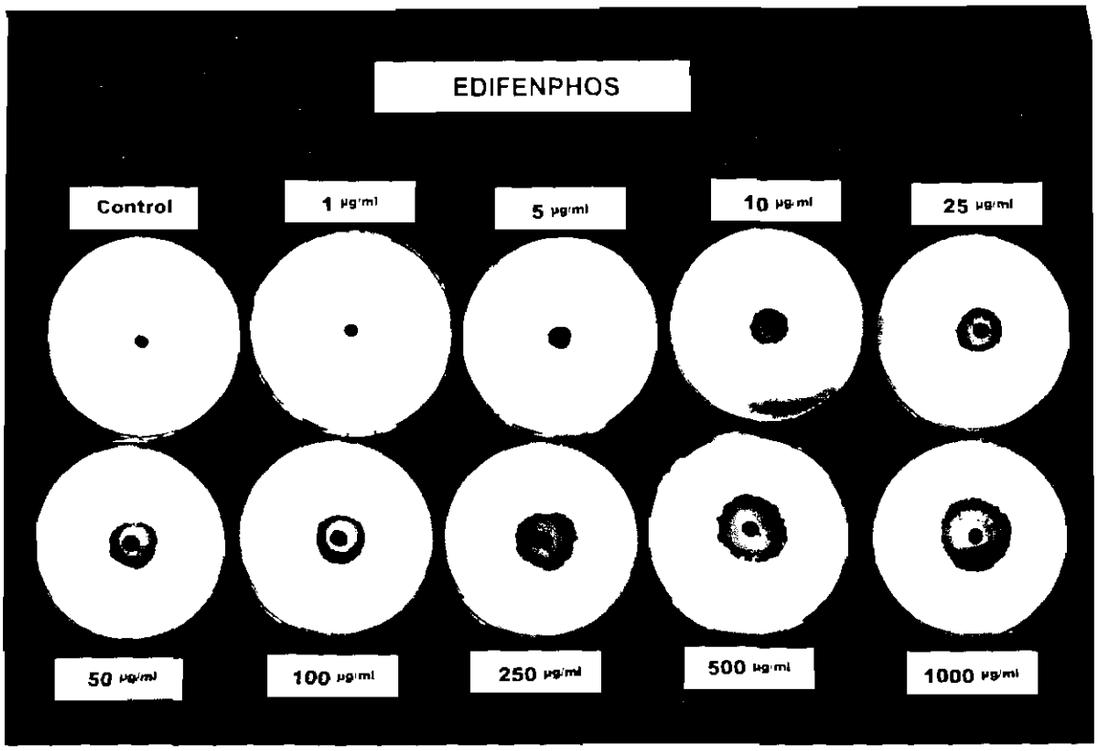


Plate 25: *In vitro* inhibition of mycelial growth of *P. grisea* at different concentrations of edifenphos 50 EC

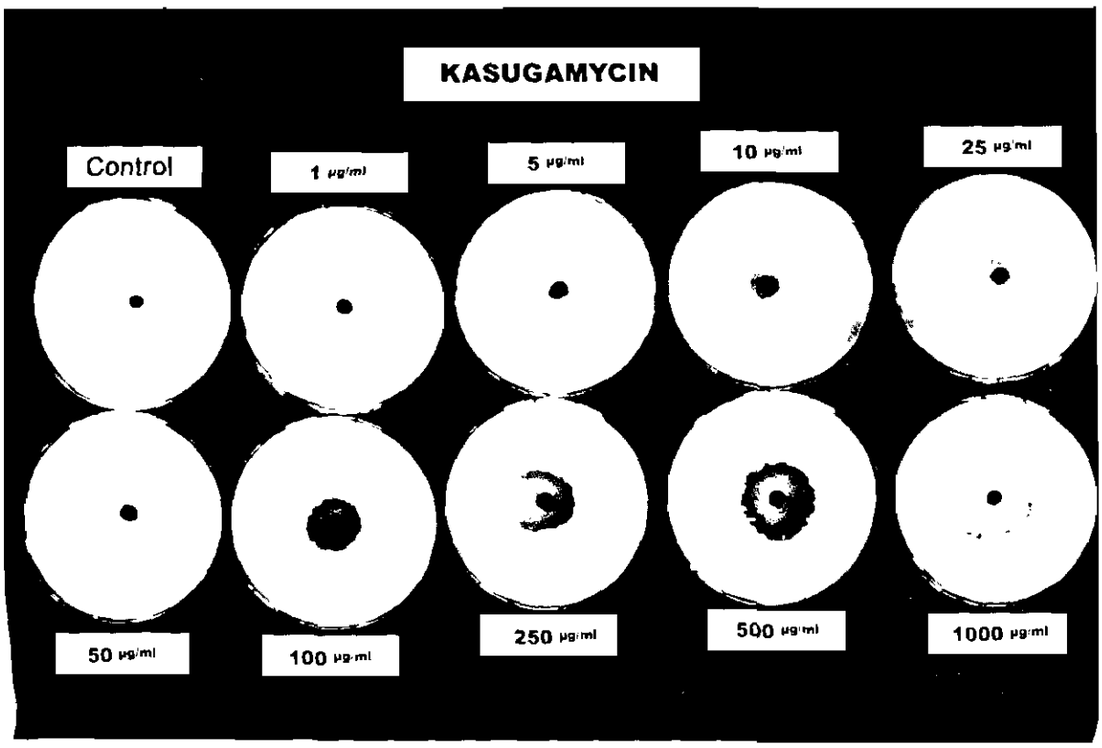


Plate 26: *In vitro* inhibition of mycelial growth of *P. grisea* at different concentrations of kasugamycin 2 L

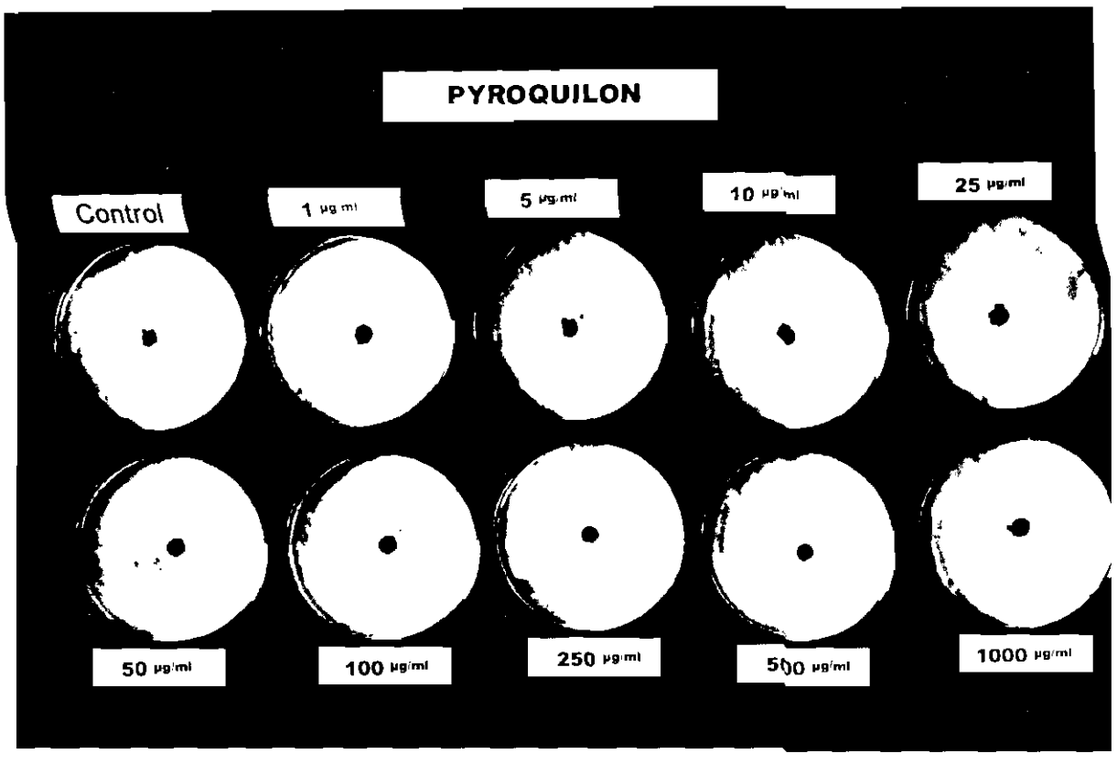


Plate 27: *In vitro* inhibition of mycelial growth of *P. grisea* at different concentrations of pyroquilon 50 WP

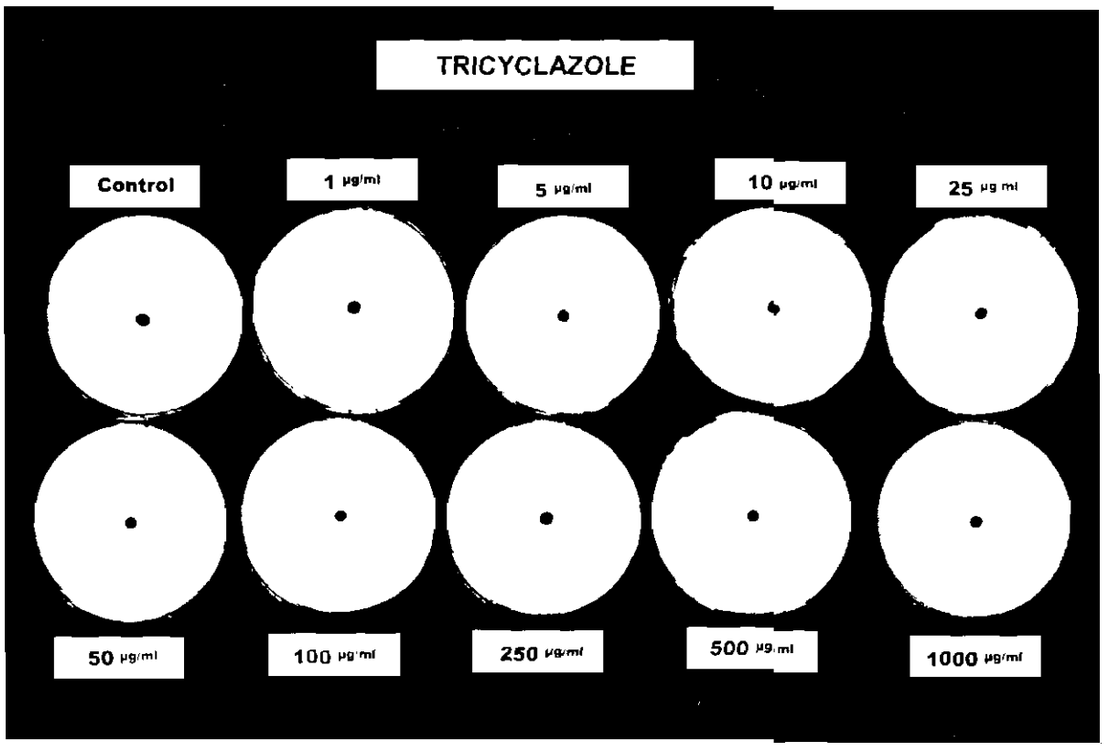


Plate 28: *In vitro* inhibition of mycelial growth of *P. grisea* at different concentrations of tricyclazole 75 WP

kasugamycin 2L (0.66 mm), difenconazole 25 EC (0.33 mm) and diniconazole 25 WP (0.33 mm) started forming inhibition zones at 10 µg/ml concentration except pyroquilon 50 WP, tricyclazole 75 WP and fenoxanil 20 SC (Plates 21 to 28).

The biggest inhibition zone was formed by carbendazim + epoxiconazole 25 EC (54.33 mm) at 1000 µg/ml concentration followed by carbendazim 50 WP (45 mm), azoxystrobin 25 SC and pyraclostrobin 25 EC (both 26.33 mm), edifenphos 50 EC (25.33 mm), iprobenfos 48 EC (24.33 mm), RIL 010 25 SC and difenconazole + propiconazole 30 EC (both 24 mm), propiconazole 25 EC (21 mm), kasugamycin 2L (20 mm), isoprothiolane 40 EC (17.66 mm), difenconazole 25 EC (15.33 mm), epoxiconazole 7.5 EC (12.33 mm) and diniconazole 25 WP (10.66 mm) while pyroquilon 50 WP, tricyclazole 75 WP and fenoxanil 20 SC did not form any inhibition zone even at the concentration of 1000 µg/ml (Table 16).

The size of the inhibition zone between any two successive concentrations varied differently for different fungicides which were reflected by the mean inhibition zone of all the concentrations up to 1000 µg/ml. The average size of the inhibition zone was highest in the case of carbendazim + epoxiconazole 25 EC (25.33 mm) followed by carbendazim 50 WP (20.63 mm), azoxystrobin 25 SC (15.91 mm), pyraclostrobin 25 EC (15.31 mm), RIL 010 25 EC (13.63 mm), isoprothiolane 40 EC (12.51 mm), difenconazole + propiconazole 30 EC (12.43 mm), iprobenfos 48 EC (10.86 mm), propiconazole 25 EC (10.81 mm), edifenphos 50 EC (9.36 mm), kasugamycin 2L (7.11 mm), difenconazole 25 EC (6.23 mm), epoxiconazole 7.5 EC (5.26 mm) and diniconazole 25 WP (4.16 mm). The difference between inhibition zones of two

successive concentration was greater at 1-5, 5-10, 10-25, 25-50, 50-100 and 100-250 µg/ml when compared 250-500 and 500-1000 µg/ml for all the fungicides except pyroquilon 50 WP, tricyclazole 75 WP and fenoxanil 20 SC (Table 16 & Plates 21 to 28).

4.5 EFFICACY OF FUNGICIDES AGAINST RICE BLAST IN VIVO:

The efficacy of the fungicides against rice blast disease was tested using the susceptible variety HR-12 under partially controlled conditions (nursery beds) as well as field conditions. Seed treatment and foliar spray were the two kinds of treatments used in the nursery beds while foliar spray alone was employed in the main field.

4.5.1 Efficacy of fungicides as seed treatment in the nursery beds

The experiments were conducted to test the efficacy of the fungicides as seed treatment for two consecutive *Kharif* seasons during 2002 and 2003 in the nursery beds under partially controlled conditions. The details of the fungicides and their respective dosages were presented in the Table 2. The investigation was carried out to know the effect of fungicides on seed germination, leaf blast severity and number of days taken for the appearance of initial disease symptoms. Observations on leaf blast severity were recorded at 5 day interval starting from 5 days after inoculation (DAI) to 20 days after inoculation (when the leaf blast severity reached cent per cent in the untreated control plot). The results of *Kharif*, 2002, *Kharif* 2003 and the pooled data of *Kharif* 2002 & 2003 are presented in the Tables 17, 18 and 19, respectively. The fungicides were presented in the descending order of their efficacy based on per cent leaf blast severity in the final as well as the penultimate observations (Plates 29a & 29b).

4.5.1.1 Efficacy of fungicides as seed treatment in the nursery beds during *Kharif*, 2002

The results presented in Table 17 reveal that all the fungicides tested were found significantly superior over the control up to 20 days after inoculation. Pyroquilon 50 WP, azoxystrobin 25 SC, pyraclostrobin 25 EC and RIL 010 25 SC offered phenomenal control of leaf blast followed by tricyclazole 75 WP and fenoxanil 20 SC. Propiconazole 25 EC had given such a good control that was comparable to carbendazim 50 WP. But the effect of other popular blasticides *viz.* edifenphos 50 EC, iprobenfos 48 EC, isoprothiolane 40 EC and kasugamycin 2 L was found minimal as seed treatment compared to their superior counterparts such as pyroquilon and tricyclazole. The quantum of control offered by rest of the fungicides as seed treatment was negligible. The least effective of the fungicides tested was diniconazole 25 WP. However, the effect of fungicides on seed germination in general was found non-significant.

At 5 days after inoculation, there was absolutely no disease incidence in the plots treated with pyroquilon 50 WP, azoxystrobin 25 SC, pyraclostrobin 25 EC, RIL 010 25 SC, tricyclazole 75 WP and fenoxanil 20 SC, where as the control plot recorded a leaf blast severity of 18.33 per cent. The lowest disease incidence was recorded in carbendazim 50 WP (1.33%) followed by propiconazole 25 EC (1.66%) that were statistically on par with each other. The next levels of disease incidence were found in carbendazim + epoxiconazole 25 EC (2.33%) followed by isoprothiolane 40 EC (2.66%), iprobenfos 48 EC (5%), edifenphos 50 EC (6%), kasugamycin 2 L (6.66%), difenconazole 25 EC (6.66%), epoxiconazole 7.5 EC (7%) and difenconazole + propiconazole 30 EC (7.33%). However, carbendazim + epoxiconazole was statistically

Table 17: Efficacy of fungicides as seed treatment against rice blast in nursery beds (2002)

S. No.	Name of the fungicide	Leaf blast severity (%)*				Appearance of initial symptoms (days)	Seed germination (%)
		5DAI	10DAI	15DAI	20DAI		
1	Pyroquilon 50 WP	0.00 (0.00)	0.00 (0.00)	0.00 (0.00)	4.00 (11.32)	16.33	89.33 (70.95)
2	Azoxystrobin 25 SC	0.00 (0.00)	0.00 (0.00)	0.00 (0.00)	4.66 (12.39)	16.33	96.33 (79.14)
3	Pyraclostrobin 25 EC	0.00 (0.00)	0.00 (0.00)	0.00 (0.00)	5.00 (12.92)	16.00	93.66 (75.61)
4	RIL 010 25 SC	0.00 (0.00)	0.00 (0.00)	0.66 (3.83)	5.66 (13.73)	14.66	94.00 (75.82)
5	Tricyclazole 75 WP	0.00 (0.00)	0.66 (2.71)	3.00 (9.88)	10.00 (18.05)	10.66	88.66 (70.34)
6	Fenoxanil 20 SC	0.00 (0.00)	0.66 (2.71)	6.66 (14.95)	18.33 (25.31)	9.00	93.33 (75.07)
7	Carbendazim 50 WP	1.33 (6.54)	7.00 (15.18)	16.66 (24.08)	26.66 (30.95)	5.33	93.66 (76.03)
8	Propiconazole 25 EC	1.66 (7.33)	6.66 (14.95)	21.66 (27.71)	33.33 (35.22)	5.00	92.00 (73.65)
9	Carbendazim + Epoxiconazole 25 EC	2.33 (8.74)	10.00 (18.43)	25.00 (30.00)	36.66 (37.22)	5.33	88.66 (75.81)
10	Isoprothiolane 40 EC	2.66 (9.39)	11.66 (19.89)	26.66 (31.07)	41.66 (40.17)	5.00	91.66 (72.23)
11	Iprobenfos 48 EC	5.00 (12.92)	15.00 (22.79)	30.00 (33.21)	43.00 (40.97)	3.00	92.66 (74.32)
12	Kasugamycin 2L	6.66 (14.95)	15.00 (22.79)	30.00 (33.21)	45.00 (42.12)	3.00	90.66 (72.23)
13	Difenconazole + Propiconazole 30 EC	7.33 (15.70)	16.66 (24.08)	33.33 (35.22)	45.00 (42.12)	3.00	84.00 (66.53)
14	Epoxiconazole 7.5 EC	7.00 (15.32)	15.00 (22.79)	33.33 (35.22)	50.00 (45.00)	3.00	91.33 (71.89)
15	Edifenphos 50 EC	6.00 (14.18)	18.33 (25.35)	36.66 (37.22)	51.66 (45.96)	3.00	86.66 (68.60)
16	Difenconazole 25 EC	6.66 (14.95)	25.00 (29.99)	45.00 (42.13)	55.00 (47.88)	3.00	91.33 (72.98)
17	Diniconazole 25 WP	15.00 (22.78)	35.00 (36.27)	76.66 (61.22)	90.00 (71.95)	3.00	92.66 (74.32)
18	Control	18.33 (25.35)	50.00 (45.00)	93.33 (77.71)	100.00 (90.00)	3.00	88.66 (75.81)
SEm±		0.43	1.04	1.54	1.59	0.71	3.69
CD (P=0.05)		1.24	3.00	4.43	4.58	2.03	ns

*Figures in parentheses are arc sin transformed values

on par with isoprothiolane; iprobenfos was on par with edifenphos, kasugamycin and difenconazole. Diniconazole 25 WP (15%) was the least effective and significantly inferior to the rest of the fungicides even though it had shown superior efficacy over the untreated control (18.33%).

At 10 days after inoculation, the disease incidence was nil in the plots treated with pyroquilon 50 WP, azoxystrobin 25 SC, pyraclostrobin 25 EC and RIL 010 25 SC where as the leaf blast severity in the control plot was 50 per cent. Even though, tricyclazole 75 WP and fenoxanil 20 SC had recoded a minimum leaf blast incidence of 0.66 per cent, it was not significant when compared with the above mentioned fungicides. The next higher levels of disease incidence was recorded in propiconazole 25 EC (6.66%) followed by carbendazim 50 WP (7%), carbendazim + epoxiconazole 25 EC (10%), isoprothiolane 40 EC (11.66%), iprobenfos 48 EC (15%), kasugamycin 2 L (15%), epoxiconazole 7.5 EC (15%), difenconazole + propiconazole 30 EC (16.66%), edifenphos 50 EC (18.33%) and difenconazole 25 EC (25%). However, carbendazim + epoxiconazole was statistically on par with isoprothiolane and there was no significant difference among iprobenfos, kasugamycin, difenconazole + propiconazole and epoxiconazole alone. Diniconazole 25 WP (35%) was the least effective and significantly inferior to the rest of the fungicides even though it had shown superior efficacy over the control (50%).

At 15 days after inoculation, pyroquilon 50 WP, azoxystrobin 25 SC and pyraclostrobin 25 EC recorded zero per cent disease incidence, where as the control plot registered 93.33 per cent leaf blast severity. Even though RIL 010 25 SC (0.66%) had recoded the lowest leaf blast incidence, it was not statistically significant when compared

to pyroquilon, azoxystrobin and pyraclostrobin. Tricyclazole 75 WP (3%) was the next best fungicide in the order of efficacy that was found significantly superior over fenoxanil 20 SC (6%) followed by carbendazim 50 WP (16.66%) and propiconazole 25 EC (21.66%). However, carbendazim and propiconazole were statistically on par with each other. Carbendazim + epoxiconazole 25 EC (25%) was the next best treatment followed by isoprothiolane 40 EC (26.66), iprobenfos 48 EC (30%), kasugamycin 2 L (30%), difenconazole + propiconazole 30 EC (33.33%), epoxiconazole 7.5 EC (33.33%), edifenphos 50 EC (36.66%) and difenconazole 25 EC (45%). However, carbendazim + epoxiconazole was statistically on par with isoprothiolane, iprobenfos and kasugamycin; there was no significant difference among difenconazole + propiconazole, epoxiconazole and edifenphos. Diniconazole 25 WP (76.66%) was the least effective and significantly inferior to the rest of the fungicides.

At 20 days after inoculation, pyroquilon 50 WP (4.00), azoxystrobin 25 SC (4.66%), pyraclostrobin 25 EC (5%) and RIL 010 25 SC had offered the most effective control against leaf blast followed by tricyclazole 75 WP (10%) when the leaf blast severity in the untreated control reached the maximum. However, there was no significant difference among pyroquilon, azoxystrobin and RIL 010 where as, tricyclazole was statistically on par with RIL 010. Fenoxanil 20 SC (18.33%) was the next best fungicide in the order of its efficacy that was found significantly superior to carbendazim 50 WP (26.66%) and propiconazole 25 EC (33.33%). However, carbendazim was statistically on par with propiconazole. Carbendazim + epoxiconazole 25 EC (36.66%) followed by isoprothiolane 40 EC (41.66%), iprobenfos 48 EC (43%), kasugamycin 2 L (45%), difenconazole + propiconazole 30 EC (45%), epoxiconazole 7.5 EC (50%), edifenphos 50 EC (51.66%) and difenconazole 25 EC (55%) were the next

best fungicides in the order of their efficacy. However, isoprothiolane was statistically on par with iprobenfos, kasugamycin and difenconazole + propiconazole while epoxiconazole, edifenphos and difenconazole were non-significant with each other. Diniconazole 25 WP (90%) was the least effective and significantly inferior to the rest of the fungicides.

It was evident from Table 17 that it took 3 days for the appearance of initial disease symptoms in the untreated control plot. Pyroquilon 50 WP (16.33 days), azoxystrobin 25 SC (16.33 days), pyraclostrobin 25 EC (16.00 days) and RIL 0101 25 SC had shown the highest efficacy in preventing the leaf blast incidence for not less than 13 days after inoculation and these four fungicides were statistically on par with each other. Tricyclazole 50 WP (10.66 days) and fenoxanil 20 SC (9 days) were successful in preventing the disease incidence for not less than 6 days after inoculation and these two fungicides were statistically on par with each other. The remaining fungicides *viz.* propiconazole 25 EC (5 days), isoprothiolane 40 EC (5 days), iprobenfos 48 EC (3 days), kasugamycin 2 L (3 days), difenconazole + propiconazole 25 EC (3 days), epoxiconazole 7.5 EC (3 days), edifenphos 50 EC (3 days), difenconazole 25 EC (3 days) and diniconazole 25 WP (3 days) were not successful in preventing disease incidence better than the untreated control (3 days) except carbendazim 50 WP and its combination with epoxiconazole (5.33 days).

4.5.1.2 Efficacy of fungicides as seed treatment in the nursery beds during *Kharif*, 2003

The results presented in Table 18 reveal that the fungicides tested were found significantly superior to the untreated control upto 20 DAI. Pyroquilon 50 WP,

azoxystrobin 25 SC, pyraclostrobin 25 EC and RIL 010 25 SC had offered phenomenal control of leaf blast incidence followed by tricyclazole 75 WP and fenoxanil 20 SC. The amount of control exhibited by carbendazim 50 WP and propiconazole 25 EC was also substantial. But the effect of other popular blasticides *viz.* edifenphos 50 EC, iprobenfos 48 EC, isoprothiolane 40 EC and kasugamycin 2 L was found minimal as seed treatment compared to their superior counterparts such as pyroquilon and tricyclazole. The quantum of control offered by rest of the fungicides *viz.* epoxiconazole 7.5 EC and difenconazole 25 EC was found negligible. However, the least effective of all the fungicides tested was diniconazole 25 WP. The effect of fungicides on seed germination in general was found non-significant.

There was absolutely no disease incidence in the plots treated with pyroquilon 50 WP, azoxystrobin 25 SC, pyraclostrobin 25 EC, RIL 010 25 SC and tricyclazole 75 WP while the untreated control plot recorded 21.66 per cent leaf blast severity at 5 DAI. Fenoxanil 20 SC (1.33%) offered the next best control followed by carbendazim 50 WP (3.66%) and propiconazole 25 EC (6.66%), carbendazim + epoxiconazole 25 EC (8.33%), isoprothiolane 40 EC (10%), iprobenfos 48 EC (12.33%), kasugamycin 2 L (13.33%), difenconazole + propiconazole 30 EC (14%), epoxiconazole 7.5 EC (15.33%), edifenphos 50 EC (16%) and difenconazole 25 EC (16%). However, carbendazim + epoxiconazole was statistically on par with propiconazole; difenconazole + propiconazole was on par with kasugamycin and iprobenfos and there was no significant difference among difenconazole, edifenphos and epoxiconazole. Diniconazole 25 WP (18.33%) was the least effective of all the fungicides tested though it was significantly superior to the untreated control.

Table 18: Efficacy of fungicides as seed treatment against rice blast in nursery beds (2003)

S. No.	Name of the fungicide	Leaf blast severity (%)*				Appearance of initial symptoms (days)	Seed germination (%)
		5DAI	10DAI	15DAI	20DAI		
1	Pyroquilon 50 WP	0.00 (0.00)	0.00 (0.00)	3.33 (10.34)	9.33 (17.77)	12	90.00 (71.58)
2	Azoxystrobin 25 SC	0.00 (0.00)	0.00 (0.00)	5.00 (12.92)	10.00 (18.43)	11.33	94.00 (75.95)
3	Pyraclostrobin 25 EC	0.00 (0.00)	0.00 (0.00)	6.00 (14.09)	11.66 (19.89)	10.33	94.00 (75.95)
4	RIL 010 25 SC	0.00 (0.00)	4.33 (11.94)	7.33 (15.60)	16.66 (24.05)	8.00	92.66 (74.40)
5	Tricyclazole 75 WP	0.00 (0.00)	4.33 (11.94)	8.66 (17.02)	18.33 (25.31)	7.66	90.33 (71.91)
6	Fenoxanil 20 SC	1.33 (5.42)	5.00 (12.49)	12.33 (20.50)	31.66 (34.24)	5.00	90.66 (72.23)
7	Carbendazim 50 WP	3.66 (11.02)	9.66 (18.05)	21.66 (27.71)	38.33 (38.22)	4.00	93.33 (75.28)
8	Propiconazole 25 EC	6.66 (14.95)	20.33 (26.80)	30.00 (33.21)	46.66 (43.09)	4.00	91.33 (73.04)
9	Carbendazim + Epoxiconazole 25 EC	8.33 (16.74)	21.66 (27.71)	40.00 (39.21)	51.66 (45.97)	3.00	92.66 (74.32)
10	Isoprothiolane 40 EC	10.00 (18.43)	21.66 (27.71)	41.66 (40.20)	51.66 (45.97)	3.00	90.00 (71.62)
11	Iprobenfos 48 EC	12.33 (20.50)	22.33 (28.18)	45.00 (42.12)	55.00 (47.91)	3.00	91.33 (72.98)
12	Kasugamycin 2L	13.33 (21.41)	28.33 (32.16)	45.00 (42.12)	58.33 (49.80)	3.00	90.00 (71.62)
13	Difenconazole + Propiconazole 30 EC	14.00 (21.95)	30.00 (33.21)	46.66 (43.09)	63.33 (52.78)	3.00	90.66 (72.37)
14	Epoxiconazole 7.5 EC	15.33 (23.05)	30.00 (33.21)	55.00 (47.88)	68.33 (55.77)	3.00	90.00 (71.62)
15	Edifenphos 50 EC	16.00 (23.57)	33.33 (35.25)	56.66 (48.84)	73.33 (58.93)	3.00	86.00 (70.07)
16	Difenconazole 25 EC	16.00 (23.57)	33.33 (35.25)	60.00 (50.79)	76.66 (61.22)	3.00	88.66 (71.35)
17	Diniconazole 25 WP	18.33 (25.34)	38.33 (38.22)	73.33 (59.05)	83.33 (66.26)	3.00	92.00 (74.10)
18	Control	21.66 (27.73)	53.33 (46.91)	83.33 (66.14)	100.00 (90.00)	3.00	93.00 (74.73)
SEm±		0.73	1.00	1.30	1.51	0.33	2.45
CD (P=0.05)		2.11	2.87	3.73	4.35	0.96	ns

*Figures in parentheses are arc sin transformed values

The disease incidence was nil in the plots treated with pyroquilon 50 WP, azoxystrobin 25 SC and pyraclostrobin 25 EC while the untreated control plot registered 53.33 per cent leaf blast severity at 10 DAI. RIL 010 25 SC (4.33%) provided the next best control along with tricyclazole 75 WP (4.33%) and fenoxanil 20 SC (5%) that were significantly superior over rest of the fungicides except pyroquilon, azoxystrobin and pyraclostrobin. Carbendazim 50 WP (9.66%) was next in the order of its efficacy followed by propiconazole 25 EC (20.33%), carbendazim + epoxiconazole 25 EC (21.66%), isoprothiolane 40 EC (21.66%), iprobenfos 48 EC (22.33%), kasugamycin 2 L (28.33%), difenconazole + propiconazole 30 EC (30%), epoxiconazole 7.5 EC (30%), edifenphos 50 EC (33.33%) and difenconazole 25 EC (33.33%). However, iprobenfos, isoprothiolane, carbendazim + epoxiconazole and propiconazole were statistically on par with each other. There was no significant difference among difenconazole, edifenphos, epoxiconazole and difenconazole + propiconazole. Diniconazole 25 WP (38.33%) was the least effective of all the fungicides tested though it was significantly superior to the untreated control.

The per cent disease severity ranged between 3.33 (pyroquilon 50 WP) and 83.33 (untreated control) at 15 DAI. Pyroquilon 50 WP (3.33%) and azoxystrobin 25 SC (5%) had given the most efficient control that was significantly superior to rest of the fungicides except pyraclostrobin 25 EC (6%) and RIL 010 25 SC (7.33%). Tricyclazole 75 WP (8.66%) was the next best fungicide in the order of its efficacy followed by fenoxanil 20 SC (12.33%), carbendazim 50 WP (21.66%) and propiconazole 25 EC (30%). However, there was no significant difference among pyraclostrobin, RIL 010 and tricyclazole while tricyclazole in turn was on par with fenoxanil, but significantly superior to carbendazim. Carbendazim + epoxiconazole 25 EC (40%), isoprothiolane 40 EC (41.66%), iprobenfos 48 EC (45%), kasugamycin 2 L (45%), difenconazole +

propiconazole 30 EC (46.66%), epoxiconazole 7.5 EC (55%), edifenphos 50 EC (56.66%) and difenconazole 25 EC (60%) were in the decreasing order of their efficacy. However, isoprothiolane, iprobenfos, kasugamycin and difenconazole + propiconazole were statistically on par with each other. Similarly, there was no significant difference among epoxiconazole, edifenphos and difenconazole. Diniconazole 25 WP (73.33%) was the least effective of all the fungicides tested.

At 20 DAI, the per cent leaf blast severity varied between 9.33 (pyroquilon 50 WP) and 100 per cent (untreated control). Pyroquilon 50 WP (9.33%), azoxystrobin 25 SC (10%) and pyraclostrobin 25 EC (11.66%) were found statistically on par with each other and exhibited excellent control compared to other fungicides. RIL 010 25 SC (16.66%) and tricyclazole 75 WP (18.33%) were the next best in their efficacy that was significantly superior to fenoxanil 20 SC (31.66%) and carbendazim 50 WP (38.33%). However, there was no significant difference between RIL010 and tricyclazole; fenoxanil and carbendazim, respectively. Propiconazole 25 EC (46.66%) was the next in the order of its efficacy followed by carbendazim + epoxiconazole 25 EC (51.66%), isoprothiolane 40 EC (51.66%), iprobenfos 48 EC (55%), kasugamycin 2 L (58.33%), difenconazole + propiconazole 30 EC (63.33%), epoxiconazole 7.5 EC (68.33%), edifenphos 50 EC (73.33%), difenconazole 25 EC (76.66%) and diniconazole 25 WP (83.33%). However, carbendazim + epoxiconazole, isoprothiolane, iprobenfos and kasugamycin were statistically on par with each other. There was no significant difference between difenconazole + propiconazole and epoxiconazole. Edifenphos was statistically on par with epoxiconazole as well as difenconazole. Diniconazole was found the least effective at 20 DAI also.

The results presented in Table 18, showed that the number of days taken for the appearance of initial disease symptoms was varied between 3 days and 12 days. Pyroquilon 50 WP (12 days) and azoxystrobin 25 SC (11.33 days) prevented the disease incidence for at least 8 to 9 days compared to the untreated control (3 days). Pyraclostrobin 25 EC (10.33 days) was successful in preventing the disease incidence for about 7 days and it was significantly superior to RIL 010 25 SC (8 days) and tricyclazole 75 WP (7.66 days). However, RIL 010 and tricyclazole are statistically on par with each other. Fenoxanil 20 SC (5 days) was the next best performer that was superior to both carbendazim 50 WP (4 days) and propiconazole 25 EC (4 days). The initial disease symptoms appeared in the plots treated with rest of the fungicides *viz.* carbendazim + epoxiconazole 25 EC, isoprothiolane 40 EC, iprobenfos 48 EC, kasugamycin 2 L, difenconazole + propiconazole 30 EC, epoxiconazole 7.5 EC, edifenphos 50 EC, difenconazole 25 EC and diniconazole 25 WP on the 3rd day itself as it happened in the untreated control (3 days).

4.5.1.3 Efficacy of fungicides as seed treatment in the nursery beds during (*Kharif*, 2002 & 2003; pooled data)

The results presented in Table 19 reveal that all the fungicides tested were significantly superior to untreated control upto 20 DAI. Pyroquilon 50 WP, azoxystrobin 25 SC, pyraclostrobin 25 EC and RIL 010 25 SC had given phenomenal control of leaf blast incidence followed by tricyclazole 75 WP and fenoxanil 20 SC. The quantum of control exhibited by carbendazim 50 WP and propiconazole was also substantial. But the effect of other popular blasticides *viz.* edifenphos 50 EC, iprobenfos 48 EC, isoprothiolane 40 EC and kasugamycin 2 L was found minimal as seed treatment when compared to their superior counterparts such as pyroquilon and tricyclazole. The control

offered by rest of the fungicides *viz.* epoxiconazole 7.5 EC and difenconazole 25 EC was negligible. The least effective of all the fungicides tested was diniconazole 25 WP. The effect of the fungicides on seed germination in general was non-significant.

At 5 DAI, the per cent leaf blast severity varied between zero and 19.99 while there was absolutely no disease incidence in the plots treated with pyroquilon 50 WP, azoxystrobin 25 SC, pyraclostrobin 25 EC, RIL 010 25 SC and tricyclazole 75 WP. Untreated control recorded the highest per cent leaf blast severity of 21.66 at 5 DAI. Fenoxanil 20 SC (0.66%) was the next best fungicide followed by carbendazim 50 WP (2.49%), propiconazole 25 EC (4.16%), carbendazim + epoxiconazole 25 EC (5.33%), isoprothiolane 40 EC (6.33%), iprobenfos 48 EC (8.66%), kasugamycin 2 L (9.99%), difenconazole + propiconazole 30 EC (10.66%), edifenphos 50 EC (11%), epoxiconazole 7.5 EC (11.16%) and difenconazole 25 EC (11.33%). However, fenoxanil, carbendazim and propiconazole differed significantly with one another while there was no significant difference among kasugamycin, difenconazole + propiconazole, epoxiconazole, edifenphos and difenconazole. Diniconazole 25 WP (16.66%) was the least effective of all the fungicides tested even though it was significantly superior to the untreated control (19.99%).

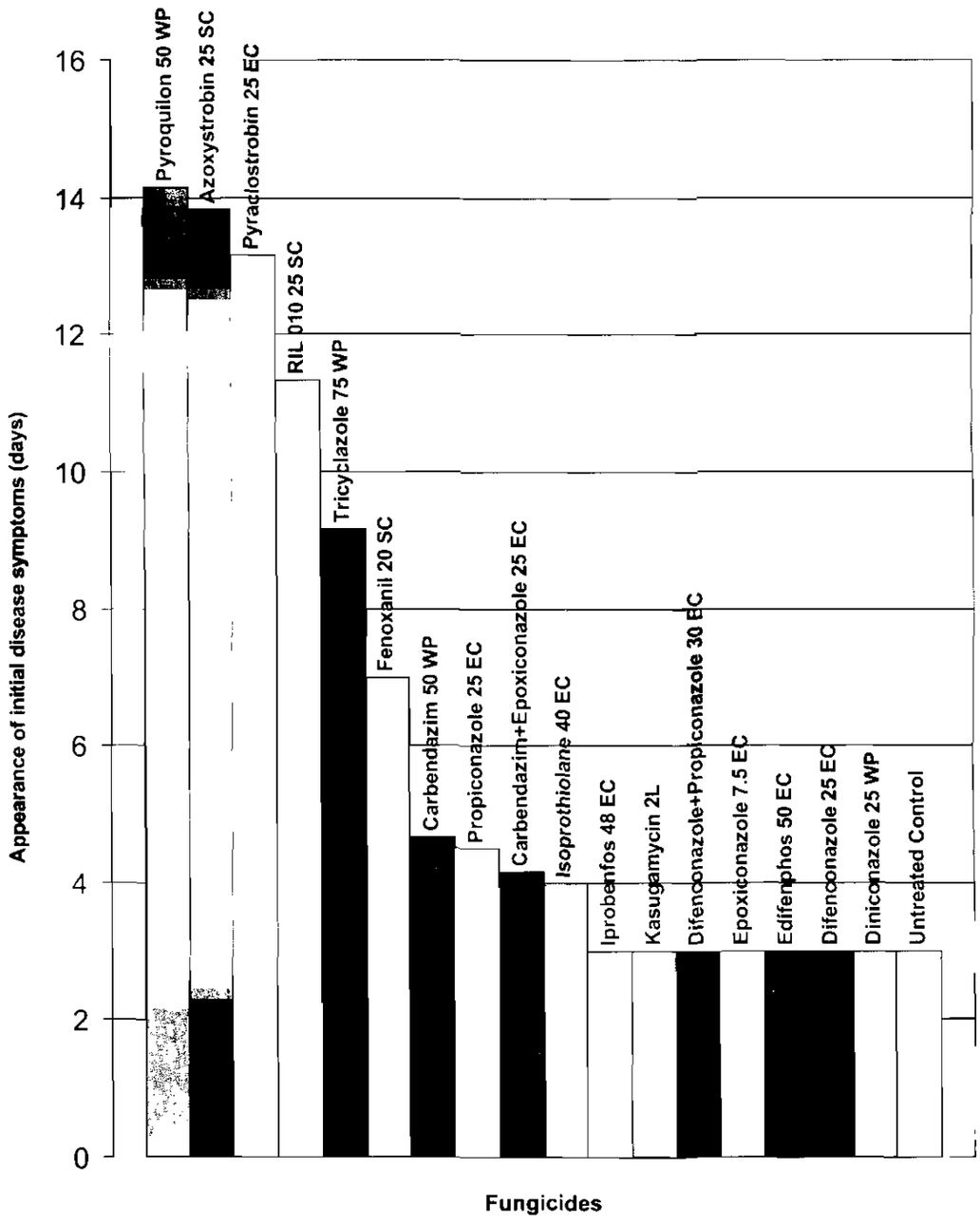
The per cent disease incidence ranged between zero and 51.66 and the disease incidence was nil in the plots treated with pyroquilon 50 WP, azoxystrobin 25 SC and pyraclostrobin 25 EC at 10 DAI. Untreated control registered the highest leaf blast severity of 51.66 at 10 DAI. RIL 010 25 SC (2.16%) had provided the next best control along with tricyclazole 75 WP (2.49%) and fenoxanil 20 SC (2.83%) which was significantly superior over rest of the fungicides except pyroquilon, azoxystrobin and pyraclostrobin. Carbendazim 50 WP (8.33%) was next in the order of its efficacy

Table 19: Efficacy of fungicides as seed treatment against rice blast in nursery beds (2002 & 2003; pooled data)

S. No.	Name of the fungicide	Leaf blast severity (%)*				Appearance of initial symptoms (days)	Seed germination (%)
		5DAI	10DAI	15DAI	20DAI		
1	Pyroquilon 50 WP	0.00 (0.00)	0.00 (0.00)	1.66 (7.49)	6.66 (15.00)	14.16	89.66 (71.28)
2	Azoxystrobin 25 SC	0.00 (0.00)	0.00 (0.00)	2.50 (9.10)	7.33 (15.68)	13.83	95.16 (77.34)
3	Pyraclostrobin 25 EC	0.00 (0.00)	0.00 (0.00)	3.00 (9.98)	8.33 (16.74)	13.16	93.83 (75.58)
4	RIL 010 25 SC	0.00 (0.00)	2.16 (8.53)	3.99 (11.54)	11.16 (19.55)	11.33	93.33 (75.00)
5	Tricyclazole 75 WP	0.00 (0.00)	2.49 (9.10)	5.83 (13.94)	14.16 (22.14)	9.16	89.49 (71.09)
6	Fenoxanil 20 SC	0.66 (4.66)	2.83 (9.63)	9.49 (17.95)	24.99 (30.00)	7.00	91.99 (73.57)
7	Carbendazim 50 WP	2.49 (9.10)	8.33 (16.74)	19.16 (25.99)	32.49 (34.76)	4.66	93.49 (75.23)
8	Propiconazole 25 EC	4.16 (11.83)	13.49 (21.56)	25.83 (30.53)	39.99 (39.23)	4.50	91.66 (73.26)
9	Carbendazim + Epoxiconazole 25 EC	5.33 (13.31)	15.83 (23.42)	32.50 (34.76)	44.16 (41.67)	4.16	90.66 (72.24)
10	Isoprothiolane 40 EC	6.33 (14.54)	16.66 (24.12)	34.16 (35.79)	46.66 (43.11)	4.00	90.83 (72.34)
11	Iprobenfos 48 EC	8.66 (17.16)	18.66 (25.62)	37.50 (37.76)	49.00 (44.43)	3.00	91.99 (73.57)
12	Kasugamycin 2L	9.99 (18.44)	21.66 (27.76)	37.50 (37.76)	51.66 (45.97)	3.00	90.33 (71.85)
13	Difenconazole + Propiconazole 30 EC	10.66 (19.09)	23.33 (28.86)	39.99 (39.23)	54.16 (47.41)	3.00	87.33 (69.12)
14	Epoxiconazole 7.5 EC	11.16 (19.55)	22.50 (28.32)	44.16 (41.67)	59.16 (50.30)	3.00	90.66 (72.24)
15	Edifenphos 50 EC	11.00 (19.37)	25.83 (30.53)	46.66 (43.11)	62.49 (52.24)	3.00	86.33 (68.28)
16	Difenconazole 25 EC	11.33 (19.64)	29.16 (32.71)	52.50 (46.43)	65.83 (54.21)	3.00	89.99 (71.56)
17	Diniconazole 25 WP	16.66 (24.12)	36.66 (37.29)	74.99 (60.00)	86.66 (68.61)	3.00	92.33 (73.89)
18	Control	19.99 (26.56)	51.66 (45.97)	88.33 (70.00)	100.00 (90.00)	3.00	90.83 (72.34)
SEm±		0.52	0.62	1.14	1.06	0.44	2.06
CD (P=0.05)		1.50	1.78	3.28	3.06	1.27	ns

*Figures in parentheses are arc sin transformed values

Fig. 19: Development of initial disease symptoms in the seed treated nursery beds (*Kharif*, 2002&03)



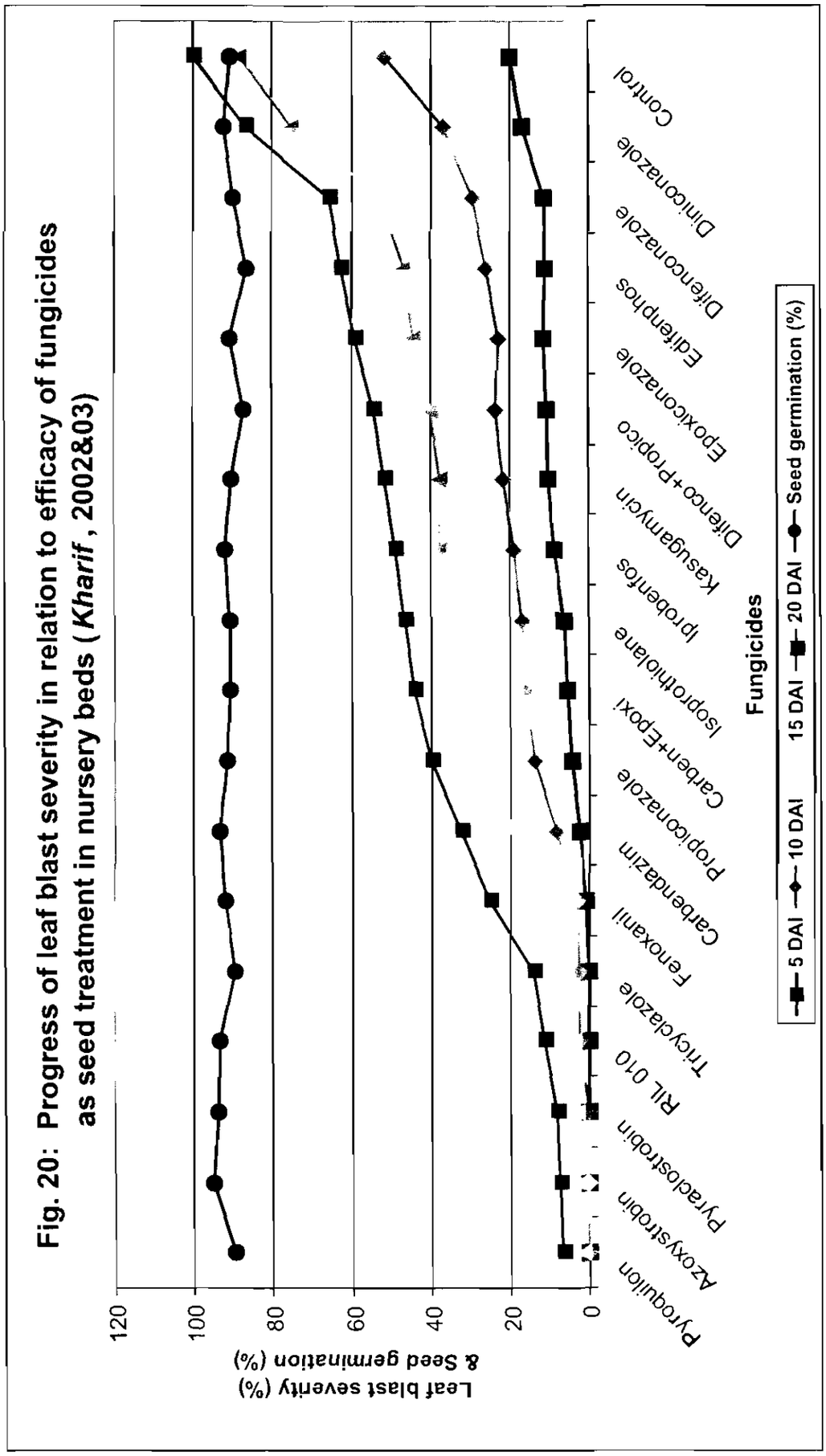


Fig. 20: Progress of leaf blast severity in relation to efficacy of fungicides as seed treatment in nursery beds (*Kharif*, 2002&03)



Plate 29a: Evaluation of fungicides as seed treatment in the nursery beds



Plate 29b: Evaluation of fungicides as seed treatment in the nursery beds (contd.)

followed by propiconazole 25 EC (13.49%), carbendazim + epoxiconazole 25 EC (15.83%), isoprothiolane 40 EC (16.66%), iprobenfos 48 EC (18.66%), kasugamycin 2 L (21.66%), difenconazole + propiconazole 30 EC (23.33%), epoxiconazole 7.5 EC (22.5%), edifenphos 50 EC (25.83%) and difenconazole 25 EC (29.16%). However, propiconazole, carbendazim + epoxiconazole and isoprothiolane were statistically on par with each other, but significantly inferior to carbendazim 50 WP. Kasugamycin, difenconazole + propiconazole and epoxiconazole were statistically on par with each other, but significantly inferior to iprobenfos. Edifenphos, difenconazole and diniconazole differed significantly with one another in the order of their efficacy. And diniconazole 25 WP (36.66%) was found the least effective of all the fungicides tested though it was significantly superior to the untreated control (51.66%).

The per cent disease severity ranged between 1.66 (pyroquilon 50 WP) and 88.33 (untreated control) at 15 DAI. Pyroquilon 50 WP (1.66%), azoxystrobin 25 SC (2.5%), pyraclostrobin 25 EC (3%), RIL 010 25 SC (3.99%) and tricyclazole 75 WP (5.83%) had exhibited superior control over rest of the fungicides. However, pyroquilon was statistically on par with azoxystrobin and pyraclostrobin while there was no significant difference between pyraclostrobin and RIL 010. Similarly, there was no significant difference between RIL 010 and tricyclazole. Fenoxanil 20 SC (9.49%) was the next best fungicide in the order of its efficacy followed by carbendazim 50 WP (19.16%) and propiconazole 25 EC (25.83%) and were statistically on par with one another, but significantly superior to the remaining fungicides. Carbendazim + epoxiconazole 25 EC (32.5%), isoprothiolane 40 EC (34.16%), iprobenfos 48 EC (37.5%), kasugamycin 2 L (37.5%), difenconazole + propiconazole 30 EC (39.99%), epoxiconazole 7.5 EC (44.16%), edifenphos 50 EC (46.66%) and difenconazole 25 EC (52.5%) were in the decreasing order of their efficacy. However, carbendazim +

epoxiconazole, isoprothiolane, iprobenfos and kasugamycin were statistically on par with each other while there was no significant difference among difenconazole + propiconazole, epoxiconazole and edifenphos. Diniconazole 25 WP (74.99%) was the least effective of all the fungicides tested even though it was superior over the untreated check (88.33%).

At 20 DAI, the per cent leaf blast severity varied from 6.66 (pyroquilon 50 WP) to 100 per cent (untreated control). Pyroquilon 50 WP (6.66%), azoxystrobin 25 SC (7.33%) and pyraclostrobin 25 EC (8.33%) were statistically on par with each other and exhibited excellent control compared to rest of the fungicides. RIL 010 25 SC (11.16%) and tricyclazole 75 WP (14.16%) were the next best fungicides that were significantly superior to fenoxanil 20 SC (24.99%) and carbendazim 50 WP (32.49%). However, RIL010 was statistically on par with tricyclazole as well as pyraclostrobin while fenoxanil was found significantly superior to carbendazim 50 WP. Propiconazole 25 EC (39.99%) was the next in the order of its efficacy followed by carbendazim + epoxiconazole 25 EC (44.16%), isoprothiolane 40 EC (46.66%), iprobenfos 48 EC (49%), kasugamycin 2 L (51.66%), difenconazole + propiconazole 30 EC (54.16%), epoxiconazole 7.5 EC (59.16%), edifenphos 50 EC (62.49%), difenconazole 25 EC (65.83%) and diniconazole 25 WP (86.66%). However, there was no significant difference among carbendazim + epoxiconazole, isoprothiolane and iprobenfos; isoprothiolane, iprobenfos and kasugamycin; iprobenfos, kasugamycin and difenconazole + propiconazole, respectively. Where as, propiconazole and carbendazim + epoxiconazole; difenconazole + propiconazole and epoxiconazole; edifenphos and difenconazole had not differed significantly between them, respectively. Diniconazole (86.66%) was the least effective of all the fungicides tested even though it was significantly superior to the untreated control (100%).

epoxiconazole, isoprothiolane, iprobenfos and kasugamycin were statistically on par with each other while there was no significant difference among difenconazole + propiconazole, epoxiconazole and edifenphos. Diniconazole 25 WP (74.99%) was the least effective of all the fungicides tested even though it was superior over the untreated check (88.33%).

At 20 DAI, the per cent leaf blast severity varied from 6.66 (pyroquilon 50 WP) to 100 per cent (untreated control). Pyroquilon 50 WP (6.66%), azoxystrobin 25 SC (7.33%) and pyraclostrobin 25 EC (8.33%) were statistically on par with each other and exhibited excellent control compared to rest of the fungicides. RIL 010 25 SC (11.16%) and tricyclazole 75 WP (14.16%) were the next best fungicides that were significantly superior to fenoxanil 20 SC (24.99%) and carbendazim 50 WP (32.49%). However, RIL010 was statistically on par with tricyclazole as well as pyraclostrobin while fenoxanil was found significantly superior to carbendazim 50 WP. Propiconazole 25 EC (39.99%) was the next in the order of its efficacy followed by carbendazim + epoxiconazole 25 EC (44.16%), isoprothiolane 40 EC (46.66%), iprobenfos 48 EC (49%), kasugamycin 2 L (51.66%), difenconazole + propiconazole 30 EC (54.16%), epoxiconazole 7.5 EC (59.16%), edifenphos 50 EC (62.49%), difenconazole 25 EC (65.83%) and diniconazole 25 WP (86.66%). However, there was no significant difference among carbendazim + epoxiconazole, isoprothiolane and iprobenfos; isoprothiolane, iprobenfos and kasugamycin; iprobenfos, kasugamycin and difenconazole + propiconazole, respectively. Where as, propiconazole and carbendazim + epoxiconazole; difenconazole + propiconazole and epoxiconazole; edifenphos and difenconazole had not differed significantly between them, respectively. Diniconazole (86.66%) was the least effective of all the fungicides tested even though it was significantly superior to the untreated control (100%).

The results presented in Table 19, showed that the number days taken for the appearance of initial disease symptoms was varied between 3 days and 12 days. Pyroquilon 50 WP (14.16 days), azoxystrobin 25 SC (13.83 days) and pyraclostrobin 25 EC (13.16 days) had shown the best performance in preventing the disease incidence for at least 10 to 11 days compared to the untreated control (3 days). RIL 010 25 SC (11.33 days) was successful in preventing the disease incidence for about ten days and it was significantly superior to tricyclazole 75 WP (9.16 days) and fenoxanil (7 days). However, tricyclazole was found significantly superior to fenoxanil. Carbendazim (4.66 days), propiconazole (4.5 days), carbendazim + epoxiconazole (4.16 days) and isoprothiolane (4 days) were found statistically on par with each other and could prevent to disease incidence for more than a day. The initial disease symptoms appeared in the plots treated with rest of the fungicides *viz.* iprobenfos 48 EC, kasugamycin 2 L, difenconazole + propiconazole 30 EC, epoxiconazole 7.5 EC, edifenphos 50 EC, difenconazole 25 EC and diniconazole 25 WP on the 3rd day itself as it happened in the untreated control (3 days).

4.5.2 Efficacy of fungicides as foliar spray in the nursery beds

The experiments were conducted to test the efficacy of various fungicides as foliar spray against rice blast incidence in the nursery beds for two consecutive seasons *viz. Kharif*, 2002 and *Kharif*, 2003. The details of the fungicides used have been presented in Table 2. Observations on leaf blast severity were recorded at 5 day interval starting from 5 DAI to 20 DAI (when the leaf blast severity in the control plot reached the maximum). The results of *Kharif*, 2002; *Kharif*, 2003 and the pooled data of *Kharif*, 2002 & 2003 are presented in Tables 20, 21 and 22.

4.5.2.1 Efficacy of fungicides as foliar spray in the nursery beds during *Kharif*, 2002

The data furnished in Table 20 disclose that all the fungicides tested as foliar spray against rice blast were found significantly superior over the control upto 20 days after inoculation. The efficacy of pyroquilon 50 WP, azoxystrobin 25 SC, RIL 010 25 SC, pyraclostrobin 25 EC and tricyclazole 75 WP was excellent. Fenoxanil 20 EC, *iprobenfos 48 EC, isoprothiolane 40 EC, edifenphos 50 EC, propiconazole 25 EC* and kasugamycin 2 L were also found superior in controlling leaf blast incidence. Carbendazim 50 WP exhibited good amount of control compared to rest of the fungicides. Diniconazole 25 WP was the least effective of all the fungicides tested even though it was significantly superior over control. The fungicides were sprayed in the nursery beds after the appearance of initial disease symptoms. The per cent leaf blast severity in all the experimental plots was found non-significant prior to the fungicidal spray.

At 5 DAI, pyroquilon 50 WP (1.33%), azoxystrobin 25 SC (1.33%), RIL 010 25 SC (1.66%) and pyraclostrobin 25 EC (1.33%) exhibited the most effective control as foliar spray against leaf blast incidence. Tricyclazole 75 WP (3.33%) and fenoxanil 20 SC (3.66%) were the next best fungicides in the order of their efficacy followed by iprobenfos 48 EC (4.33%), isoprothiolane 40 EC (4.66%), edifenphos 50 EC (5%), propiconazole 25 EC (5%) and kasugamycin 2 L (5.33%). However, isoprothiolane, edifenphos, propiconazole and kasugamycin were statistically on par with each other. Iprobenfos was found on par with fenoxanil as well as isoprothiolane. Difenconazole + propiconazole 30 EC (6.66%) had come next in the order of its efficacy followed by carbendazim + epoxiconazole 25 EC (7.33%), carbendazim 50 WP (8.33%)

Table 20: Efficacy of fungicides as foliar spray against rice blast in nursery beds (2002)

S. No.	Name of the fungicide	Leaf blast severity (%) Before spray	Leaf blast severity (%)*			
			5DAI	10DAI	15DAI	20DAI
1	Pyroquilon 50 WP	0.37 (3.44)	1.33 (6.62)	4.66 (12.46)	6.33 (14.53)	9.66 (18.11)
2	Azoxystrobin 25 SC	0.33 (3.25)	1.33 (6.62)	6.33 (14.57)	9.66 (18.11)	14.33 (22.24)
3	RIL 010 25 SC	0.43 (3.75)	1.66 (7.40)	7.33 (15.71)	10.66 (19.05)	15.66 (23.23)
4	Pyraclostrobin 25 EC	0.40 (3.54)	1.33 (6.62)	7.66 (16.06)	11.33 (19.66)	17.66 (24.79)
5	Tricyclazole 75 WP	0.43 (3.77)	3.33 (10.51)	10.33 (18.74)	15.65 (23.23)	19.33 (26.07)
6	Fenoxanil 20 SC	0.43 (3.69)	3.66 (10.94)	11.65 (19.96)	17.33 (24.59)	26.65 (31.01)
7	Iprobenfos 48 EC	0.33 (3.25)	4.33 (12.01)	13.66 (21.57)	22.33 (28.20)	27.66 (31.73)
8	Isoprothiolane 40 EC	0.40 (3.63)	4.66 (12.46)	14.33 (22.24)	23.66 (29.10)	30.00 (33.21)
9	Edifenphos 50 EC	0.37 (3.46)	5.00 (12.73)	15.00 (22.78)	26.66 (31.08)	31.33 (34.04)
10	Propiconazole 25 EC	0.43 (3.77)	5.00 (12.92)	15.33 (23.04)	27.00 (31.30)	32.33 (34.64)
11	Kasugamycin 2L	0.40 (3.54)	5.33 (13.29)	15.66 (23.31)	27.33 (31.52)	33.66 (35.46)
12	Carbendazim 50 WP	0.40 (3.63)	8.33 (16.76)	20.33 (26.80)	29.33 (32.79)	38.66 (38.66)
13	Difenconazole + Propiconazole 30 EC	0.27 (2.92)	6.66 (14.95)	18.66 (25.59)	32.00 (34.45)	39.66 (38.93)
14	Carbendazim + Epoxiconazole 25 EC	0.33 (3.30)	7.33 (15.71)	19.33 (26.08)	33.33 (35.26)	41.33 (40.01)
15	Epoxiconazole 7.5 EC	0.37 (3.41)	10.33 (18.75)	23.33 (28.88)	38.33 (38.25)	56.66 (48.91)
16	Difenconazole 25 EC	0.33 (3.30)	11.00 (19.37)	25.66 (30.36)	41.66 (40.10)	59.33 (50.50)
17	Diniconazole 25 WP	0.37 (3.41)	13.66 (21.69)	34.33 (35.87)	49.66 (44.81)	67.66 (55.43)
18	Control	0.30 (3.11)	24.66 (29.77)	66.66 (54.73)	97.32 (81.10)	100.00 (90.00)
SEm±		0.35	0.38	0.40	0.73	1.60
CD (P=0.05)		ns	1.10	1.15	2.11	4.61

*Figures in parentheses are arc sin transformed values

alone, epoxiconazole 7.5 EC (10.33%) and difenconazole 25 EC (11%). However, carbendazim 50 WP alone was as effective as its combination with epoxiconazole and there was no significant difference between carbendazim + epoxiconazole and difenconazole + propiconazole. Similarly, epoxiconazole and difenconazole were also statistically on par with each other. Diniconazole 25 WP (13.66%) was the least effective of all the fungicides tested even though it was superior to untreated control (24.66%).

At 10 DAI, pyroquilon 50 WP (4.66%) exhibited phenomenal control compared to other fungicides while the untreated control plot recorded 66.66 per cent leaf blast severity. Azoxystrobin 25 SC (6.33%) and RIL 010 25 SC (7.33%) were the next best fungicides in the order of their efficacy followed by pyraclostrobin 25 EC (7.66%), tricyclazole 75 WP (10.33%), fenoxanil 20 SC (11.65%), iprobenfos 48 EC (13.66%), isoprothiolane 40 EC (14.33%), edifenphos 50 EC (15%), propiconazole 25 EC (15.33%) and kasugamycin 2 L (15.66%). There was no significant difference between tricyclazole and fenoxanil. Edifenphos was statistically on par with propiconazole and kasugamycin while iprobenfos was as good as isoprothiolane and fenoxanil. Difenconazole + propiconazole 30 EC (18.66%) had come next in the order of its efficacy followed by carbendazim + epoxiconazole 25 EC (19.33%), carbendazim 50 WP (20.33%), epoxiconazole 7.5 EC (23.33%) and difenconazole 25 EC (25.66%). There was no significant difference between carbendazim alone and its combination with epoxiconazole where as epoxiconazole alone is significantly superior to difenconazole that in turn superior to diniconazole. Diniconazole 25 WP (34.33%) was the least effective of all the fungicides tested, but it had exhibited far better efficacy when compared to the untreated control (66.66%).

At 15 DAI, pyroquilon 50 WP (6.33%), azoxystrobin 25 SC (9.66%), RIL 010 25 SC (10.66%) and pyraclostrobin 25 EC (11.33%) were the most efficient over rest of the fungicides while the untreated control plot registered 97.32 per cent leaf blast severity. Pyroquilon was significantly superior to azoxystrobin, RIL 010 and pyraclostrobin that in turn were statistically on par with each other. Tricyclazole 75 WP (15.65%) and fenoxanil 20 SC (17.33%) exhibited the next best control followed by iprobenfos 48 EC (22.33%), isoprothiolane 40 EC (23.66%), edifenphos 50 EC (26.66%), propiconazole 23 EC (27%), kasugamycin 2 L (27.33%), carbendazim 50 WP (29.33%), difenconazole + propiconazole 30 EC (32%), carbendazim + epoxiconazole 25 EC (33.33%), epoxiconazole 7.5 EC (38.33%) and difenconazole 25 EC (41.66%). However, tricyclazole was found as good as fenoxanil and there was no significant difference among edifenphos, propiconazole, kasugamycin and carbendazim. Iprobenfos was statistically on par with fenoxanil as well as isoprothiolane that in turn was on par with edifenphos. Similarly, difenconazole + propiconazole and carbendazim + epoxiconazole were statistically on par with each other, but significantly superior to epoxiconazole. Difenconazole was significantly superior to diniconazole but inferior to epoxiconazole. Finally, diniconazole 25 WP (49.66%) was the least effective of all the fungicides tested even though it had offered significantly superior efficacy over the untreated control (97.32%).

AT 20 DAI, pyroquilon 50 WP (9.66%), azoxystrobin 25 SC (14.33%), RIL 010 (15.66%), pyraclostrobin 25 EC (17.66%) and tricyclazole 75 WP (19.33%) were the most efficient over rest of the fungicides where as the control plot recorded cent per cent leaf blast severity. Azoxystrobin was found on par with pyroquilon and there was no significant difference among azoxystrobin, RIL 010, pyraclostrobin and

tricyclazole. Fenoxanil 20 SC (26.65%) was the next best fungicide in the order of its efficacy followed by iprobenfos 48 EC (27.66%), isoprothiolane 40 EC (30%), edifenphos 50 EC (31.33%), propiconazole 25 EC (32.33%) and kasugamycin 2 L (33.66%) that were statistically on par with each other, but inferior to tricyclazole. Carbendazim 50 WP (38.66%), difenconazole + propiconazole 30 EC (39.66%) and carbendazim + epoxiconazole 25 EC (41.33%) were found on par with each other, but significantly superior to epoxiconazole 7.5 EC (56.66%) and difenconazole 25 EC (59.33%). Diniconazole 25 WP (67.66) was the least effective of all the fungicides tested even though it had exhibited greater efficacy compared to the untreated control.

4.5.2.2 Efficacy of fungicides as foliar spray in the nursery beds during *Kharif*, 2003

The data furnished in Table 21 reveal that all the fungicides tested as foliar spray against rice blast were found significantly superior over the control up to 20 days after inoculation. The efficacy of pyroquilon 50 WP, azoxystrobin 25 SC, RIL 010 25 SC, pyraclostrobin 25 EC and tricyclazole 75 WP against rice blast was excellent. Fenoxanil 20 EC, iprobenfos 48 EC, isoprothiolane 40 EC, edifenphos 50 EC, propiconazole 25 EC and kasugamycin 2 L were also found superior in reducing leaf blast incidence. Carbendazim 50 WP exhibited good control of leaf blast compared to rest of the fungicides. Diniconazole 25 WP was the least effective of all the fungicides tested even though it was significantly superior over untreated control. The fungicides were sprayed in the nursery beds after the appearance of initial disease symptoms. The per cent leaf blast severity in all the experimental plots was found non-significant prior to the fungicidal spray.

The per cent leaf blast severity at 5 DAI varied from 2.00 (pyroquilon 50 WP) to 28.66 (untreated control). Pyroquilon 50 WP (2%) exhibited superior efficacy as foliar spray in the nursery beds followed by azoxystrobin 25 SC (2.66%), RIL 010 25 SC (3.33%), pyraclostrobin 25 EC (3.66%), tricyclazole 75 WP (5.65%) and fenoxanil 20 SC (5.65%). There was no significant difference between RIL 010 and pyraclostrobin; tricyclazole and fenoxanil, respectively. Iprobenfos 48 EC (6.33%) was the next best fungicide in the order of its efficacy followed by isoprothiolane 40 EC (7%), propiconazole 25 EC (7%), edifenphos 50 EC (7.33%) and kasugamycin 2 L (7.66%). However, there was no significant difference among isoprothiolane, edifenphos and propiconazole and these were significantly superior to kasugamycin while isoprothiolane was statistically on par with iprobenfos. Difenconazole + propiconazole 30 EC (9%) was the next in the order of its efficacy followed by carbendazim + epoxiconazole 25 EC (9.66%), carbendazim 50 WP (10%), epoxiconazole 7.5 EC (12%) and difenconazole 25 EC (13.66%). However, there was no significant difference between difenconazole + propiconazole and carbendazim + epoxiconazole, respectively, where as carbendazim 50 WP alone was on par with its combination with epoxiconazole. Epoxiconazole 7.5 EC was significantly superior to difenconazole 25 EC, but inferior to carbendazim 50 WP. Diniconazole 25 WP (15.33%) was the least effective of all the fungicides tested even though it was significantly superior to the untreated control (28.66%).

The per cent leaf blast severity at 10 DAI varied from 5.33 (pyroquilon 50 WP) to 73.66 (untreated control). Pyroquilon 50 WP (5.33%), azoxystrobin 25 SC (6.66%), RIL 010 25 SC (7%) and pyraclostrobin 25 EC (7.33%) were found statistically on par with each other and exhibited significantly superior control over rest of the fungicides. Tricyclazole 75 WP (9.65%) and fenoxanil 20 SC (11%) had offered the next

Table 21: Efficacy of fungicides as foliar spray against rice blast in nursery beds (2003)

S. No.	Name of the fungicide	Leaf blast severity (%) Before spray	Leaf blast severity (%)*			
			5DAI	10DAI	15DAI	20DAI
1	Pyroquilon 50 WP	0.70 (4.78)	2.00 (8.13)	5.33 (13.35)	7.60 (16.00)	10.33 (18.67)
2	Azoxystrobin 25 SC	0.80 (5.12)	2.66 (9.39)	6.66 (14.82)	9.33 (17.78)	12.66 (20.81)
3	RIL 010 25 SC	0.77 (5.02)	3.33 (10.42)	7.00 (15.34)	10.65 (19.05)	14.66 (22.35)
4	Pyraclostrobin 25 EC	0.63 (4.56)	3.66 (11.03)	7.33 (15.62)	11.33 (19.67)	16.66 (23.85)
5	Tricyclazole 75 WP	0.87 (5.33)	5.65 (13.76)	9.65 (18.10)	14.65 (22.50)	18.33 (25.34)
6	Fenoxanil 20 SC	0.73 (4.90)	5.65 (13.76)	11.00 (19.36)	15.33 (23.04)	24.66 (29.73)
7	Iprobenfos 48 EC	0.53 (4.16)	6.33 (14.57)	13.99 (21.74)	23.66 (29.10)	28.33 (32.16)
8	Isoprothiolane 40 EC	0.63 (4.54)	7.00 (15.34)	14.66 (22.51)	25.33 (30.22)	34.33 (35.85)
9	Edifenphos 50 EC	0.60 (4.40)	7.33 (15.71)	14.99 (22.78)	26.00 (30.65)	34.65 (36.06)
10	Propiconazole 25 EC	0.67 (4.67)	7.00 (15.34)	15.33 (23.04)	26.33 (30.87)	35.33 (36.29)
11	Kasugamycin 2L	0.80 (5.12)	7.66 (16.07)	15.33 (23.04)	27.66 (31.53)	36.33 (37.06)
12	Carbendazim 50 WP	0.57 (4.31)	10.33 (18.75)	18.66 (25.59)	30.66 (33.62)	39.66 (39.03)
13	Difenconazole + Propiconazole 30 EC	0.67 (4.66)	9.00 (17.45)	21.33 (27.50)	31.66 (34.24)	40.32 (39.35)
14	Carbendazim + Epoxiconazole 25 EC	0.83 (5.24)	9.66 (18.11)	22.66 (28.42)	33.00 (35.06)	44.99 (42.12)
15	Epoxiconazole 7.5 EC	0.80 (5.13)	12.00 (20.27)	24.66 (29.71)	38.66 (38.44)	47.33 (43.47)
16	Difenconazole 25 EC	0.90. (5.44)	13.66 (21.69)	26.33 (30.87)	40.66 (39.62)	51.33 (45.78)
17	Diniconazole 25 WP	0.70 (4.75)	15.33 (23.04)	41.33 (40.01)	50.33 (45.19)	62.66 (52.39)
18	Control	0.70 (4.79)	28.66 (32.32)	73.66 (59.13)	98.66 (83.71)	100.00 (90.00)
SEm±		0.26	0.28	0.89	0.58	1.00
CD (P=0.05)		ns	0.79	2.57	1.68	2.90

*Figures in parentheses are arc sin transformed values

best control followed by iprobenfos 48 EC (13.99%), isoprothiolane 40 EC (14.66%), edifenphos 50 EC (14.99%), propiconazole 25 EC (15.33%) and kasugamycin 2 L (15.33%). However, iprobenfos, isoprothiolane, edifenphos, propiconazole and kasugamycin were statistically on par with each other and were superior to carbendazim except propiconazole and kasugamycin. Difenconazole + propiconazole 30 EC (21.33%) had come next in the order of its efficacy followed by carbendazim + epoxiconazole 25 EC (22.66%), epoxiconazole 7.5 EC (24.66%) and difenconazole 25 EC (26.33%). However, difenconazole in combination with propiconazole was significantly superior to difenconazole alone and carbendazim in combination with epoxiconazole was significantly inferior to carbendazim alone where as epoxiconazole alone was on par with its combination with carbendazim. Diniconazole 25 WP (41.33%) was the least effective of all the fungicides tested even though it had offered far better control over the untreated check (73.33%).

The per cent leaf blast severity at 15 DAI ranged between 7.60 (pyroquilon 50 WP) and 98.66 (untreated check). The performance of pyroquilon 50 WP (7.6%) was significantly superior to rest of the fungicides. Azoxystrobin 25 SC (9.33%) was next best fungicide followed by RIL 010 25 SC (10.65%), pyraclostrobin 25 EC (11.33%), tricyclazole 75 WP (14.65%) and fenoxanil 20 SC (15.33%). However, there was no significant difference between azoxystrobin and RIL 010; RIL 010 and pyraclostrobin; pyraclostrobin and tricyclazole; tricyclazole and fenoxanil, respectively. But each fungicide significantly differed with the other alternately in the order of their efficacy. Iprobenfos 48 EC (23.66%) had come next in the order of its efficacy followed by isoprothiolane 40 EC (25.33%), edifenphos 50 EC (26%), propiconazole 25 EC (26.33%) and kasugamycin 2 L (27.66%). However, isoprothiolane, edifenphos,

propiconazole and kasugamycin were statistically on par with each other and superior to carbendazim 50 WP (30.66%). But they were significantly inferior to iprobenfos except isoprothiolane. Difenconazole + propiconazole 30 EC (31.66%) followed by carbendazim + epoxiconazole 25 EC (33%), epoxiconazole 7.5 EC (38.66%) and difenconazole 25 WP (40.66%) were in the decreasing order of their efficacy. However, carbendazim alone was significantly superior to its combination with epoxiconazole where as difenconazole in combination with propiconazole was significantly superior to difenconazole alone. Diniconazole 25 WP (50.33%) was the least effective of all the fungicides tested even though it had significantly reduced the disease incidence when compared to the untreated check (98.66%).

The per cent leaf blast severity at 20 DAI varied from 10.33 (pyroquilon 50 WP) to 100 per cent (untreated control). Pyroquilon 50 WP (10.33%), azoxystrobin 25 SC (12.66%), RIL 010 25 SC (14.66%), pyraclostrobin 25 EC (16.66%) and tricyclazole 75 WP (18.33%) were first in the order of their efficacy. However, there was no significant difference between pyroquilon and azoxystrobin; azoxystrobin and RIL 010; RIL 010 and pyraclostrobin; pyraclostrobin and tricyclazole, respectively. But each fungicide significantly differed with the other alternately in the order of their efficacy. Fenoxanil 20 SC (24.66%), iprobenfos 48 EC (28.33%), isoprothiolane 40 EC (34.33%), edifenphos 50 EC (34.65%), propiconazole 25 EC (35.33%), kasugamycin 2 L (36.33%) and carbendazim 50 WP (39.66%) were next in the order of their efficacy. However, there was no significant difference between fenoxanil and iprobenfos where as isoprothiolane was statistically on par with edifenphos, propiconazole and kasugamycin. Carbendazim was found on par with kasugamycin, but significantly inferior to

propiconazole. Difenconazole + propiconazole 30 EC (40.32%), carbendazim + epoxiconazole 25 EC (44.99%), epoxiconazole 7.5 EC (47.33%), difenconazole 25 EC (51.33%) and diniconazole 25 WP (62.66%) were the last in the order of their efficacy. However, carbendazim alone was significantly superior to its combination with epoxiconazole where as difenconazole was significantly inferior to its combination with propiconazole. Diniconazole was the least effective and significantly inferior to all the fungicides tested.

4.5.2.3 Efficacy of fungicides as foliar spray in the nursery beds (*Kharif*, 2002 & 2003; pooled data)

The data furnished in Table 22 reveal that all the fungicides tested as foliar spray against rice blast were found significantly superior over the control up to 20 days after inoculation. The efficacy of pyroquilon 50 WP, azoxystrobin 25 SC, RIL 010 25 SC, pyraclostrobin 25 EC and tricyclazole 75 WP was excellent. Fenoxanil 20 EC, iprobenfos 48 EC, isoprothiolane 40 EC, edifenphos 50 EC, propiconazole 25 EC and kasugamycin 2 L were also superior in minimizing leaf blast incidence. Carbendazim 50 WP exhibited good amount of control compared to rest of the fungicides. Diniconazole 25 WP was the least effective of all the fungicides tested even though it was significantly superior over untreated control. The fungicides were sprayed in the nursery beds after the appearance of initial disease symptoms. The per cent leaf blast severity in all the experimental plots was found non-significant prior to the fungicidal spray.

At 5 DAI, the per cent leaf blast severity varied from 1.66 (pyroquilon 50 WP) to 26.66 (untreated control). Pyroquilon 50 WP (1.66%) exhibited its superiority as

foliar spray in the nursery beds followed by azoxystrobin 25 SC (1.99%), RIL 010 25 SC (2.49%), pyraclostrobin 25 EC (2.49%), tricyclazole 75 WP (4.49%) and fenoxanil 20 SC (4.65%). There was no significant difference between pyroquilon and azoxystrobin; pyraclostrobin and RIL 010; tricyclazole and fenoxanil, respectively. Iprobenfos 48 EC (5.33%) was the next best fungicide in the order of its efficacy followed by isoprothiolane 40 EC (5.83%), propiconazole 25 EC (6%), edifenphos 50 EC (6.16%) and kasugamycin 2 L (6.49%). However, there was no significant difference between iprobenfos and isoprothiolane while edifenphos, propiconazole and kasugamycin were statistically on par with each other. In addition, isoprothiolane was found non-significant with edifenphos and propiconazole also. Difenconazole + propiconazole 30 EC (7.83%) was the next in the order of its efficacy followed by carbendazim + epoxiconazole 25 EC (8.49%), carbendazim 50 WP (9.33%), epoxiconazole 7.5 EC (11.16%) and difenconazole 25 EC (12.33%). However, all these fungicides significantly differed with one another in the order of their efficacy except difenconazole + propiconazole and carbendazim + epoxiconazole that were on par with each other. Diniconazole 25 WP (14.49%) was the least effective of all the fungicides tested even though it was found significantly superior over the untreated control (26.66%).

The per cent leaf blast severity at 10 DAI varied from 4.99 (pyroquilon 50 WP) to 70.16 (untreated control). Pyroquilon 50 WP (4.99%) exhibited its supremacy as foliar spray over other fungicides. Azoxystrobin 25 SC (6.49%), RIL 010 25 SC (7.16%) and pyraclostrobin 25 EC (7.49%) were found statistically on par with each other and exhibited significantly superior control over rest of the fungicides. Tricyclazole 75 WP (9.99%) and fenoxanil 20 SC (11.32%) were the next best fungicides followed by

Table 22: Efficacy of fungicides as foliar spray against rice blast in nursery beds (2002 & 2003; pooled data)

S. No.	Name of the fungicide	Leaf blast severity (%) Before spray	Leaf blast severity (%)*			
			5DAI	10DAI	15DAI	20DAI
1	Pyroquilon 50 WP	0.53 (4.17)	1.66 (7.49)	4.99 (12.92)	6.96 (15.34)	9.99 (18.44)
2	Azoxystrobin 25 SC	0.56 (4.29)	1.99 (8.13)	6.49 (14.77)	9.49 (17.95)	13.49 (21.56)
3	RIL 010 25 SC	0.60 (4.44)	2.49 (9.10)	7.16 (15.56)	10.65 (19.00)	15.16 (22.95)
4	Pyraclostrobin 25 EC	0.51 (4.09)	2.49 (9.10)	7.49 (15.89)	11.33 (19.64)	17.16 (24.50)
5	Tricyclazole 75 WP	0.65 (4.62)	4.49 (12.25)	9.99 (18.44)	15.15 (22.87)	18.83 (25.70)
6	Fenoxanil 20 SC	0.58 (4.37)	4.65 (12.39)	11.32 (19.64)	16.33 (23.81)	25.65 (30.40)
7	Iprobenfos 48 EC	0.43 (3.76)	5.33 (13.31)	13.82 (21.81)	22.99 (28.66)	27.99 (31.95)
8	Isoprothiolane 40 EC	0.51 (4.09)	5.83 (13.94)	14.49 (22.38)	24.49 (29.67)	32.16 (34.57)
9	Edifenphos 50 EC	0.48 (3.97)	6.16 (14.42)	14.99 (22.79)	26.33 (30.85)	32.83 (34.94)
10	Propiconazole 25 EC	0.55 (4.25)	6.00 (14.18)	15.33 (23.03)	26.66 (31.11)	33.83 (35.55)
11	Kasugamycin 2L	0.60 (4.44)	6.49 (14.77)	15.49 (23.19)	27.49 (31.63)	34.99 (36.27)
12	Carbendazim 50 WP	0.48 (3.97)	9.33 (17.76)	19.49 (26.21)	29.99 (33.21)	39.16 (38.76)
13	Difenconazole + Propiconazole 30 EC	0.47 (3.93)	7.83 (16.22)	19.99 (26.56)	31.83 (34.33)	39.99 (39.23)
14	Carbendazim + Epoxiconazole 25 EC	0.58 (4.37)	8.49 (16.95)	20.99 (27.28)	33.16 (35.18)	43.16 (41.09)
15	Epoxiconazole 7.5 EC	0.58 (4.37)	11.16 (19.55)	23.99 (29.33)	38.49 (38.35)	51.99 (46.15)
16	Difenconazole 25 EC	0.61 (4.48)	12.33 (20.53)	25.99 (30.66)	41.16 (39.93)	55.33 (48.04)
17	Diniconazole 25 WP	0.53 (4.17)	14.49 (22.38)	37.83 (37.94)	49.99 (45.00)	65.16 (53.85)
18	Control	0.50 (4.05)	26.66 (31.11)	70.16 (56.91)	97.99 (81.87)	100.00 (90.00)
SEm±		0.17	0.27	0.56	0.56	1.12
CD (P=0.05)		ns	0.78	1.62	1.61	3.22

*Figures in parentheses are arc sin transformed values

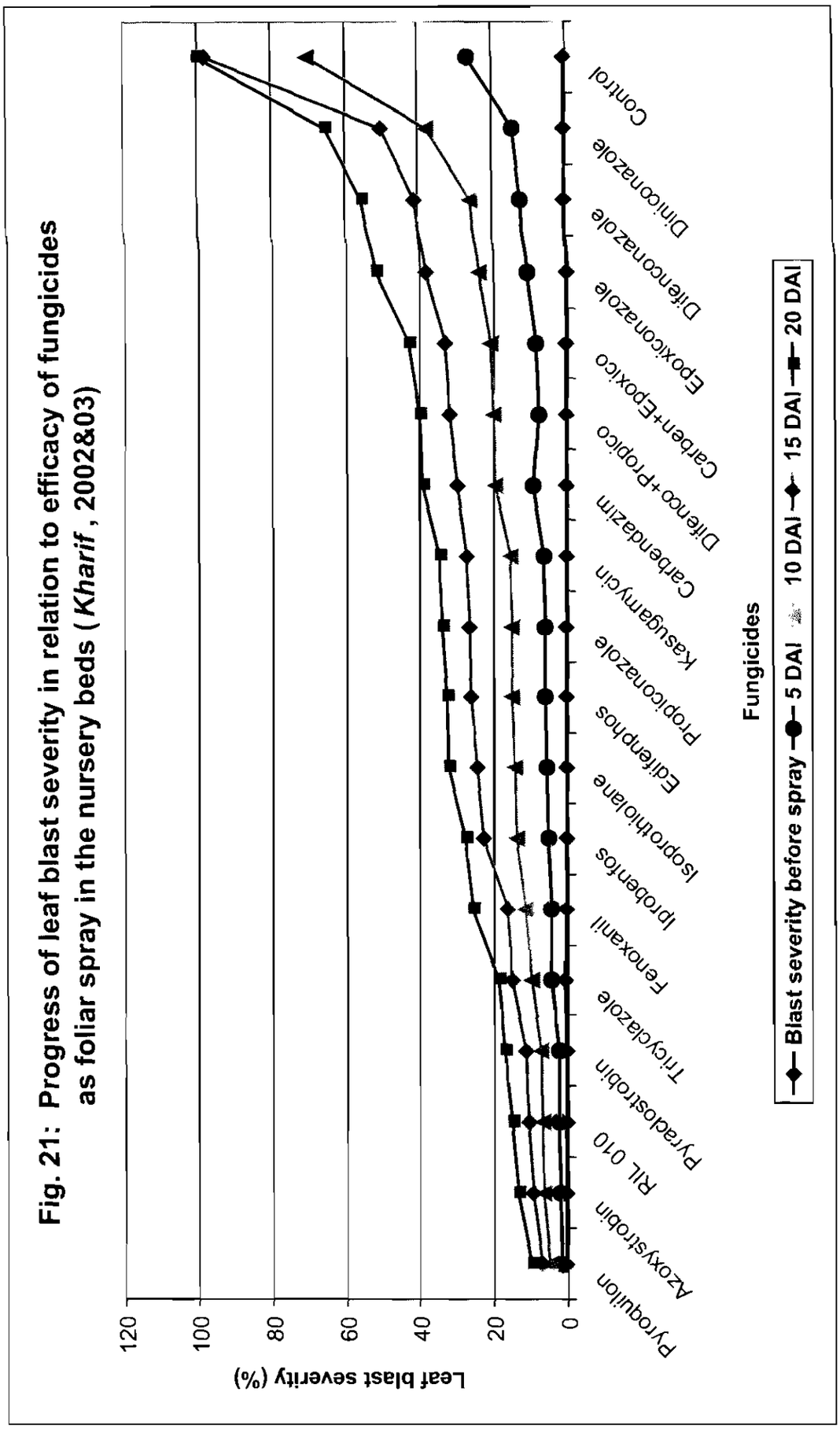


Fig. 21: Progress of leaf blast severity in relation to efficacy of fungicides as foliar spray in the nursery beds (*Kharif*, 2002&03)

iprobenfos 48 EC (13.82%), isoprothiolane 40 EC (14.49%), edifenphos 50 EC (14.99%), propiconazole 25 EC (15.33%) and kasugamycin 2 L (15.49%). However, tricyclazole was statistically on par with fenoxanil while there was no significant difference among iprobenfos, isoprothiolane, edifenphos, propiconazole and kasugamycin. Carbendazim 50 WP (19.49%) was next in the order of its efficacy followed by difenconazole + propiconazole 30 EC (19.99%), carbendazim + epoxiconazole 25 EC (20.99%), epoxiconazole 7.5 EC (23.99%) and difenconazole 25 EC (25.99%). Difenconazole in combination with propiconazole was significantly superior to difenconazole alone while epoxiconazole in combination with carbendazim was significantly superior to epoxiconazole alone. Diniconazole 25 WP (37.83%) was found the least effective of all the fungicides tested even though it had offered far better control over the untreated check (70.16%).

At 15 DAI, the per cent leaf blast severity ranged between 6.96 (pyroquilon 50 WP) and 97.99 (untreated check). The performance of pyroquilon 50 WP (6.96%) was significantly superior to rest of the fungicides. Azoxystrobin 25 SC (9.49%) was next best fungicide followed by RIL 010 25 SC (10.65%), pyraclostrobin 25 EC (11.33%), tricyclazole 75 WP (15.15%) and fenoxanil 20 SC (16.33%). There was no significant difference between azoxystrobin and RIL 010; RIL 010 and pyraclostrobin, respectively. But, these four fungicides were significantly superior to tricyclazole and fenoxanil that in turn were on par with each other. Iprobenfos 48 EC (22.99%) was next in the order of its efficacy followed by isoprothiolane 40 EC (24.49%), edifenphos 50 EC (26.33%), propiconazole 25 EC (26.66%) and kasugamycin 2 L (27.49%). Iprobenfos was statistically on par with isoprothiolane while there was no significant difference

between edifenphos, propiconazole and kasugamycin. In addition, isoprothiolane, edifenphos and propiconazole were statistically on par with each other while there was no significant difference between edifenphos, propiconazole and kasugamycin. Carbendazim 50 WP (29.99%) followed by difenconazole + propiconazole 30 EC (31.83%), carbendazim + epoxiconazole 25 EC (33.16%), epoxiconazole 7.5 EC (38.49%) and difenconazole 25 WP (41.16%) were in the decreasing order of their efficacy. Carbendazim was statistically on par with kasugamycin as well as difenconazole + propiconazole. Difenconazole alone was significantly inferior to its combination with propiconazole while epoxiconazole alone was significantly inferior to its combination with carbendazim even though difenconazole and epoxiconazole were on par with each other. Diniconazole 25 WP (49.99%) was the least effective of all the fungicides tested even though it had significantly reduced the disease incidence compared to the untreated check (97.99%).

The per cent leaf blast severity at 20 DAI varied from 9.99 (pyroquilon 50 WP) to 100 per cent (untreated control). Pyroquilon 50 WP (9.99%), azoxystrobin 25 SC (13.49%), RIL 010 25 SC (15.16%), pyraclostrobin 25 EC (17.16%) and tricyclazole 75 WP (18.83%) were first in the order of their efficacy. There was no significant difference between pyroquilon and azoxystrobin; azoxystrobin, RIL 010 and Pyraclostrobin; RIL 010, pyraclostrobin and tricyclazole, respectively. Fenoxanil 20 SC (25.65%), iprobenfos 48 EC (27.99%), isoprothiolane 40 EC (32.16%), edifenphos 50 EC (32.83%), propiconazole 25 EC (33.83%), kasugamycin 2 L (34.99%) and carbendazim 50 WP (39.16%) were next in the order of their efficacy. However, there was no significant difference between fenoxanil and iprobenfos while isoprothiolane was statistically on par

with edifenphos, propiconazole and kasugamycin. Carbendazim was found on par with kasugamycin, propiconazole and also difenconazole + propiconazole. Difenconazole + propiconazole 30 EC (39.99%), carbendazim + epoxiconazole 25 EC (43.16%), epoxiconazole 7.5 EC (51.99%), difenconazole 25 EC (55.33%) and diniconazole 25 WP (65.16%) were the last in the order of their efficacy. Difenconazole and epoxiconazole were significantly inferior to their combinations with propiconazole and carbendazim, respectively. Diniconazole (65.16%) was the least effective of all the fungicides tested but was significantly superior to untreated control (100%).

4.5.3 Efficacy of fungicides in the main field

The experiments were conducted to test the efficacy of various fungicides as foliar spray against leaf blast and neck blast incidence in the main field during *Kharif*, 2002 and *Kharif*, 2003. The details of the fungicides and the respective dosage were presented in the Table 2. Observations on leaf blast severity were recorded at 10 day interval starting from 10 days to 30 days after spray of the fungicides (DASP). Observations on neck blast were taken in terms of lesioned necks and broken necks. Observations on grain yield were recorded in the form of ill-filled grain, filled grain and total grain. The results of *Kharif*, 2002; *Kharif*, 2003 and the pooled data of *Kharif*, 2002 & 2003 are presented in the Tables 23, 24 and 25 (Plate 30).

4.5.3.1 Efficacy of fungicides on leaf blast severity in the main field during *Kharif*, 2002

The results presented in Table 23 shows that the disease incidence in all *the experimental plots was found non-significant prior to the fungicide spray. At 10 days*

after spray (DASP), the per cent leaf blast severity ranged between 6.00 (pyroquilon 50 WP) and 19.66 (untreated control). Pyroquilon 50 WP (6%) has exhibited the most effective control followed by azoxystrobin 25 SC (7%), pyraclostrobin 25 EC (7.66%) and RIL 010 25 SC (7.66%). Azoxystrobin 25 SC, pyraclostrobin 25 EC and RIL 010 25 SC were statistically on par with each other, but significantly inferior to pyroquilon. Fenoxanil 20 SC (8.66%) was the next in the order of its efficacy followed by iprobenfos 48 EC (9.33%), propiconazole 25 EC (9.66%), isoprothiolane 40 EC (9.66%), edifenphos 50 EC (10%), tricyclazole 75 WP (10.33%) and kasugamycin 2 L (10.66%). However, iprobenfos was statistically on par with fenoxanil and there was no significant difference among propiconazole, isoprothiolane, edifenphos, tricyclazole and kasugamycin. Carbendazim + epoxiconazole (11%) was next in the order of its efficacy followed by carbendazim 50 WP (11.33%), difenconazole + propiconazole 30 EC (12.66%), difenconazole 25 EC (13.66%), epoxiconazole 25 EC (14%) and diniconazole 25 WP (15.33%). Difenconazole in combination with propiconazole had exhibited its superiority over difenconazole 25 EC alone, while epoxiconazole in combination with carbendazim was significantly superior to epoxiconazole 7.5 EC alone. Diniconazole was found significantly inferior to all other fungicides, but it was superior to untreated check (19.66%).

At 20 DASP, the per cent leaf blast severity ranged between 8.66 (pyroquilon) and 35.66 (untreated control). Pyroquilon 50 WP (8.66%) and azoxystrobin 25 SC (9.33%) were excellent followed by pyraclostrobin 25 EC (11.33%), RIL010 (11.66%) and tricyclazole 75 WP (12%). Pyroquilon and azoxystrobin were statistically on par with each other and they were significantly superior to other fungicides tested. There was no significant difference among pyraclostrobin, RIL 010 and tricyclazole.

Edifenphos 50 EC (14.33%) was the next best fungicide followed by fenoxanil 20 SC (15%), iprobenfos 48 EC (15%), propiconazole 25 EC (15.66%) and kasugamycin 2 L (16.33%). But, there was no significant difference among all these fungicides and these were statistically on par with difenconazole + propiconazole 30 EC (15.66%). Carbendazim 50 WP (16.66%) was next in the order of its efficacy followed by carbendazim + epoxiconazole 25 EC (16.66%), difenconazole 25 EC (19.66%), epoxiconazole 7.5 EC (20.33%) and diniconazole 25 WP (24.66%). Epoxiconazole and difenconazole were inferior to their combinations with carbendazim and propiconazole, respectively. Diniconazole 25 WP (24.66%) was significantly inferior to all other fungicides, but superior to the untreated control (35.66%).

The per cent leaf blast severity at 30 DAI ranged between 13.33 (pyroquilon 50 WP) and 51.33 (untreated control). Pyroquilon 50 WP (13.33%) recorded the minimum leaf blast severity followed by azoxystrobin 25 SC (14.66%), pyraclostrobin 25 EC (16%), RIL 010 (17.33%) and tricyclazole 75 WP (17.66%). There was no significant difference between pyroquilon and azoxystrobin; azoxystrobin and pyraclostrobin; pyraclostrobin and RIL 010, while tricyclazole was on par with both pyraclostrobin and RIL 010. Each fungicide significantly differed with the other alternately except tricyclazole. Fenoxanil 20 SC (20.33%) was the next best fungicide followed by iprobenfos 48 EC (21.33%), isoprothiolane 40 EC (22.33%), edifenphos 50 EC (22.33%), propiconazole 25 EC (22.33%) and kasugamycin 2 L (24.66%). However, fenoxanil, iprobenfos, isoprothiolane and edifenphos were on par with each other and there was no significant difference among isoprothiolane, edifenphos, propiconazole and kasugamycin. Carbendazim + epoxiconazole 25 EC (26.32%) was next in the order of its efficacy followed by carbendazim 50 WP (27.66%), difenconazole + propiconazole 30 EC (30.66%), difenconazole 25 EC (32.66%) and epoxiconazole 7.5 EC (34%). All the

Table 23: Efficacy of fungicides as foliar spray against rice blast in the main field (*Kharif*, 2002)

S. No.	Name of the fungicide	Disease incidence before spray	Leaf blast severity (%)*			Neck blast incidence (%)		Grain yield (kg/ha)		
			10 DASP	20 DASP	30 DASP	Lesioned necks	Broken necks	Ill-filled grains	Filled grains	Total
1	Pyroquilon 50 WP	0.60 (4.43)	6.00 (14.15)	8.66 (17.08)	13.33 (21.41)	0.00	0.00	23.66	2868.33	2870.66
2	Azoxystrobin 25 SC	0.60 (4.43)	7.00 (15.34)	9.33 (17.77)	14.66 (22.57)	0.00	0.00	25.00	2725.00	2750.00
3	Pyraclostrobin 25 EC	0.80 (5.13)	7.66 (16.06)	11.33 (19.64)	16.00 (23.57)	0.00	0.00	33.33	2690.33	2723.65
4	RIL 010 25 SC	1.00 (5.73)	7.66 (16.06)	11.66 (19.95)	17.33 (24.58)	0.00	0.00	26.33	2706.66	2732.99
5	Tricyclazole 75 WP	0.50 (4.05)	10.33 (18.71)	12.00 (20.26)	17.66 (24.85)	2.66	0.00	36.66	2612.66	2837.66
6	Fenoxanil 20 SC	1.00 (5.74)	8.66 (17.11)	15.00 (22.78)	20.33 (26.80)	4.00	1.66	34.66	2593.66	2628.32
7	Iprobenfos 48 EC	1.20 (6.29)	9.33 (17.78)	15.00 (22.77)	21.33 (27.50)	4.66	0.00	41.33	2524.00	2565.33
8	Isoprothiolane 40 EC	0.90 (5.44)	9.66 (18.09)	15.00 (22.78)	22.33 (28.18)	2.00	0.00	38.33	2564.66	2602.99
9	Edifenphos 50 EC	0.40 (3.63)	10.00 (18.43)	14.33 (22.24)	22.33 (28.20)	5.33	0.00	42.66	2482.66	2525.32
10	Propiconazole 25 EC	0.90 (5.44)	9.66 (18.10)	15.66 (23.27)	23.00 (28.60)	3.66	0.00	44.66	2416.00	2460.65
11	Kasugamycin 2L	0.80 (5.13)	10.66 (19.00)	16.33 (23.83)	24.66 (29.77)	6.33	0.33	45.00	2422.00	2467.00
12	Carbendazim 50 WP	0.60 (4.00)	11.33 (19.67)	16.66 (24.09)	27.66 (31.73)	6.66	1.33	48.66	2338.33	2386.99
13	Difenconazole + Propiconazole 30 EC	0.40 (3.61)	12.66 (20.83)	15.66 (23.37)	30.66 (33.62)	6.66	2.00	56.33	2283.00	2339.33
14	Carbendazim + Epoxiconazole 25 EC	0.80 (5.13)	11.00 (19.35)	16.66 (24.09)	26.32 (30.87)	8.66	3.66	67.66	2186.33	2253.99
15	Difenconazole 25 EC	1.00 (5.74)	13.66 (21.69)	19.66 (26.32)	32.66 (34.85)	10.66	3.66	82.33	2040.33	2122.66
16	Epoxiconazole 7.5 EC	0.80 (5.13)	14.00 (21.97)	20.33 (26.77)	34.00 (35.67)	12.66	7.00	107.33	1935.00	2042.33
17	Diniconazole 25 WP	1.00 (5.74)	15.33 (23.02)	24.66 (29.77)	36.33 (37.06)	14.33	7.66	128.66	1880.66	2009.32
18	Control	0.60 (4.43)	19.66 (26.31)	35.66 (36.66)	51.33 (45.76)	21.66	17.33	386.30	1534.66	1920.96
SEm±		0.11	0.36	0.52	0.59	0.25	0.22	2.75	45.08	44.23
CD (P=0.05)		ns	1.03	1.46	1.70	0.72	0.63	7.92	129.57	127.12

*Figures in parentheses are arc sin transformed values; DASP = Number of days after spray of fungicide

fungicides significantly differed with one another except carbendazim and its combination with epoxiconazole. Diniconazole 25 WP (36.33%) was the least effective of all the fungicides tested even though it was significantly superior to the untreated control (51.33%).

4.5.3.2 Efficacy of fungicides on neck blast incidence during *Kharif*, 2002

The per cent lesioned necks varied between zero and 21.66 while the per cent broken necks ranged between zero and 17.33 (Table 23). There were no lesioned necks in the plots treated with pyroquilon 50 WP, azoxystrobin 25 SC, pyraclostrobin 25 EC and RIL 010 25 SC. Isoprothiolane 40 EC (2%) registered the lowest number of lesioned necks followed by tricyclazole 75 WP (2.66%), propiconazole 25 EC (3.66%), fenoxanil 20 SC (4%), iprobenfos 48 EC (4.66%), edifenphos 50 WP (5.33%), kasugamycin 2 L (6.33%), carbendazim 50 WP (6.66%) and difenconazole + propiconazole 30 EC (6.66%). There was no significant difference between isoprothiolane and tricyclazole; propiconazole and fenoxanil; fenoxanil and iprobenfos, respectively. But each fungicide differed significantly with the other alternately. Kasugamycin was on par with carbendazim and difenconazole + propiconazole, but it was significantly inferior to edifenphos. Carbendazim + epoxiconazole 25 EC (8.66%) had mustered the next higher numbers of lesioned necks followed by difenconazole 25 EC (10.66%) and epoxiconazole 7.5 EC (12.66%). However, they had significantly differed with one another. Diniconazole 25 WP (14.33%) recorded the highest number of lesioned necks, but it was superior to untreated control (21.66%).

There was absolutely no broken necks in the plots treated with pyroquilon 50 WP, azoxystrobin 25 SC, pyraclostrobin 25 EC, RIL 010 25 SC, tricyclazole 75 WP,

iprobenfos 48 EC, isoprothiolane 40 EC, edifenphos 50 EC and propiconazole 25 EC. Kasugamycin 2 L (0.33%) recorded the lowest number of broken necks followed by carbendazim 50 WP (1.33%), fenoxanil 20 SC (1.66%), difenconazole + propiconazole 30 EC (2.00%), carbendazim + epoxiconazole 25 EC (3.66%), difenconazole 25 EC (3.66%), epoxiconazole 25 EC (7.00%) and diniconazole 25 WP (7.66%). However, fenoxanil, carbendazim and difenconazole + propiconazole were statistically on par with each other and there was no significant difference between carbendazim + epoxiconazole and difenconazole; epoxiconazole and diniconazole, respectively, but they significantly differed with one another and also with the untreated control (17.33%) in general.

4.5.3.3 Efficacy of fungicides in relation to grain yield during *Kharif*, 2002

The amount of ill-filled grain varied from 25 kg/ha to 386 kg/ha while the quantity of filled grains ranged between 2868.33 kg/ha and 1534.66 kg/ha in pyroquilon 50 WP and untreated control plots, respectively. Similarly, the total grain yield varied from 2891.99 kg/ha and 1920.96 kg/ha respectively (Table 23). All the fungicides tested were significantly superior to untreated control. Pyroquilon 50 WP (23.66 kg/ha) recorded the lowest quantity of ill-filled grain followed by azoxystrobin 25 SC (25.00 kg/ha), RIL 010 25 SC (26.33 kg/ha), pyraclostrobin 25 EC (33.33 kg/ha), fenoxanil 20 SC (34.66 kg/ha), tricyclazole 75 WP (36.66 kg/ha) and isoprothiolane 40 EC (38.33 kg/ha). However, pyroquilon, azoxystrobin and RIL 010 were statistically on par with each other, but significantly superior over the other fungicides while there was no significant difference among pyraclostrobin, fenoxanil, tricyclazole and isoprothiolane. Iprobenfos 48 EC (41.33 kg/ha) recorded the next lowest quantity of ill-filled grain and it was statistically on par with edifenphos 50 WP (42.66 kg/ha), propiconazole 25 EC (44.66 kg/ha), kasugamycin 2 L (45.00 kg/ha) and carbendazim 50 WP (48.66 kg/ha), but

were significantly superior to rest of the fungicides. Difenconazole + propiconazole 30 EC (56.33 kg/ha), carbendazim + epoxiconazole 25 EC (67.66 kg/ha), difenconazole 25 EC (82.33 kg/ha), epoxiconazole 7.5 EC (107.33 kg/ha) and diniconazole 25 WP (128.66 kg/ha) were in the order of increasing quantities of ill-filled grains and differed significantly with one another.

Pyroquilon 50 WP (2868.33 kg/ha) had given the maximum quantity of filled grain that was significantly superior to all other fungicides tested while untreated control (1534.66 kg/ha) has registered the lowest. Azoxystrobin 25 SC (2725 kg/ha) recorded the next highest quantity of filled grain even though it was statistically on par with pyraclostrobin 25 EC (2690.33 kg/ha), RIL 010 25 SC (2706.66 kg/ha), tricyclazole 75 WP (2612.66 kg/ha) and fenoxanil 20 SC (2593.66 kg/ha). Iprobenfos 48 EC (2524 kg/ha) registered the highest quantity of filled grain even though it was statistically on par with isoprothiolane 40 EC (2564.66 kg/ha), edifenphos 50 EC (2482.66 kg/ha), propiconazole 25 EC (2416 kg/ha) and kasugamycin 2 L (2422 kg/ha), but significantly superior to rest of the fungicides. Iprobenfos and isoprothiolane were statistically on par with fenoxanil and tricyclazole. Carbendazim 50 WP (2338.33 kg/ha) followed by difenconazole + propiconazole 30 EC (2283.00 kg/ha), carbendazim + epoxiconazole 25 EC (2186.33 kg/ha), difenconazole 25 EC (2040.33 kg/ha), epoxiconazole 25 EC (1935.00 kg/ha) and diniconazole 25 WP (1880.66 kg/ha) were in the decreasing order with reference to the quantities of filled grain. However, carbendazim alone was superior to its combination with epoxiconazole where as difenconazole was inferior to its combination with propiconazole. Difenconazole was statistically on par with epoxiconazole, but superior to diniconazole. There was no significant difference between epoxiconazole and diniconazole.

The same trend was reflected in case of total grain yield also (Table 23). Pyroquilon 50 WP (2870.66 kg/ha) was superior to all other fungicides while the untreated control recorded the lowest total grain yield of 1920.96 kg/ha. Azoxystrobin 25 SC (2750 kg/ha), pyraclostrobin 25 EC (2723.66 kg/ha), RIL 010 25 SC (2732.99 kg/ha), tricyclazole 75 WP (2837.66 kg/ha) and fenoxanil 20 SC (2628.32 kg/ha) were statistically on par with each other and significantly superior to rest of the fungicides. Iprobenfos 48 EC (2565.32 kg/ha), isoprothiolane 40 EC (2602.99 kg/ha), edifenphos 50 EC (2525.32 kg/ha), propiconazole 25 EC (2460.65 kg/ha) and kasugamycin 2 L (2467 kg/ha) were statistically on par with each other. Carbendazim 50 WP (2386.99 kg/ha), difenconazole + propiconazole 30 EC (2339.33 kg/ha), carbendazim + epoxiconazole 25 EC (2253.99 kg/ha), difenconazole 25 EC (2122.66 kg/ha), epoxiconazole 7.5 EC (2042.33 kg/ha) and diniconazole 25 WP (2009.32 kg/ha) were in the decreasing order of their efficacy. However, carbendazim was statistically on par with difenconazole + propiconazole and there was no significant difference among difenconazole, epoxiconazole and diniconazole.

4.5.3.4 Efficacy of fungicides on leaf blast severity in the main field during *Kharif*, 2003

The results presented in Table 24 reveal that the disease incidence in all the experimental plots was found non-significant prior to the fungicide spray. At 10 days after spray (DASP), the per cent leaf blast severity ranged between 4.66 (pyroquilon 50 WP) and 22.33 (untreated control). Pyroquilon 50 WP (4.66%) has exhibited its superiority over the other fungicides followed by azoxystrobin 25 SC (5.33%), pyraclostrobin 25 EC (5.33%), RIL 010 25 SC (6%) and tricyclazole 75 WP (6.66%).

There was no significant difference between azoxystrobin and pyraclostrobin; RIL010 and tricyclazole, respectively. Fenoxanil 20 SC (8.33%) was next in the order of its efficacy followed by iprobenfos 48 EC (8.33%), isoprothiolane 40 EC (9.66%), edifenphos 50 EC (9.66%), propiconazole 25 EC (10.66%), kasugamycin 2 L (10.66%) and carbendazim 50 WP (11.33%). However, iprobenfos was statistically on par with fenoxanil and isoprothiolane was on par with edifenphos where as propiconazole, kasugamycin and carbendazim had not shown any significant difference among them. Difenconazole + propiconazole 30 EC (12.66%) was next in the order of its efficacy followed by carbendazim + epoxiconazole (15.33%), difenconazole 25 EC (15.33%) and epoxiconazole 25 EC (15.66%). Difenconazole in combination with propiconazole exhibited superiority over difenconazole alone, where as epoxiconazole in combination with carbendazim was exceptionally on par with epoxiconazole 7.5 EC alone. Diniconazole 25 WP (17%) was found significantly inferior to all other fungicides, but it was superior to untreated control (22.33%).

At 20 DASP, the per cent leaf blast severity ranged between 7.33 (pyroquilon) and 37.33 (untreated control). Pyroquilon 50 WP (7.33%), azoxystrobin 25 SC (7.49%) and pyraclostrobin 25 EC (8%) were excellent followed by tricyclazole 75 WP (9.33%), RIL010 (9.66%) and fenoxanil 20 SC (10.66%). Pyroquilon, azoxystrobin and pyraclostrobin were statistically on par with each other and significantly superior to all other fungicides tested. There was no significant difference between tricyclazole and RIL 010; RIL 010 and fenoxanil, respectively, but tricyclazole differed significantly with fenoxanil. Iprobenfos 48 EC (14.33%) was the next best fungicide followed by isoprothiolane 40 EC (14.66%), edifenphos 50 EC (15%), propiconazole 25 EC (15.66%)

and kasugamycin 2 L (17.33%). There was no significant difference among all these fungicides except kasugamycin that in turn was statistically on par with difenconazole + propiconazole 30 EC (17.33%). Carbendazim 50 WP (17.66%) was next in the order of its efficacy followed by carbendazim + epoxiconazole 25 EC (20.66%), difenconazole 25 EC (24.66%), epoxiconazole 7.5 EC (26.33%) and diniconazole 25 WP (29.66%). Epoxiconazole was inferior to its combination with carbendazim and difenconazole was inferior to its combination with propiconazole, respectively. Diniconazole 25 WP (29.66%) was significantly inferior to all other fungicides, but superior to the untreated control (37.33%).

At 30 DASP, the per cent leaf blast severity ranged between 10.33 (pyroquilon 50 WP) and 53.00 (untreated control). Pyroquilon 50 WP (10.33%) recorded the lowest leaf blast severity followed by azoxystrobin 25 SC (10.66%), pyraclostrobin 25 EC (11.66%), RIL 010 (12.33%) and tricyclazole 75 WP (13.33%). There was no significant difference between pyroquilon and azoxystrobin; pyraclostrobin and RIL 010, respectively, but were superior to tricyclazole. Fenoxanil 20 SC (17%) was the next best fungicide followed by iprobenfos 48 EC (20.33%), isoprothiolane 40 EC (23%), edifenphos 50 EC (24%) and propiconazole 25 EC (24.66%). However, fenoxanil and iprobenfos differed significantly with each other, but were superior to isoprothiolane, edifenphos and propiconazole that in turn were on par with each other. Kasugamycin 2 L (26.33%) was next in the order of its efficacy followed by carbendazim 50 WP (28.33%), difenconazole + propiconazole 30 EC (29.33%), carbendazim + epoxiconazole 25 EC (29.66%), difenconazole 25 EC (34.66%) and diniconazole 25 WP (38%). However, kasugamycin, carbendazim, difenconazole + propiconazole and carbendazim +

Table 24: Efficacy of fungicides as foliar spray against rice blast in the main field (*Kharif*, 2003)

S. No.	Name of the fungicide	Disease incidence before spray	Leaf blast severity (%)*			Neck blast incidence (%)		Grain yield (kg/ha)		
			10 DASP	20 DASP	30 DASP	Lesioned necks	Broken necks	Ill-filled grains	Filled grains	Total
1	Pyroquilon 50 WP	1.03 (6.55)	4.66 (12.46)	7.33 (15.70)	10.33 (18.74)	0.00	0.00	26.33	2977.66	3003.99
2	Azoxystrobin 25 SC	1.00 (5.74)	5.33 (13.35)	7.49 (16.38)	10.66 (19.06)	0.00	0.00	30.79	2958.33	2988.99
3	Pyraclostrobin 25 EC	0.70 (4.80)	5.33 (13.35)	8.00 (16.43)	11.66 (19.96)	0.00	0.00	31.66	2922.33	2953.99
4	RIL 010 25 SC	1.00 (5.74)	6.00 (14.18)	9.66 (18.10)	12.33 (20.55)	0.00	0.00	32.00	2866.66	2898.65
5	Tricyclazole 75 WP	0.80 (5.13)	6.66 (14.95)	9.33 (17.77)	13.33 (21.41)	2.00	0.00	38.33	2753.66	2791.99
6	Fenoxanil 20 SC	1.30 (6.55)	8.33 (16.77)	10.66 (19.05)	17.00 (24.34)	5.00	2.00	61.33	2546.66	2607.99
7	Iprobenfos 48 EC	1.20 (6.29)	8.33 (16.77)	14.33 (22.24)	20.33 (26.80)	5.33	0.00	55.66	2601.33	2656.99
8	Isoprothiolane 40 EC	0.80 (5.13)	9.66 (18.10)	14.66 (22.51)	23.00 (28.66)	3.00	0.00	47.00	2513.00	2560.00
9	Edifenphos 50 EC	0.60 (4.44)	9.66 (18.11)	15.00 (22.51)	24.00 (29.33)	5.66	0.00	58.33	2567.99	2526.32
10	Propiconazole 25 EC	0.60 (4.44)	10.66 (19.05)	15.66 (23.31)	24.66 (29.78)	5.33	0.66	59.00	2489.33	2548.33
11	Kasugamycin 2L	1.60 (7.27)	10.66 (19.05)	17.33 (24.60)	26.33 (30.87)	7.00	0.66	90.33	2325.33	2415.65
12	Carbendazim 50 WP	0.80 (5.12)	11.33 (19.67)	17.66 (24.82)	28.33 (28.20)	7.66	2.00	69.66	2259.33	2328.99
13	Difenconazole + Propiconazole 30 EC	0.70 (4.80)	12.66 (20.84)	17.33 (24.60)	29.33 (32.78)	6.66	2.66	118.66	2270.66	2389.32
14	Carbendazim + Epoxiconazole 25 EC	0.90 (5.44)	15.33 (23.05)	20.66 (27.03)	29.66 (32.99)	10.33	3.66	140.33	2160.66	2300.99
15	Difenconazole 25 EC	1.20 (6.29)	15.33 (23.05)	24.66 (29.77)	34.66 (36.07)	13.66	8.33	162.66	1852.66	2015.31
16	Epoxiconazole 7.5 EC	1.20 (6.29)	15.66 (23.31)	26.33 (30.86)	38.66 (38.44)	20.00	10.33	204.66	1742.66	1947.33
17	Diniconazole 25 WP	0.60 (4.44)	17.00 (24.35)	29.66 (32.97)	38.00 (38.06)	16.33	9.66	186.33	1800.00	1986.33
18	Control	0.60 (4.44)	22.33 (28.19)	37.33 (37.66)	53.00 (46.72)	23.33	18.00	358.66	1478.33	1836.98
SEm±		0.09	0.26	0.53	0.42	0.37	0.08	4.34	41.95	42.17
CD (P=0.05)		ns	0.75	1.52	1.21	1.08	0.26	12.50	120.59	121.20

*Figures in parentheses are arc sin transformed values; DASP = Number of days after spray of fungicide

epoxiconazole were statistically on par with each other but, superior to difenconazole and diniconazole. Surprisingly, epoxiconazole 7.5 EC (38.66%) was the least effective of all the fungicides tested even though it was significantly superior to the untreated control (53%). But, there was no significant difference between epoxiconazole and diniconazole (38.66%).

4.5.3.5 Efficacy of fungicides on neck blast incidence during *Kharif*, 2003

The per cent lesioned necks varied from zero to 23.33 while the per cent broken necks ranged between zero and 18.00 (Table 24). There were no lesioned necks in the plots treated with pyroquilon 50 WP, azoxystrobin 25 SC, pyraclostrobin 25 EC and RIL 010 25 SC. Tricyclazole 75 WP (2%) registered the lowest number of lesioned necks followed by isoprothiolane 40 EC (3%), fenoxanil 20 SC (5%), iprobenfos 48 EC (5.33%), propiconazole 25 EC (5.33%) and edifenphos 50 WP (5.66%). Tricyclazole was statistically on par with isoprothiolane and there was no significant difference among fenoxanil, iprobenfos, propiconazole and edifenphos, but the later were significantly inferior to tricyclazole and isoprothiolane. Difenconazole + propiconazole 30 EC (6.66%), Kasugamycin 2 L (7%) and carbendazim 50 WP (7.66%) were statistically on par with each other, but inferior to edifenphos. Carbendazim + epoxiconazole 25 EC (10.33%) recorded the next higher numbers of lesioned necks followed by difenconazole 25 EC (13.66%) and diniconazole 25 WP (16.33%) and epoxiconazole 7.5 EC (20%). But, they had significantly differed with one another. Epoxiconazole 7.5 EC (20%) recorded the highest number of lesioned necks, but it was superior to untreated control (23.33%).

There was absolutely no broken necks in the plots treated with pyroquilon 50 WP, azoxystrobin 25 SC, pyraclostrobin 25 EC, RIL 010 25 SC, tricyclazole 75 WP, iprobenfos 48 EC, isoprothiolane 40 EC and edifenphos 50 EC. Propiconazole 25 EC (0.66%) and kasugamycin 2 L (0.66%) recorded the lowest number of broken necks followed by carbendazim 50 WP (2%), fenoxanil 20 SC (2%) and difenconazole + propiconazole 30 EC (2.66%). However, propiconazole was statistically on par with kasugamycin and there was no significant difference among fenoxanil, carbendazim and difenconazole + propiconazole, but each group differed significantly with the other. Carbendazim + epoxiconazole 25 EC (3.66%) recorded the next higher numbers of broken necks followed by difenconazole 25 EC (8.33%) and diniconazole 25 WP (9.66%) and epoxiconazole 7.5 EC (10.33%). However, they significantly differed with one another. Epoxiconazole 7.5 EC (20%) recorded the highest number of lesioned necks, but it was superior to the untreated control (23.33%).

4.5.3.6 Efficacy of fungicides in relation to grain yield during *Kharif*, 2003

The amount of ill-filled grain varied from 26.33 kg/ha to 358.66 kg/ha where as the quantity of filled grains ranged between 2977.66 kg/ha and 1478.33 kg/ha in pyroquilon 50 WP and control plots, respectively. The total grain yield varied between 3003.99 kg/ha and 1836.98 kg/ha (Table 24). All the fungicides tested were found significantly superior to untreated control. Pyroquilon 50 WP (26.33 kg/ha) recorded the lowest quantity of ill-filled grain followed by azoxystrobin 25 SC (30.79 kg/ha), pyraclostrobin 25 EC (31.66 kg/ha), RIL 010 25 SC (32 kg/ha) and tricyclazole 75 WP (38.33 kg/ha). All the four fungicides were significantly on par with each other.

Isoprothiolane 40 EC (47 kg/ha) recorded the next lowest quantity of ill-filled grain, but it was statistically on par with iprobenfos 48 EC (55.66 kg/ha), edifenphos 50 WP (58.33 kg/ha), propiconazole 25 EC (59 kg/ha) and fenoxanil 20 EC (61.33 kg/ha). All the five fungicides were statistically on par with each other, but significantly superior to rest of the fungicides. Kasugamycin 2 L (90.33 kg/ha) and carbendazim 50 WP (69.66 kg/ha), Difenconazole + propiconazole 30 EC (118.66 kg/ha), carbendazim + epoxiconazole 25 EC (140.33 kg/ha), difenconazole 25 EC (162.66 kg/ha) and diniconazole 25 WP (186.33 kg/ha), epoxiconazole 7.5 EC (204.66 kg/ha) were in the order of increasing quantities of ill-filled grains and differed significantly with one another.

Pyroquilon 50 WP (2977.66 kg/ha) registered the maximum quantity of filled grain where as the control plot registered the lowest (1478.33 kg/ha). Azoxystrobin 25 SC (2958.33 kg/ha) recorded the next highest amount of filled grain followed by pyraclostrobin 25 EC (2922.33 kg/ha) and RIL 010 25 SC (2866.66 kg/ha). All the four fungicides were statistically on par with each other and significantly superior to all other fungicides. Tricyclazole 75 WP (2753.66 kg/ha), iprobenfos 48 EC (2601.33 kg/ha), isoprothiolane 40 EC (2567.99 kg/ha), fenoxanil 20 SC (2546.66 kg/ha), edifenphos 50 EC (2513.00 kg/ha) and propiconazole 25 EC (2489.33 kg/ha) recorded the next higher quantities of filled grain. However, iprobenfos was statistically on par with isoprothiolane, fenoxanil, edifenphos and propiconazole except tricyclazole that superior to all these five fungicides. Kasugamycin 2 L (2325.33 kg/ha), carbendazim 50 WP (2259.33 kg/ha), difenconazole + propiconazole 30 EC (2270.66 kg/ha), carbendazim + epoxiconazole 25 EC (2160.66 kg/ha), difenconazole 25 EC (1852.66 kg/ha) and diniconazole 25 WP (1800.00 kg/ha) and epoxiconazole 25 EC (1742.66 kg/ha) were in

the decreasing order with reference to the quantities of filled grain. However, kasugamycin, carbendazim and difenconazole + propiconazole were on par with each other, but superior to carbendazim + epoxiconazole. There was no significant difference among difenconazole, diniconazole and epoxiconazole.

The same trend was reflected in case of total grain yield also (Table 24). Pyroquilon 50 WP (3003.99 kg/ha) was superior to all other fungicides where as the untreated control recorded a total grain yield of 1836.98 kg/ha. Azoxystrobin 25 SC (2989.12 kg/ha), pyraclostrobin 25 EC (2953.99 kg/ha) and RIL 010 25 SC (2898.66 kg/ha) were statistically on par with each other and significantly superior to rest of the fungicides. Tricyclazole 75 WP (2791.99 kg/ha), iprobenfos 48 EC (2656.99 kg/ha), isoprothiolane 40 EC (2614.99 kg/ha), fenoxanil 20 SC (2607.99 kg/ha), edifenphos 50 EC (2571.33 kg/ha) and propiconazole 25 EC (2548.33 kg/ha) were next in the decreasing order of their efficacy. However, iprobenfos, isoprothiolane, fenoxanil, edifenphos and propiconazole were statistically on par with each other except tricyclazole. Kasugamycin 2 L (2415.66 kg/ha), carbendazim 50 WP (2328.99 kg/ha), difenconazole + propiconazole 30 EC (2389.32 kg/ha), carbendazim + epoxiconazole 25 EC (2300.00 kg/ha), difenconazole 25 EC (2015.32 kg/ha), diniconazole 25 WP (1986.33 kg/ha) and epoxiconazole 7.5 EC (1947.33 kg/ha) were in the next order of their decreasing efficacy. However, kasugamycin, carbendazim and difenconazole + propiconazole were on par with each other, but superior to carbendazim + epoxiconazole. There was no significant difference among difenconazole, diniconazole and epoxiconazole.

4.5.3.7 Efficacy of fungicides on leaf blast severity in the main field (*Kharif*, 2002&2003; pooled data)

The data furnished in Table 25 reveal that the disease incidence in all the experimental plots was found non-significant prior to the fungicide spray. At 10 days after spray (DASP), the per cent leaf blast severity ranged between 5.33 (pyroquilon 50 WP) and 20.99 (untreated control). Pyroquilon 50 WP (5.33%) has exhibited the most effective and superior control over all other fungicides followed by azoxystrobin 25 SC (6.16%), pyraclostrobin 25 EC (6.49%) and RIL 010 25 SC (6.83%). There was no significant difference between azoxystrobin and pyraclostrobin; RIL010 and pyraclostrobin, respectively. Tricyclazole 75 WP and fenoxanil 20 SC (8.49%) were the next best fungicides in the order of their efficacy followed by iprobenfos 48 EC (8.83%), isoprothiolane 40 EC (9.66%), edifenphos 50 EC (9.83%), propiconazole 25 EC (10.16%), kasugamycin 2 L (10.66%) and carbendazim 50 WP (11.33%). However, tricyclazole, fenoxanil and iprobenfos were statistically on par with each other and significantly superior over rest of the fungicides.

Isoprothiolane was statistically on par with edifenphos and propiconazole while kasugamycin and carbendazim had not shown any significant difference between them. There was no significant difference between propiconazole and kasugamycin. Difenconazole + propiconazole 30 EC (12.66%) was next in the order of its efficacy followed by carbendazim + epoxiconazole (13.16%), difenconazole 25 EC (14.49%) and epoxiconazole 25 EC (14.83%). However, difenconazole in combination with propiconazole exhibited superior efficacy over difenconazole alone, while epoxiconazole in combination with carbendazim was also superior to epoxiconazole alone. There was no

significant difference between difenconazole + propiconazole and carbendazim + epoxiconazole; difenconazole and epoxiconazole, respectively. Diniconazole 25 WP (16.16%) was significantly inferior to all other fungicides, but it was found superior to untreated control (20.99%).

At 20 DASP, the per cent leaf blast severity ranged between 7.99 (pyroquilon) and 36.49 (untreated control). Pyroquilon 50 WP (7.99%) and azoxystrobin 25 SC (8.41%) excellent followed by pyraclostrobin 25 EC (9.66%), RIL010 (10.66%), tricyclazole 75 WP (10.66%) and fenoxanil 20 SC (12.83%). Pyroquilon was statistically on par with azoxystrobin while there was no significant difference among pyraclostrobin, tricyclazole and RIL 010. But each group significantly differed with the other and also with fenoxanil. Iprobenfos 48 EC and edifenphos 50 EC (14.66%) were the next best fungicides followed by isoprothiolane 40 EC (14.83%), propiconazole 25 EC (15.66%), difenconazole + propiconazole 30 EC (16.49%), kasugamycin 2 L (16.83%) and carbendazim 50 WP (17.16%). However, there was no significant difference among iprobenfos, edifenphos, isoprothiolane and propiconazole while kasugamycin, difenconazole + propiconazole and carbendazim were on par with each other. Carbendazim + epoxiconazole 25 EC (18.66%) was next in the order of its efficacy followed by difenconazole 25 EC (22.16%), epoxiconazole 7.5 EC (23.33%) and diniconazole 25 WP (27.16%). However, epoxiconazole was inferior to its combination with carbendazim and difenconazole was inferior to its combination with propiconazole, respectively. Difenconazole was statistically on par with epoxiconazole. Diniconazole 25 WP (27.16%) was significantly inferior to all other fungicides, but superior to the untreated control (36.49%).

Table 25: Efficacy of fungicides as foliar spray against rice blast in the main field (*Kharif*, 2002 & 2003; pooled data)

S. No.	Name of the fungicide	Disease incidence before spray	Leaf blast severity (%)*			Neck blast incidence (%)		Grain yield (kg/ha)		
			10 DASP	20 DASP	30 DASP	Lesioned necks	Broken necks	Ill-filled grains	Filled grains	Total
1	Pyroquilon 50 WP	0.80 (5.16)	5.33 (13.31)	7.99 (16.43)	11.83 (20.09)	0.00	0.00	24.99	2922.99	2947.99
2	Azoxystrobin 25 SC	0.80 (5.13)	6.16 (14.42)	8.41 (16.85)	12.66 (20.88)	0.00	0.00	27.89	2841.66	2869.56
3	Pyraclostrobin 25 EC	0.75 (4.97)	6.49 (14.77)	9.66 (18.15)	13.83 (21.81)	0.00	0.00	32.49	2806.33	2838.82
4	RIL 010 25 SC	1.00 (5.74)	6.83 (15.12)	10.66 (19.09)	14.83 (22.63)	0.00	0.00	29.16	2786.66	2815.82
5	Tricyclazole 75 WP	0.65 (4.62)	8.49 (16.95)	10.66 (19.09)	15.49 (23.19)	2.33	0.00	37.49	2683.16	2720.65
6	Fenoxanil 20 SC	1.15 (6.02)	8.49 (16.95)	12.83 (20.96)	18.66 (25.62)	4.5	1.83	47.99	2570.16	2618.15
7	Iprobenfos 48 EC	1.20 (6.29)	8.83 (17.26)	14.66 (22.55)	20.83 (27.13)	4.99	0.00	48.49	2562.66	2611.16
8	Isoprothiolane 40 EC	0.85 (5.29)	9.66 (18.15)	14.83 (22.63)	22.66 (28.45)	2.50	0.00	42.66	2566.32	2608.99
9	Edifenphos 50 EC	0.50 (4.05)	9.83 (18.24)	14.66 (22.55)	23.16 (28.79)	5.49	0.00	50.49	2497.83	2548.32
10	Propiconazole 25 EC	0.75 (4.97)	10.16 (18.63)	15.66 (23.34)	23.83 (29.20)	4.49	0.33	51.83	2452.66	2504.49
11	Kasugamycin 2L	1.20 (6.29)	10.66 (19.09)	16.83 (24.20)	25.49 (30.33)	6.66	0.49	67.66	2373.66	2441.32
12	Carbendazim 50 WP	0.70 (4.80)	11.33 (19.64)	17.16 (24.50)	29.90 (33.15)	7.16	1.66	59.16	2298.83	2357.99
13	Difenconazole + Propiconazole 30 EC	0.55 (4.25)	12.66 (20.88)	16.49 (23.97)	29.99 (33.21)	6.66	2.33	87.49	2276.83	2364.32
14	Carbendazim + Epoxiconazole 25 EC	0.85 (5.29)	13.16 (21.30)	18.66 (25.62)	27.99 (31.95)	9.49	3.66	103.99	2173.49	2277.49
15	Difenconazole 25 EC	1.10 (6.02)	14.49 (22.38)	22.16 (28.11)	33.66 (35.49)	12.16	5.99	122.49	1946.49	2068.98
16	Epoxiconazole 7.5 EC	1.00 (5.74)	14.83 (22.63)	23.33 (28.86)	36.33 (37.05)	16.33	8.66	155.99	1838.83	1994.83
17	Diniconazole 25 WP	0.80 (5.13)	16.16 (23.73)	27.16 (31.44)	37.16 (37.58)	15.33	8.66	157.49	1840.33	1997.82
18	Control	0.60 (4.44)	20.99 (27.28)	36.49 (37.17)	52.16 (46.26)	22.49	17.66	372.48	1506.49	1878.97
SEm±		0.07	0.21	0.35	0.36	0.27	0.13	2.32	25.79	29.79
CD (P=0.05)		ns	0.61	1.00	1.04	0.77	0.37	6.67	74.14	85.64

*Figures in parentheses are arc sin transformed values; DASP = Number of days after spray of fungicide

Fig. 22: Progress of leaf blast severity in relation to efficacy of fungicides against leaf blast in the main field (*Kharif* , 2002&03)

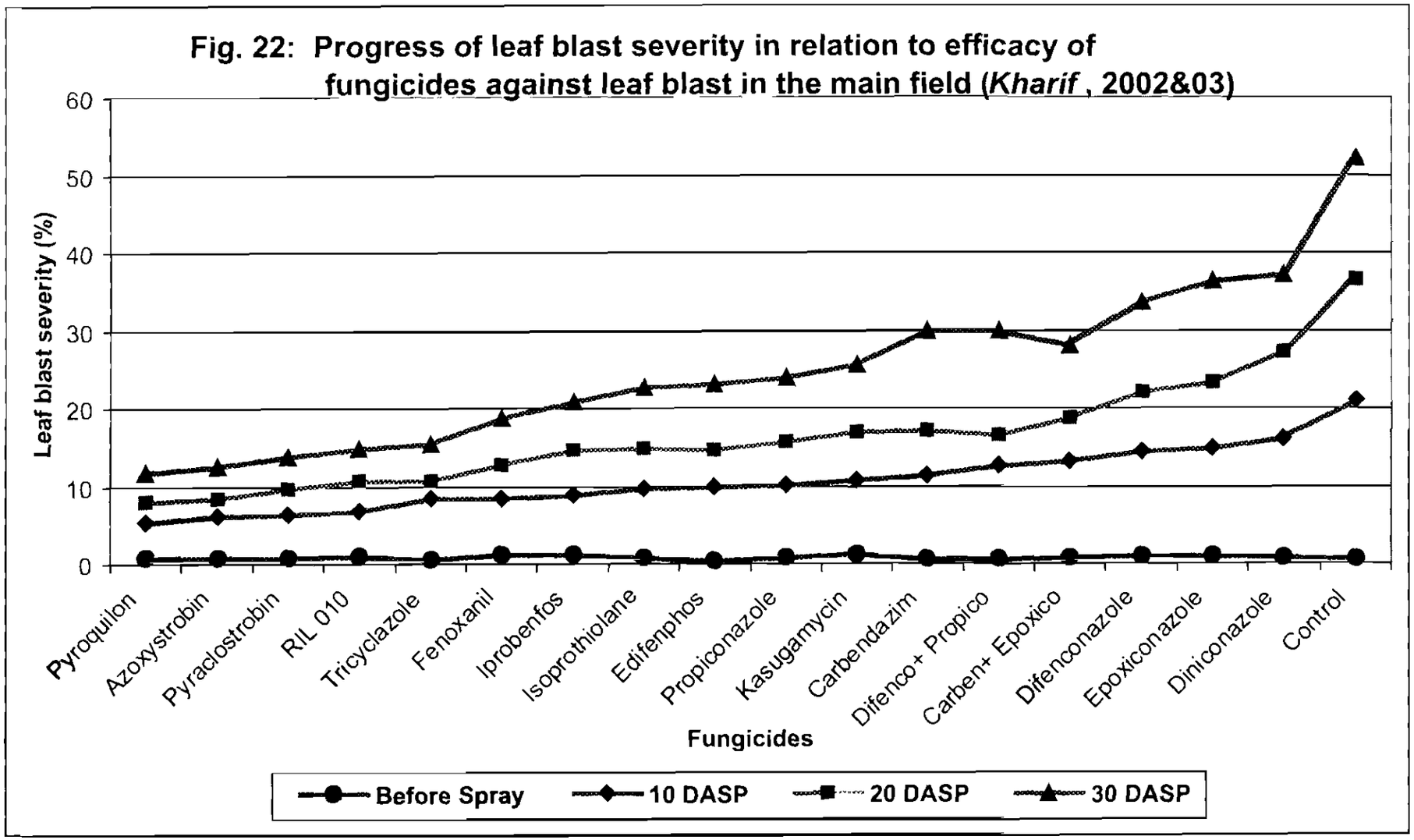


Fig. 23: Neck blast incidence in relation to efficacy of fungicides against rice blast in the main field (*Kharif* , 2002&03)

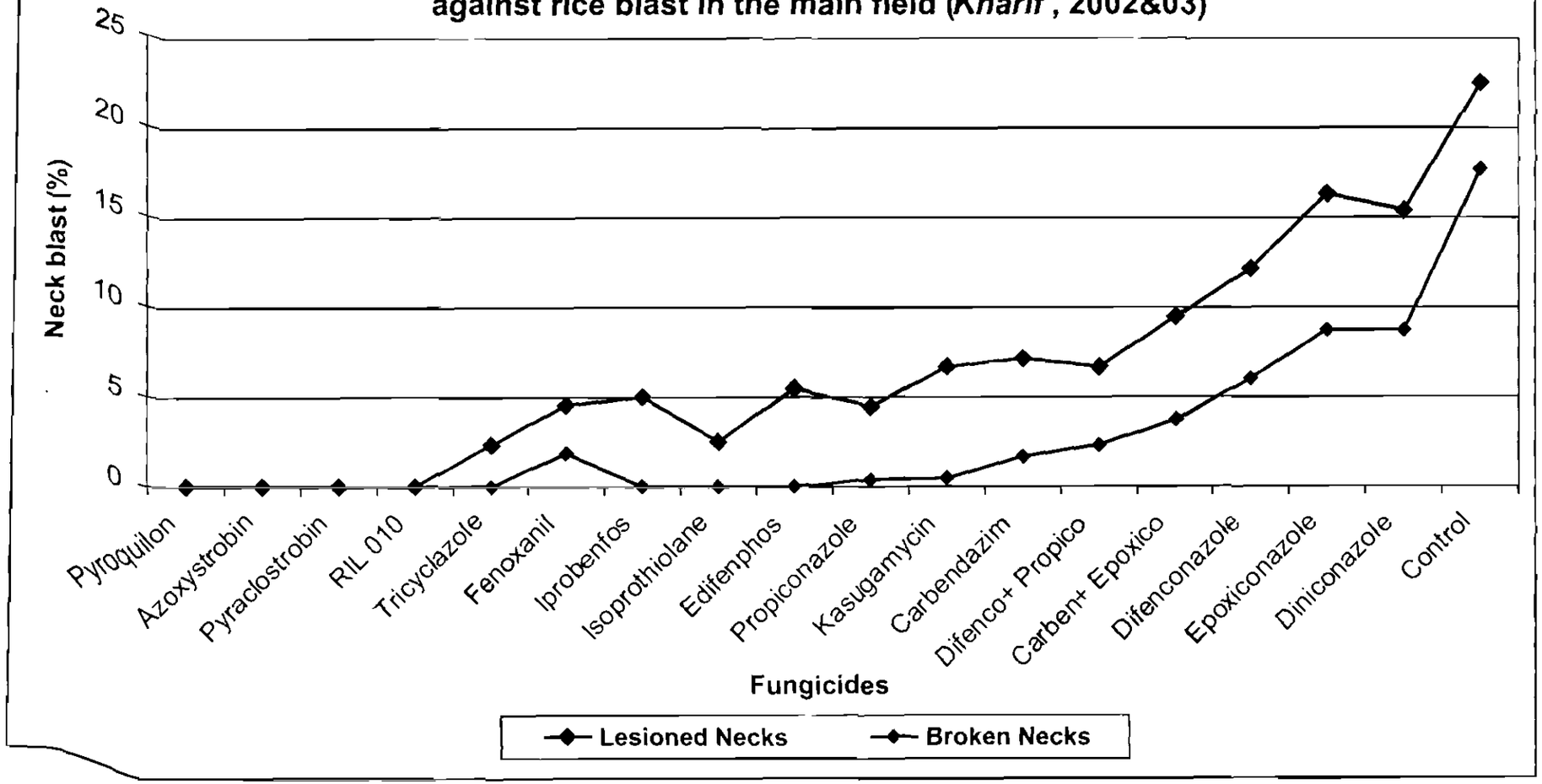
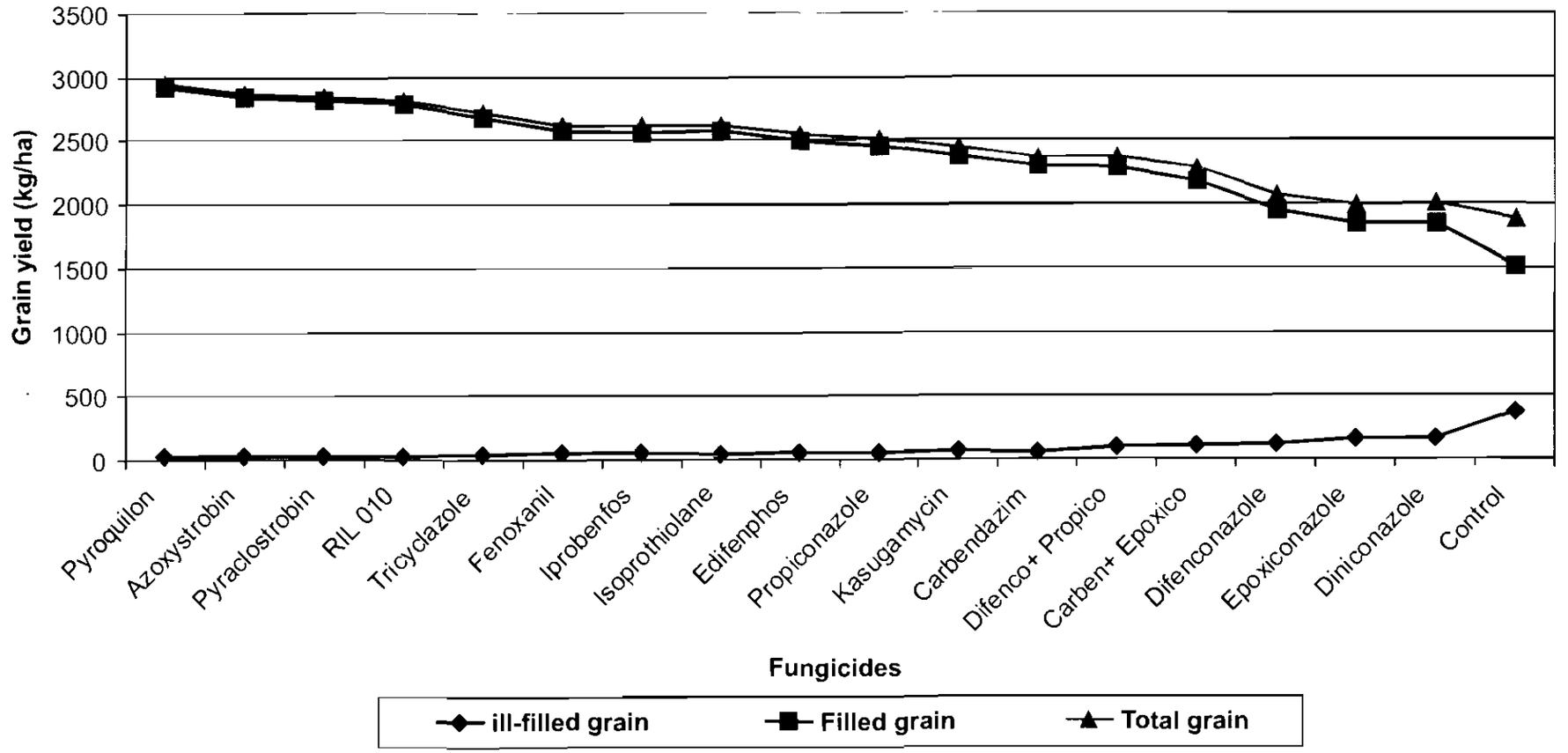


Fig. 24: Grain yield (kg/ha) in relation to efficacy of fungicides against rice blast in the main field (*Kharif*, 2002&03)



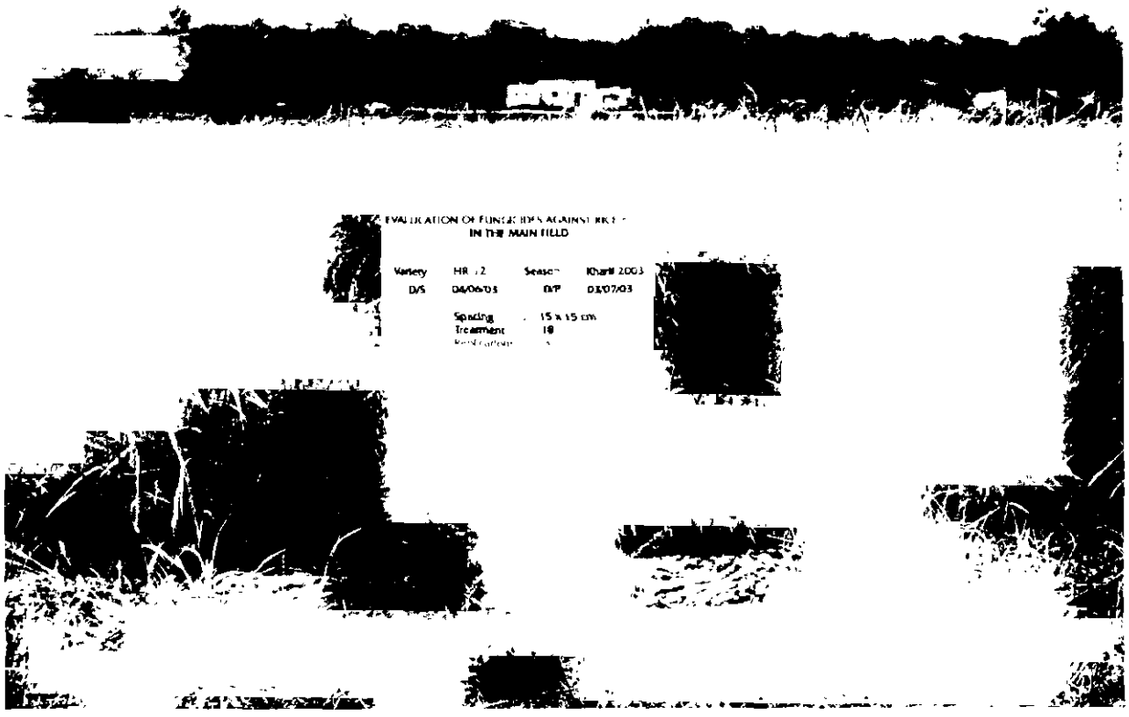


Plate 30: Evaluation of fungicides against rice blast in the main field

At 30 DASP, the per cent leaf blast severity ranged between 11.83 (pyroquilon 50 WP) and 52.16 (untreated control). Pyroquilon 50 WP (11.83%) recorded the lowest leaf blast severity followed by azoxystrobin 25 SC (12.66%), pyraclostrobin 25 EC (13.83%), RIL 010 (14.83%) and tricyclazole 75 WP (15.49%). There was no significant difference between pyroquilon and azoxystrobin; azoxystrobin and pyraclostrobin; pyraclostrobin and RIL 010, respectively, but each fungicide differed significantly with the other alternately in the order of their efficacy. Fenoxanil 20 SC (18.66%) was the next best fungicide followed by iprobenfos 48 EC (20.83%), isoprothiolane 40 EC (22.66%), edifenphos 50 EC (23.16%), propiconazole 25 EC (23.83%) and kasugamycin 2 L (25.49%). However, fenoxanil and iprobenfos differed significantly with each other, but found superior to rest of the fungicides in the order of their efficacy. Isoprothiolane, edifenphos and propiconazole were statistically on par with each other but found significantly superior to kasugamycin. Surprisingly for the first time, carbendazim + epoxiconazole 25 EC (27.99%) was found significantly superior to carbendazim 50 WP alone (29.9%). Difenconazole + propiconazole 30 EC (29.99%) was next in the order of its efficacy followed by difenconazole 25 EC (33.66%), epoxiconazole 7.5 EC (36.33%) and diniconazole 25 WP (37.16%). Epoxiconazole was found statistically on par with diniconazole but it was significantly inferior to difenconazole.

4.5.3.8 Efficacy of fungicides on neck blast incidence (*Kharif*, 2002&2003; pooled data)

Neck blast incidence was measured in terms of lesioned necks and broken necks before the actual harvest of the crop. The per cent lesioned necks varied from zero to 22.49 while the per cent broken necks ranged between zero and 17.66 (Table 25). The number of lesioned necks were nil in the plots treated with pyroquilon 50 WP, azoxystrobin 25 SC, pyraclostrobin 25 EC and RIL 010 25 SC. Tricyclazole 75 WP

(2.33%) registered the lowest number of lesioned necks followed by isoprothiolane 40 EC (2.5%), propiconazole 25 EC (4.49%), fenoxanil 20 SC (4.5%), iprobenfos 48 EC (4.99%), and edifenphos 50 WP (5.49%). However, tricyclazole was statistically on par with isoprothiolane and there was no significant difference among fenoxanil, iprobenfos and propiconazole while edifenphos was statistically on par with iprobenfos and propiconazole. Kasugamycin 2 L and difenconazole + propiconazole 30 EC (6.66%) were statistically on par with carbendazim 50 WP (7.16%), but inferior to edifenphos. Carbendazim + epoxiconazole 25 EC (9.49%) registered the next higher numbers of lesioned necks followed by difenconazole 25 EC (12.16%) and diniconazole 25 WP (15.33%) and epoxiconazole 7.5 EC (16.33%). However, they had significantly differed with one another. Epoxiconazole 7.5 EC (16.33%) recorded the highest number of lesioned necks, but it was superior to untreated control (22.49%).

There was absolutely no broken necks in the plots treated with pyroquilon 50 WP, azoxystrobin 25 SC, pyraclostrobin 25 EC, RIL 010 25 SC, tricyclazole 75 WP, iprobenfos 48 EC, isoprothiolane 40 EC and edifenphos 50 EC. Propiconazole 25 EC (0.33%) and kasugamycin 2 L (0.49%) have recorded the lowest number of broken necks followed by carbendazim 50 WP (1.66%) and fenoxanil 20 SC (1.83%). However, propiconazole was statistically on par with kasugamycin while there was no significant difference between fenoxanil and carbendazim, but each group differed significantly with the other. Difenconazole + propiconazole 30 EC (2.33%) recorded the next higher numbers of lesioned necks followed by carbendazim + epoxiconazole 25 EC (3.66%), difenconazole 25 EC (5.99%) epoxiconazole 7.5 EC and diniconazole 25 WP (8.66%). However, each fungicide significantly differed with the other except epoxiconazole and diniconazole while they had been superior to the untreated control (17.66%).

4.5.3.9 Efficacy of fungicides in relation to grain yield (*Kharif*, 2002&2003; pooled data)

The amount of ill-filled grain varied from 24.99 kg/ha to 372.48 kg/ha while the quantity of filled grains ranged between 2922.99 kg/ha and 1506.49 kg/ha in pyroquilon 50 WP and control plots, respectively. Similarly, the total grain yield varied from 2947.99 kg/ha and 1878.97 kg/ha in pyroquilon 50 WP and untreated control, respectively (Table 25). All the fungicides tested were significantly superior compared to control. Pyroquilon 50 WP (24.99 kg/ha) recorded the lowest quantity of ill-filled grain followed by azoxystrobin 25 SC (27.89 kg/ha), RIL 010 25 SC (29.16 kg/ha), pyraclostrobin 25 EC (32.49 kg/ha) and tricyclazole 75 WP (37.49 kg/ha). There was no significant difference among pyroquilon, azoxystrobin and RIL 010 while pyraclostrobin was statistically on par with azoxystrobin as well as tricyclazole. Isoprothiolane 40 EC (42.66 kg/ha) recorded the next lowest quantity of ill-filled grain followed by fenoxanil 20 EC (47.99 kg/ha), iprobenfos 48 EC (48.99 kg/ha), edifenphos 50 WP (50.49 kg/ha) and propiconazole 25 EC (51.83 kg/ha). There was no significant difference among isoprothiolane, fenoxanil and iprobenfos; similarly, fenoxanil, iprobenfos, edifenphos and propiconazole did not differ significantly with one another. Interestingly, isoprothiolane had performed on par with tricyclazole in reducing the amount of ill filled grain. Carbendazim 50 WP (59.16 kg/ha), kasugamycin 2 L (67.66 kg/ha), difenconazole + propiconazole 30 EC (87.49 kg/ha), carbendazim + epoxiconazole 25 EC (103.99 kg/ha), difenconazole 25 EC (122.49 kg/ha), epoxiconazole 7.5 EC (155.99 kg/ha) and diniconazole 25 WP (157.49 kg/ha) were in the order of increasing quantities of ill-filled grain and differed significantly with one another except epoxiconazole and diniconazole.

Pyroquilon 50 WP (2922.99 kg/ha) has given the maximum quantity of filled grain where as the control plot registered the lowest (1506.49 kg/ha). Azoxystrobin 25 SC (2841.66 kg/ha) recorded the next highest amount of filled grain followed by pyraclostrobin 25 EC (2806.33 kg/ha) and RIL 010 25 SC (2786.66 kg/ha). Pyroquilon was statistically on par with azoxystrobin while there was no significant difference among azoxystrobin, pyraclostrobin and RIL 010, but these fungicides were significantly superior to rest of the fungicides. Tricyclazole 75 WP (2683.16 kg/ha), fenoxanil 20 SC (2570.16 kg/ha), isoprothiolane 40 EC (2566.32 kg/ha), iprobenfos 48 EC (2562.66 kg/ha), edifenphos 50 EC (2497.83 kg/ha), propiconazole 25 EC (2452.66 kg/ha) and kasugamycin 2 L (2373.66 kg/ha) had recorded the next higher quantities of filled grain. However, there was no significant difference among fenoxanil, isoprothiolane, iprobenfos and edifenphos, but these four fungicides were significantly superior to kasugamycin. Propiconazole and edifenphos were statistically on par with each other. Carbendazim 50 WP (2298.83 kg/ha), difenconazole + propiconazole 30 EC (2276.83 kg/ha), carbendazim + epoxiconazole 25 EC (2173.49 kg/ha), difenconazole 25 EC (1946.49 kg/ha) and diniconazole 25 WP (1840.33 kg/ha) and epoxiconazole 25 EC (1838.83 kg/ha) were in the decreasing order with reference to the quantities of filled grain. All of them differed significantly with each other except carbendazim and difenconazole + propiconazole; epoxiconazole and diniconazole, respectively.

The same trend was reflected in case of total grain yield also (Table 25). Pyroquilon 50 WP (2947.99 kg/ha) was superior to other fungicides where as the untreated control recorded a total grain yield of 1878.97 kg/ha. Azoxystrobin 25 SC

(2869.56 kg/ha), pyraclostrobin 25 EC (2838.82 kg/ha) and RIL 010 25 SC (2815.82 kg/ha) were statistically on par with each other and significantly superior to rest of the fungicides. Tricyclazole 75 WP (2720.65 kg/ha), fenoxanil 20 SC (2618.15 kg/ha), iprobenfos 48 EC (2611.16 kg/ha), isoprothiolane 40 EC (2608.99 kg/ha), edifenphos 50 EC (2548.32 kg/ha) and propiconazole 25 EC (2504.49 kg/ha) were next in the decreasing order of their efficacy. However, there was no significant difference among fenoxanil, iprobenfos, isoprothiolane, fenoxanil and edifenphos, but statistically inferior to tricyclazole while propiconazole was statistically on par edifenphos. Kasugamycin 2 L (2441.32 kg/ha), carbendazim 50 WP (2357.99 kg/ha), difenconazole + propiconazole 30 EC (2364.32 kg/ha), carbendazim + epoxiconazole 25 EC (2277.49 kg/ha), difenconazole 25 EC (2068.98 kg/ha), diniconazole 25 WP (1997.82 kg/ha) and epoxiconazole 7.5 EC (1994.83 kg/ha) were in the next in the order of decreasing efficacy. Kasugamycin was found on par with carbendazim while carbendazim was in turn on par with difenconazole + propiconazole and carbendazim + epoxiconazole. There was no significant difference among difenconazole, diniconazole and epoxiconazole, all these were found significantly superior to untreated control (1879.97 kg/ha).

4.6 RELATIVE SENSITIVITY OF P. GRISEA ISOLATES TO DIFFERENT FUNGICIDES

Rice blast samples were collected from 12 different parts of India including Andhra Pradesh. Single spore isolations of *P. grisea* were made to test their sensitivity towards carbendazim, edifenphos and iprobenfos at the concentrations of 0, 1, 5, 10, 25, 50, 100, 250, 500 and 1000 µg/ml using oat meal agar (OMA) medium in the

laboratory. The mycelial colony diameter was recorded at different concentrations and per cent growth inhibition was calculated in each treatment by comparison with control plates. Similarly, per cent inhibition of spore germination was also calculated. The individual ED_{50} values were derived from the dose response curves. The results were presented in the Tables 26, 28 and 30. Rice blast samples were also collected from three different locations in Andhra Pradesh *viz*. Kadapa, Naidupeta and Tirupathi to know the base line sensitivity as the samples were collected from the rice fields that had not received any fungicide sprays. The results of base line sensitivity for carbendazim, edifenphos and iprobenfos were presented in the Tables 27, 29 and 31 (Plates 31 to 39).

4.6.1 Relative sensitivity of *P. grisea* isolates to carbendazim

A highly marked and varied response of different isolates was observed with respect to their sensitivity towards carbendazim. The ED_{50} values of these isolates ranged between 0.39-54.63 µg/ml and 0.55-56.18 µg/ml for mycelial growth (poisoned food) and spore germination, respectively. The minimum inhibitory concentrations (MIC) for the complete inhibition of both mycelial growth and spore germination ranged between 5-500 µg/ml (Table 26).

Cuttak isolate recorded the highest ED_{50} values of 54.63 and 56.18 µg/ml for mycelial growth (poisoned food) and spore germination, respectively. Where as, the minimum inhibitory concentration (MIC) for both mycelial growth and spore germination of this isolate was 500 µg/ml. Malan isolate recorded the next highest ED_{50} values of 43.92 and 43.95 g/ml for mycelial growth and spore germination, respectively, followed by Nellore (29.25 and 31.73 µg/ml), Gerua (16.41 and 16.47 µg/ml), Mandya (13.10 and

Table 26: Relative sensitivity of *Pyricularia grisea* isolates to carbendazim *in vitro*.

S. No.	Name of the isolate	Mycelial growth (poisoned food)		Spore germination	
		ED_{50} (µg/ml)	MIC (µg/ml)	ED_{50} (µg/ml)	MIC (µg/ml)
1	Cuttak	54.63	500	56.18	500
2	Malan	43.92	500	43.95	500
3	Nellore	29.25	250	31.73	250
4	Gerua	16.41	250	16.47	250
5	Mandya	13.10	250	13.51	250
6	Almora	12.48	250	13.28	250
7	Pondicherry	7.91	100	7.92	100
8	Maruteru	7.79	100	8.87	100
9	Kapurthala	5.59	50	5.74	50
10	Rajendranagar	2.04	10	2.09	10
11	Pattambi	1.68	10	1.76	10
12	Medchel	0.39	5	0.55	5

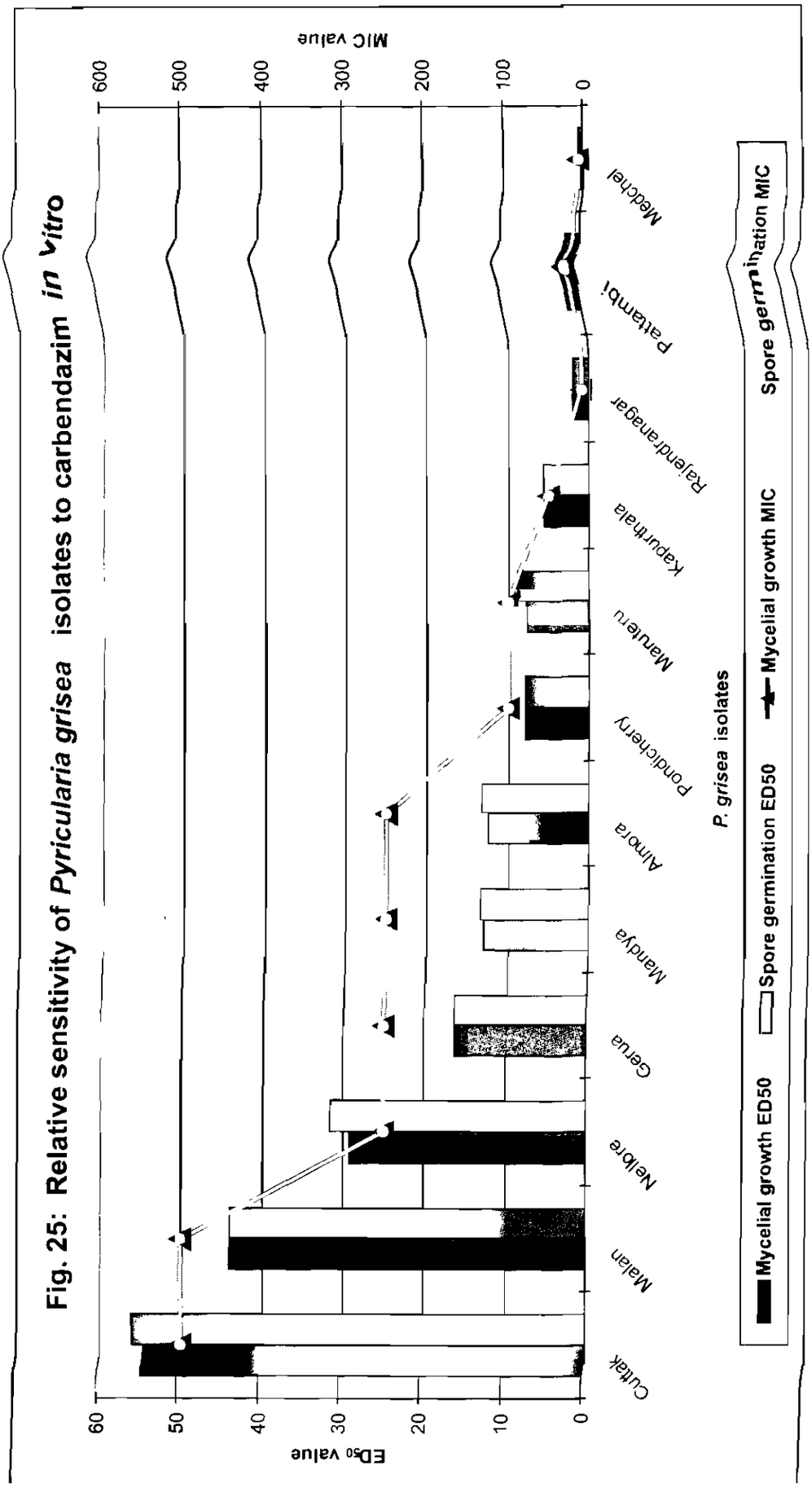


Fig. 25: Relative sensitivity of *Pyricularia grisea* isolates to carbendazim *in vitro*

Table 27: *In vitro* sensitivity of three reference sensitive isolates of *Pyricularia grisea* to Carbendazim

Name of the isolate	Poisoned food technique					Spore germination method				
	Average colony diameter (mm)			ED_{50} (µg/ml)	MIC (µg/ml)	Conidia germinated (%)			ED_{50} (µg/ml)	MIC (µg/ml)
	Control	1 ppm	5 ppm			Control	1 ppm	5 ppm		
Kadapa	89.00	23.33	0	0.57	5	100	32.67	0	0.68	5
Naidupeta	90.00	20.00	0	0.40	5	100	32.67	0	0.68	5
Tirupathi	88.60	28.33	0	0.65	5	100	36.33	0	0.75	5
Mean	89.20	23.89	0	0.54	5	100	33.89	0	0.70	5

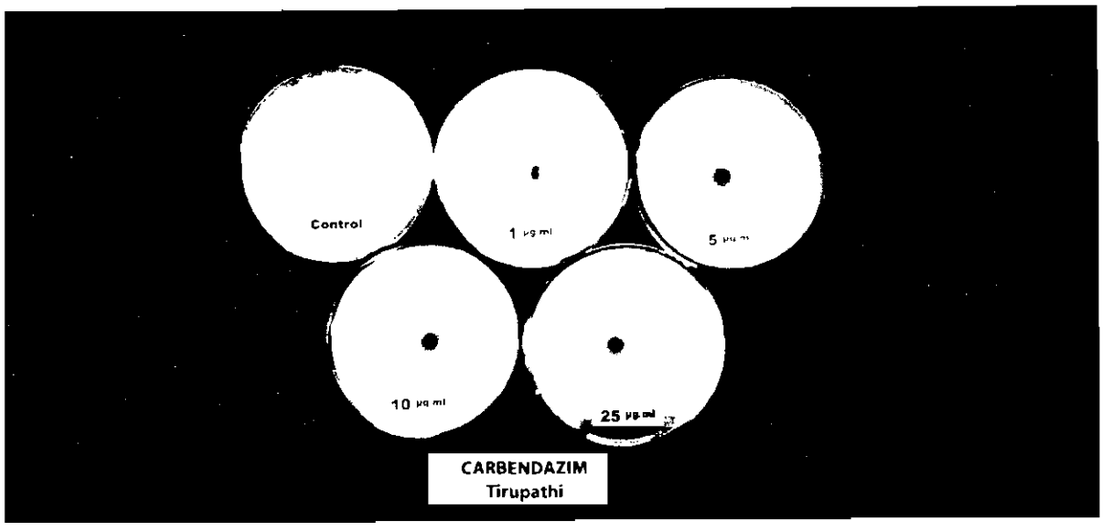


Plate 31: *In vitro* sensitivity of Tirupathi isolate towards carbendazim

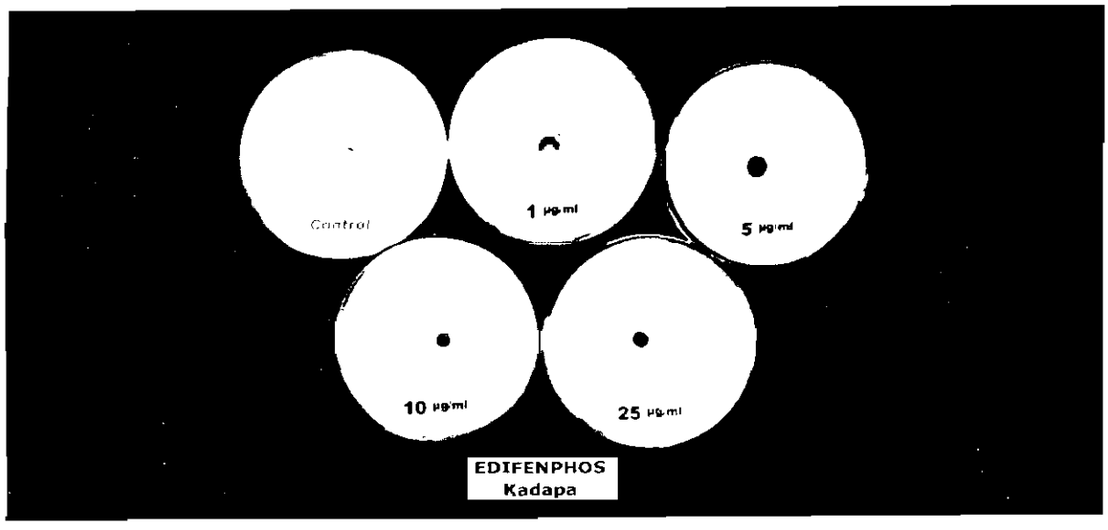


Plate 32: *In vitro* sensitivity of Kadapa isolate towards edifenphos

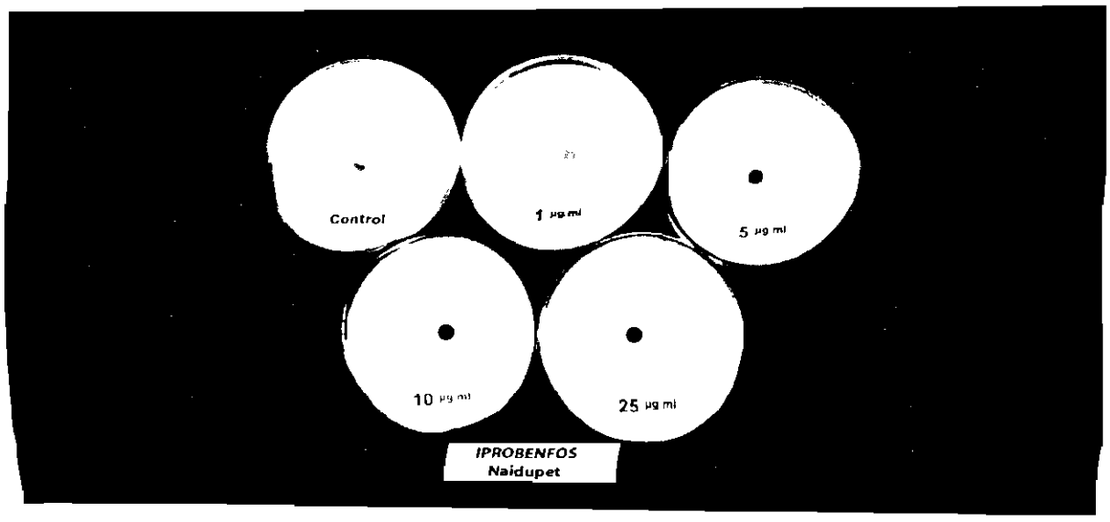


Plate 33: *In vitro* sensitivity of Naidupet isolate towards iprobenfos

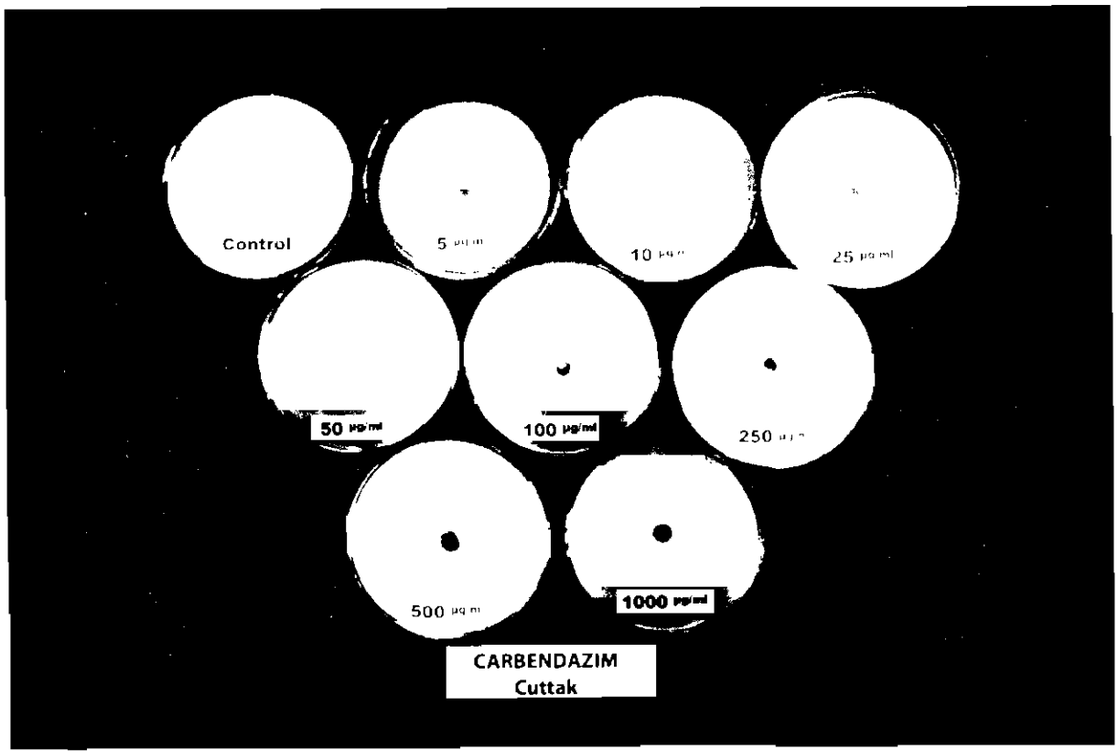


Plate 34: Inhibition of mycelial growth by Cuttak isolate (highly resistant) at different concentrations of carbendazim

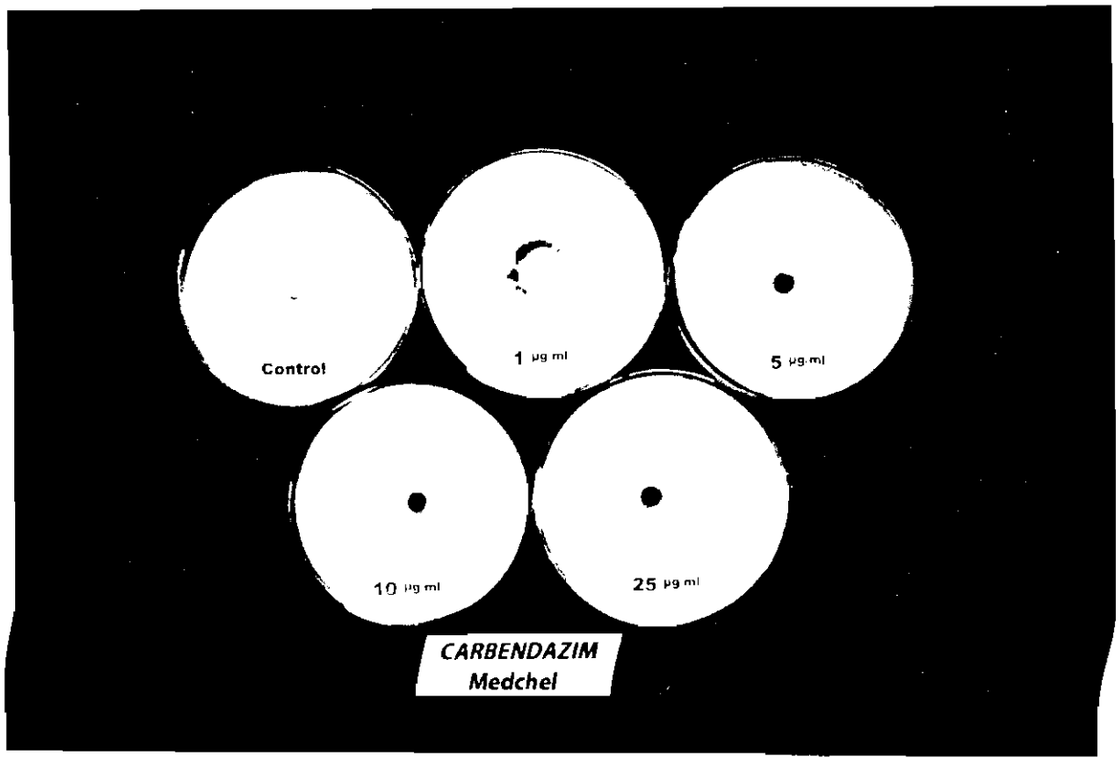


Plate 35: Inhibition of mycelial growth by Medchel isolate (highly sensitive) at different concentrations of carbendazim

13.51 µg/ml) and Almora (12.48 and 13.28 µg/ml) isolates in the decreasing order of their resistance. The MIC value for inhibition of both the mycelial growth and spore germination of Malan isolate was 500 µg/ml while it was 250 µg/ml in case of Nellore, Gerua, Mandya and Almora isolates (Plates 34 & 35).

Medchel isolate recorded the lowest ED_{50} values of 0.39 and 0.55 µg/ml for inhibition of mycelial growth (poisoned food) and spore germination, respectively. Where as, the MIC value for the complete inhibition of mycelial growth and spore germination of this isolate was 5 µg/ml. Pattambi isolate recorded the next lowest ED_{50} values of 1.68 and 1.76 µg/ml for mycelial growth (poisoned food) and spore germination, respectively, followed by Rajendranagar (2.04 and 2.09 µg/ml), Kapurthala (5.59 and 5.74 µg/ml), Maruteru (7.79 and 8.87 µg/ml) and Pondicherry (7.91 and 7.92 µg/ml) isolates in the increasing order of their resistance. The MIC value for the complete inhibition of mycelial growth and spore germination of Pattambi and Rajendranagar isolates was 10 µg/ml followed by Kapurthala (50 µg/ml) and Maruteru and Pondicherry (100 µg/ml) isolates.

4.6.2 Base line sensitivity for carbendazim

The response of three reference isolates *viz.* Kadapa, Naidupeta and Tirupathi at different concentrations of carbendazim was determined. The ED_{50} values of these isolates ranged between 0.40-0.65 µg/ml and 0.68-0.75 µg/ml with an average of 0.54 and 0.70 µg/ml for mycelial growth and spore germination, respectively (Table 27). Whereas, the MIC value for the complete inhibition of both mycelial growth and spore germination of all the three isolates was 5 µg/ml (Plate 31).

4.6.3 Relative sensitivity of *P. grisea* isolates to edifenphos

A highly marked and varied response of different isolates was observed with respect to their sensitivity towards edifenphos. The ED_{50} values of these isolates ranged between 0.73-30.56 µg/ml and 0.77-32.35 µg/ml for inhibition of mycelial growth and spore germination, respectively. Where as, the minimum inhibitory concentrations (MIC) for the complete inhibition of mycelial growth and spore germination of these isolates ranged between 5-250 µg/ml (Table 28).

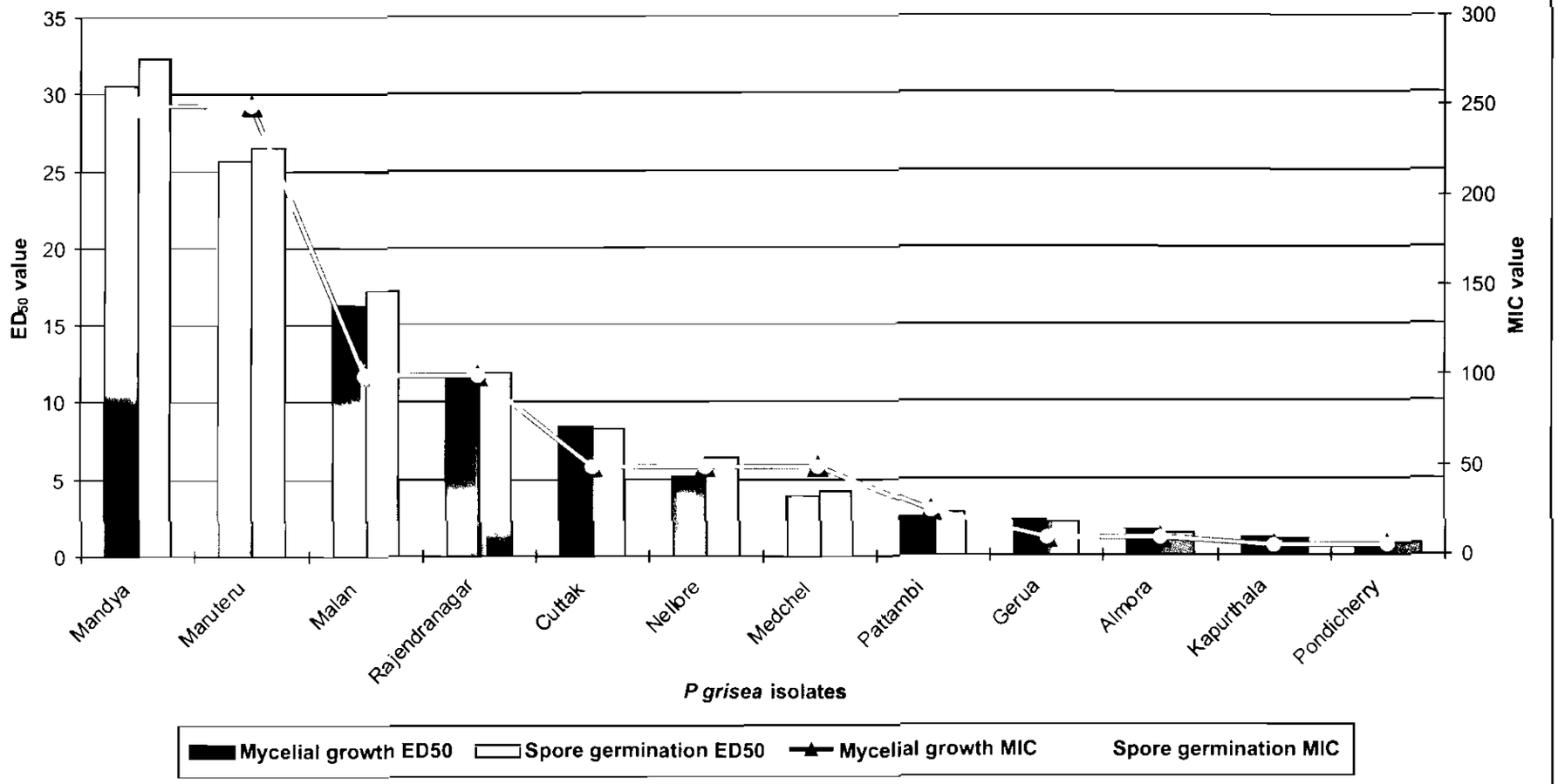
Mandya isolate recorded the highest ED_{50} values of 30.56 and 32.35 µg/ml for mycelial growth (poisoned food) and spore germination, respectively. Where as, the minimum inhibitory concentration (MIC) for the complete inhibition of mycelial growth and spore germination of this isolate was 250 µg/ml. Maruteru isolate recorded the next highest ED_{50} values of 25.64 and 26.49 µg/ml for mycelial growth and spore germination, respectively, followed by Malan (16.29 and 17.24 µg/ml), Rajendranagar (11.82 and 11.87 µg/ml), Cuttak (8.41 and 8.33 µg/ml) and Nellore (5.20 and 6.46 µg/ml) isolates in the decreasing order of their resistance towards edifenphos. Where as, the MIC value for complete inhibition of mycelial growth and spore germination of Maruteru isolate was 250 µg/ml while it was 100 µg/ml in case of Malan and Rajendranagar isolates followed by Cuttak and Nellore (50 µg/ml) isolates (Plates 36 & 37).

Pondicherry isolate had recorded the lowest ED_{50} values of 0.73 and 0.77 µg/ml for mycelial growth and spore germination, respectively. The MIC value for the complete inhibition of both mycelial growth and spore germination of this isolate was 5 µg/ml. Kapurthala isolate recorded the next lowest ED_{50} values of 1.16 and 1.00 µg/ml

Table 28: Relative sensitivity of *Pyricularia grisea* isolates to edifenphos *in vitro*.

S. No.	Name of the isolate	Mycelial growth (poisoned food)		Spore germination	
		ED_{50} (µg/ml)	MIC (µg/ml)	ED_{50} (µg/ml)	MIC (µg/ml)
1	Mandya	30.56	250	32.35	250
2	Maruteru	25.64	250	26.49	250
3	Malan	16.29	100	17.24	100
4	Rajendranagar	11.82	100	11.87	100
5	Cuttak	8.41	50	8.33	50
6	Nellore	5.20	50	6.46	50
7	Medchel	3.97	50	4.27	50
8	Pattambi	2.47	25	2.78	25
9	Gerua	2.23	10	2.21	10
10	Almora	1.64	10	1.49	10
11	Kapurthala	1.16	5	1.00	5
12	Pondicherry	0.73	5	0.77	5

Fig. 26: Relative sensitivity of *Pyricularia grisea* isolates to edifenphos *in vitro*



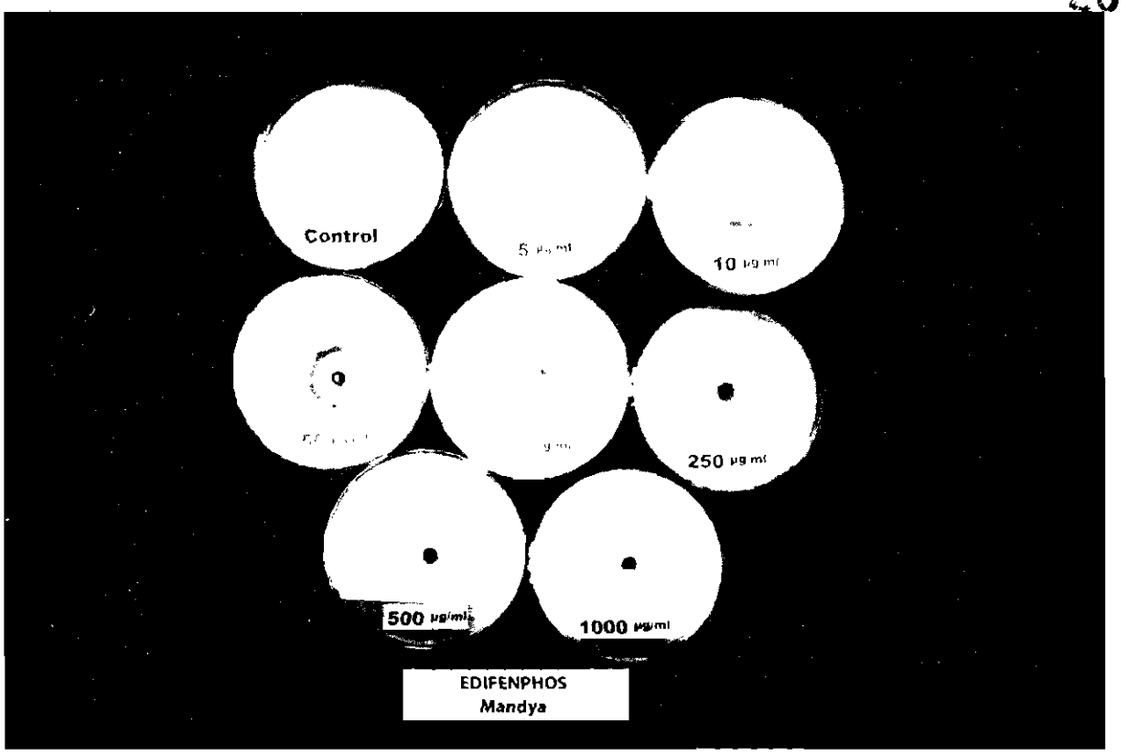


Plate 36: Inhibition of mycelial growth by Mandya isolate (highly resistant) at different concentrations of edifenphos

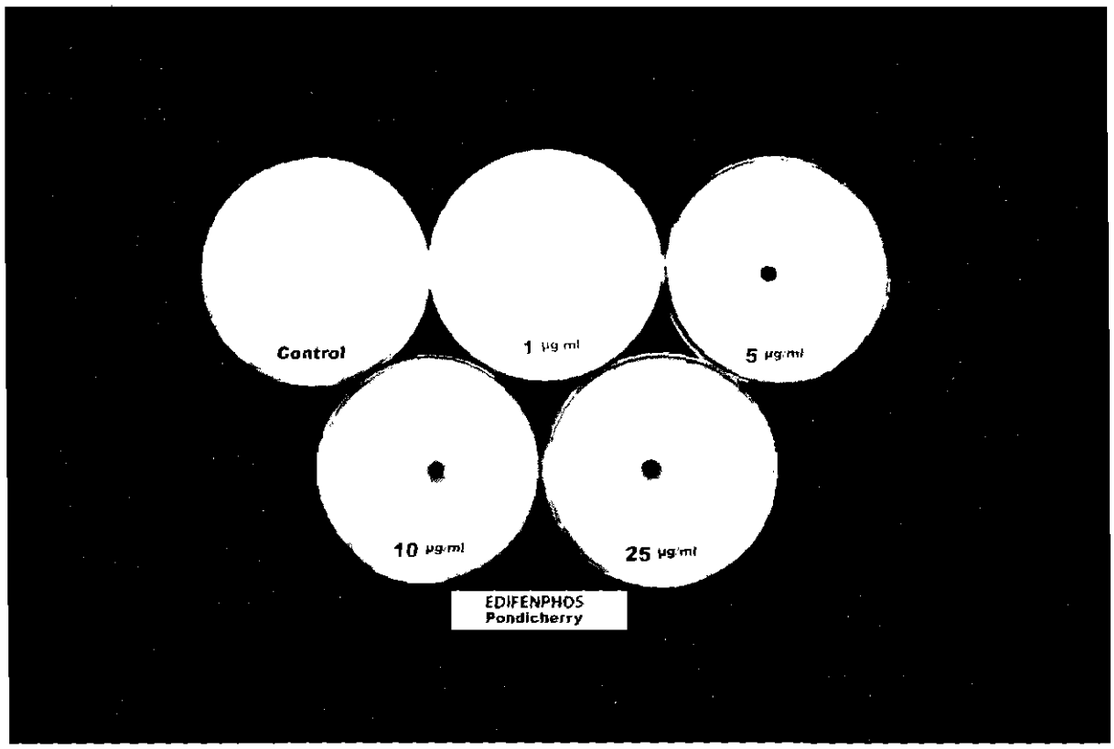


Plate 37: Inhibition of mycelial growth by Pondicherry isolate (highly sensitive) at different concentrations of edifenphos

Table 29: *In vitro* sensitivity of three reference sensitive isolates of *Pyricularia grisea* to Edifenphos

Name of the isolate	Poisoned food technique					Spore germination method				
	Average colony diameter (mm)			ED_{50} (µg/ml)	MIC (µg/ml)	Conidia germinated (%)			ED_{50} (µg/ml)	MIC (µg/ml)
	Control	1 ppm	5 ppm			Control	1 ppm	5 ppm		
Kadapa	90.00	20.00	0	0.46	5	100	29.33	0	0.64	5
Naidupeta	90.00	22.67	0	0.52	5	100	34.33	0	0.71	5
Tirupathi	89.00	25.33	0	0.57	5	100	31.66	0	0.66	5
Mean	89.67	22.67	0	0.52	5	100	31.77	0	0.67	5

for mycelial growth and spore germination, respectively, followed by Almora (1.64 and 1.49 µg/ml), Gerua (2.23 and 2.21 µg/ml), Pattambi (2.47 and 2.78 µg/ml) and Medchel (3.97 and 4.27 µg/ml) isolates in the increasing order of their tolerance towards edifenphos. The MIC value for the complete inhibition of both mycelial growth and spore germination of Kapurthala isolate was 5 µg/ml, while it was 10 µg/ml for Almora and Gerua isolates followed by Pattambi (25 µg/ml) and Medchel (50 µg/ml) isolates.

4.6.4 Base line Sensitivity for edifenphos

The response of three reference isolates *viz.* Kadapa, Naidupeta and Tirupathi at different concentrations of edifenphos was determined. The ED_{50} values of these isolates ranged between 0.46-0.57 µg/ml and 0.64-0.71 µg/ml with an average of 0.52 and 0.67 µg/ml for mycelial growth and spore germination, respectively (Table 29). Where as, the MIC value for the complete inhibition of both mycelial growth and spore germination of these three isolates was 5 µg/ml (Plate 32).

4.6.5 Relative sensitivity of P. *grisea* isolates to iprobenfos

A highly marked and varied response of different isolates was observed with respect to their sensitivity towards iprobenfos. The ED_{50} values of these isolates ranged between 0.66-31.85 µg/ml and 0.68-32.57 µg/ml for mycelial growth (poisoned food) and spore germination, respectively. Where as, the minimum inhibitory concentrations (MIC) for the complete inhibition of both mycelial growth and spore germination of these isolates ranged between 5-250 µg/ml (Table 30; Plates 38 & 39).

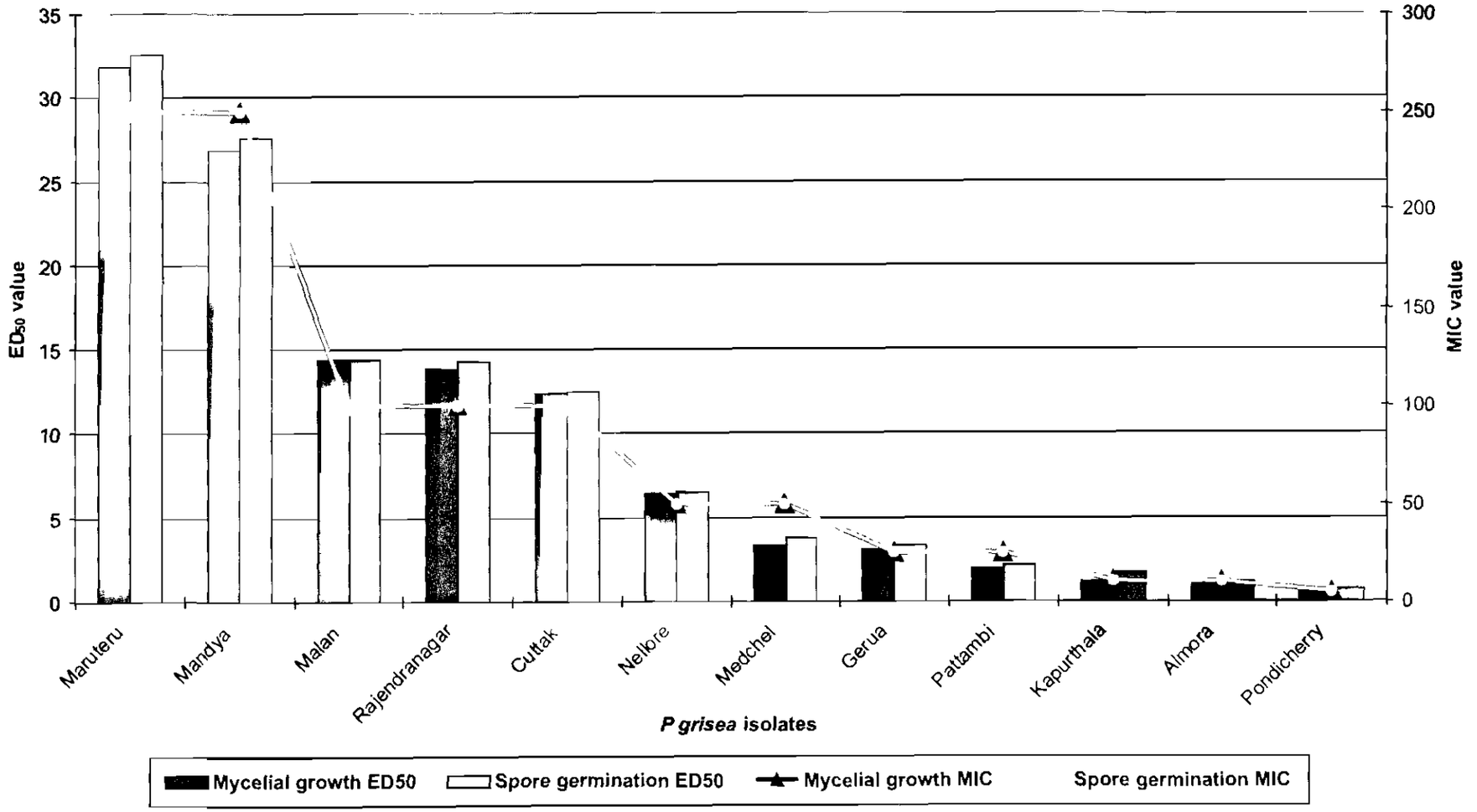
Maruteru isolate recorded the highest ED_{50} values of 31.85 and 32.57 µg/ml for mycelial growth and spore germination, respectively. Where as, the minimum inhibitory concentration (MIC) value for the complete inhibition of both mycelial growth and spore germination of this isolate was 250 µg/ml. Mandya isolate recorded the next highest ED_{50} values of 26.90 and 27.55 µg/ml for mycelial growth and spore germination, respectively, followed by Malan (14.42 and 14.35 µg/ml), Rajendranagar (13.81 and 14.26 µg/ml), Cuttak (12.40 and 12.47 µg/ml) and Nellore (6.38 and 6.49 µg/ml) isolates in the decreasing order of their resistance. The MIC value for both mycelial growth and spore germination of Mandya isolate was 250 µg/ml and it was 100 µg/ml in case of Malan, Rajendranagar and Cuttak isolates followed by Nellore isolate (50 µg/ml).

Pondicherry isolate recorded the lowest ED_{50} values of 0.66 and 0.68 µg/ml for mycelial growth and spore germination, respectively. Where as, the MIC value for both mycelial growth and spore germination of this isolate was 5 µg/ml. Almora isolate recorded the next lowest ED_{50} values of 1.11 and 1.20 µg/ml for mycelial growth and spore germination, respectively, followed by Kapurthala (1.20 and 1.70 µg/ml), Pattambi (2.00 and 2.21 µg/ml), Gerua (3.12 and 3.32 µg/ml) and Medchel (3.38 and 3.78 µg/ml) isolates in the increasing order of their tolerance towards iprobenfos. However, the MIC value for both mycelial growth and spore germination of Almora and Kapurthala isolates was same (10 µg/ml). The MIC value for both mycelial growth and spore germination of Pattambi and Gerua isolates was 25 µg/ml followed by Medchel isolate (50 µg/ml).

Table 30: Relative sensitivity of *Pyricularia grisea* isolates to iprobenfos *in vitro*.

S. No.	Name of the isolate	Mycelial growth (poisoned food)		Spore germination	
		ED_{50} (µg/ml)	MIC (µg/ml)	ED_{50} (µg/ml)	MIC (µg/ml)
1	Maruteru	31.85	250	32.57	250
2	Mandya	26.90	250	27.55	250
3	Malan	14.42	100	14.35	100
4	Rajendranagar	13.81	100	14.26	100
5	Cuttak	12.40	100	12.47	100
6	Nellore	6.39	50	6.49	50
7	Medchel	3.38	50	3.78	50
8	Gerua	3.12	25	3.32	25
9	Pattambi	2.00	25	2.21	25
10	Kapurthala	1.20	10	1.70	10
11	Almora	1.11	10	1.20	10
12	Pondicherry	0.66	5	0.68	5

Fig. 27: Relative sensitivity of *Pyricularia grisea* isolates to iprobenfos *in vitro*



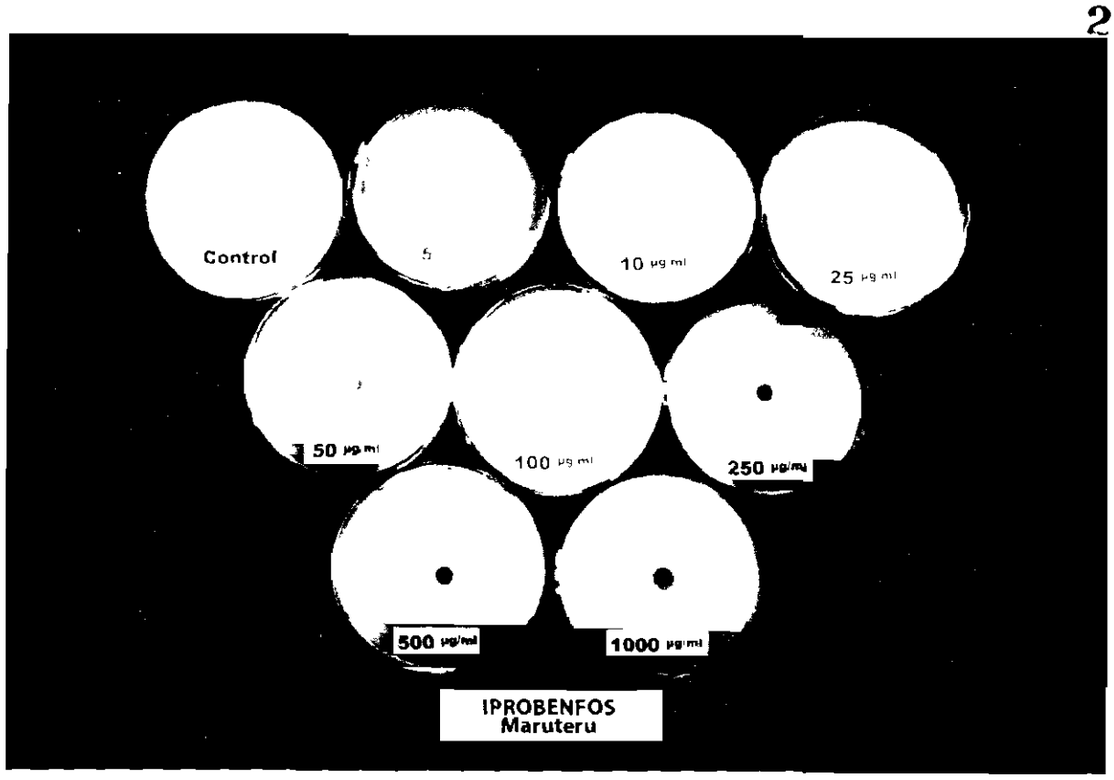


Plate 38: Inhibition of mycelial growth by Maruteru isolate (highly resistant) at different concentrations of iprobenfos

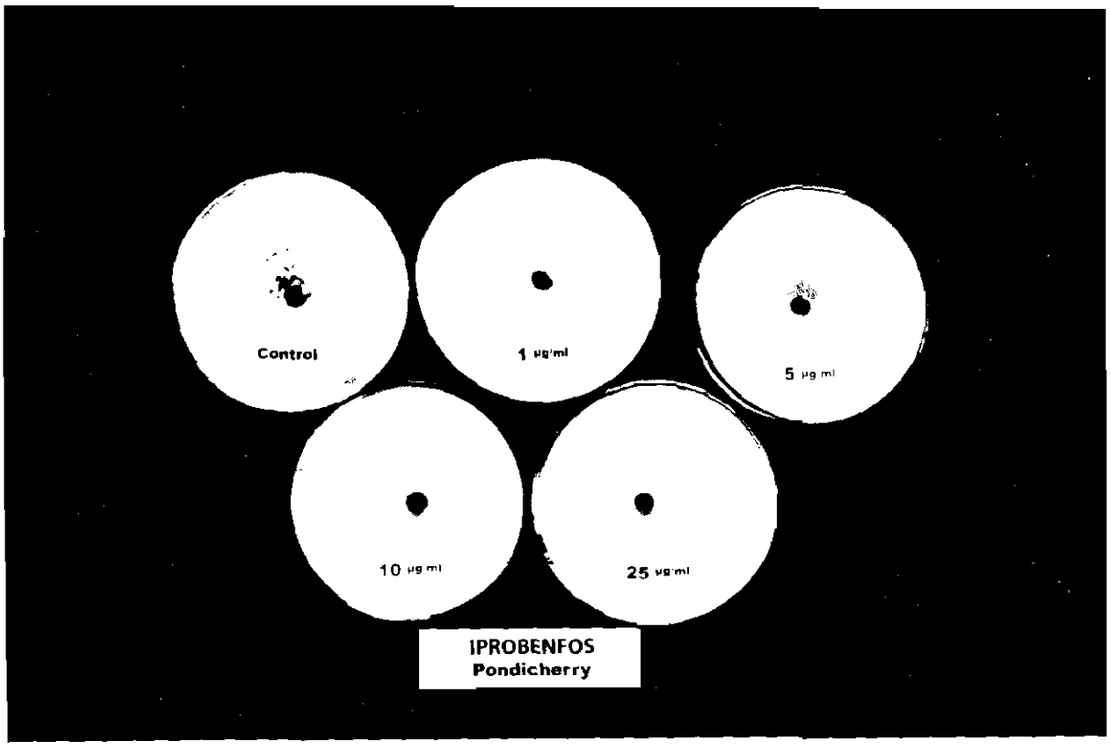


Plate 39: Inhibition of mycelial growth by Pondicherry isolate (highly sensitive) at different concentrations of iprobenfos

Table 31: *In vitro* sensitivity of three reference sensitive isolates of *Pyricularia grisea* to Iprobenfos

Name of the isolate	Poisoned food technique					Spore germination method				
	Average colony diameter (mm)			ED_{50} (µg/ml)	MIC (µg/ml)	Conidia germinated (%)			ED_{50} (µg/ml)	MIC (µg/ml)
	Control	1 ppm	5 ppm			Control	1 ppm	5 ppm		
Kadapa	90	19.33	0	0.42	5	100	29.67	0	0.59	5
Naidupeta	88	25.33	0	0.58	5	100	35.33	0	0.73	5
Tirupathi	89	23.33	0	0.53	5	100	30.33	0	0.65	5
Mean	89	22.66	0	0.51	5	100	31.78	0	0.66	5

4.6.6 Base line Sensitivity for iprobenfos

The response of three reference isolates *viz.* Kadapa, Naidupeta and Tirupathi at different concentrations of iprobenfos was determined. The ED_{50} values of these isolates ranged between 0.42-0.58 µg/ml and 0.59-0.73 µg/ml with an average of 0.51 and 0.66 µg/ml for mycelial growth and spore germination, respectively (Table 31). Where as, the MIC value for both mycelial growth and spore germination of all the three isolates was 5 µg/ml (Plate 33).

4.7 CROSS RESISTANCE OF CARBENDAZIM RESISTANT ISOLATE TO OTHER FUNGICIDES

The carbendazim resistant isolate (Cuttak) of *P. grisea* was tested for cross-tolerance to other fungicides *viz.*, edifenphos, iprobenfos, pyraclostrobin, isoprothiolane and kasugamycin. Cross-resistance experiments were conducted by taking the most sensitive isolate (Medchel) and comparing its response with the most resistant isolate. Both the isolates were grown on fungicide amended oat meal agar containing different concentrations *viz.*, 0, 1, 5, 10, 25, 50, 100, 250, 500 and 1000 µg/ml of the above mentioned fungicides. The colony diameter was recorded at different concentrations and per cent mycelial growth inhibition was calculated for each treatment in comparison with untreated control plates. Similarly, per cent inhibition of spore germination was also recorded. ED_{50} and MIC values of each fungicide for carbendazim resistant and sensitive isolates were derived from the dose response curves (Table 32; Plates 40 to 43).

4.7.1 Cross resistance of Cuttak isolate to edifenphos

The ED_{50} values for mycelial growth of carbendazim resistant and sensitive isolates were 8.41 and 3.97 µg/ml, respectively, while they were 8.33 and 4.27 µg/ml, respectively, for spore germination. The MIC value for the complete inhibition of both mycelial growth and spore germination of both the isolates was 50 µg/ml indicating that the cross resistance of carbendazim resistant isolated to edifenphos was partial when compared to the sensitive isolate (Table 32).

4.7.2 Cross resistance of Cuttak isolate to iprobenfos

The ED_{50} values for mycelial growth of carbendazim resistant and sensitive isolates were 12.40 and 3.38 µg/ml, respectively, while they were 12.47 and 3.78 µg/ml, respectively for spore germination. The MIC value for the complete inhibition of both mycelial growth and spore germination of both the isolates was 50 µg/ml indicating that the cross resistance of carbendazim resistant isolated to edifenphos was partial when compared to the sensitive isolate (Table 32).

4.7.3 Cross resistance of Cuttak isolate to pyraclostrobin

The ED_{50} values for mycelial growth of carbendazim resistant and sensitive isolates were 0.57 and 0.50 µg/ml, respectively, while they were 0.70 and 0.51 µg/ml, respectively, for spore germination. The mycelial growth and spore germination of both resistant and sensitive isolates were completely inhibited (MIC) at 5 µg/ml indicating that resistance to pyraclostrobin was unaltered when compared to its sensitive isolate (Plates 40 & 41).

Table 32: Cross resistance of Carbendazim resistant and sensitive isolates to other blasticides

S. No.	Name of the Blasticide	Resistant isolate (Cuttak)				Sensitive isolate (Medchel)			
		Mycelial growth		Spore germination		Mycelial growth		Spore germination	
		ED_{50} (µg/ml)	MIC (µg/ml)	ED_{50} (µg/ml)	MIC (µg/ml)	ED_{50} (µg/ml)	MIC (µg/ml)	ED_{50} (µg/ml)	MIC (µg/ml)
1	Carbendazim	54.63	500	56.18	500	0.39	5	0.55	5
2	Edifenphos	8.41	50	8.33	50	3.97	50	4.27	50
3	Iprobenfos	12.40	100	12.47	50	3.38	50	3.78	50
4	Pyraclostrobin	0.57	5	0.70	5	0.50	5	0.51	5
5	Isoprothiolane	2.13	25	1.96	25	1.68	10	1.67	10
6	Kasugamycin	14.38	100	26.67	250	13.11	100	23.17	250

Fig. 28: Cross resistance of Carbendazim resistant (Cuttak) and sensitive (Medchel) isolates to other blasticides (Mycelial growth)

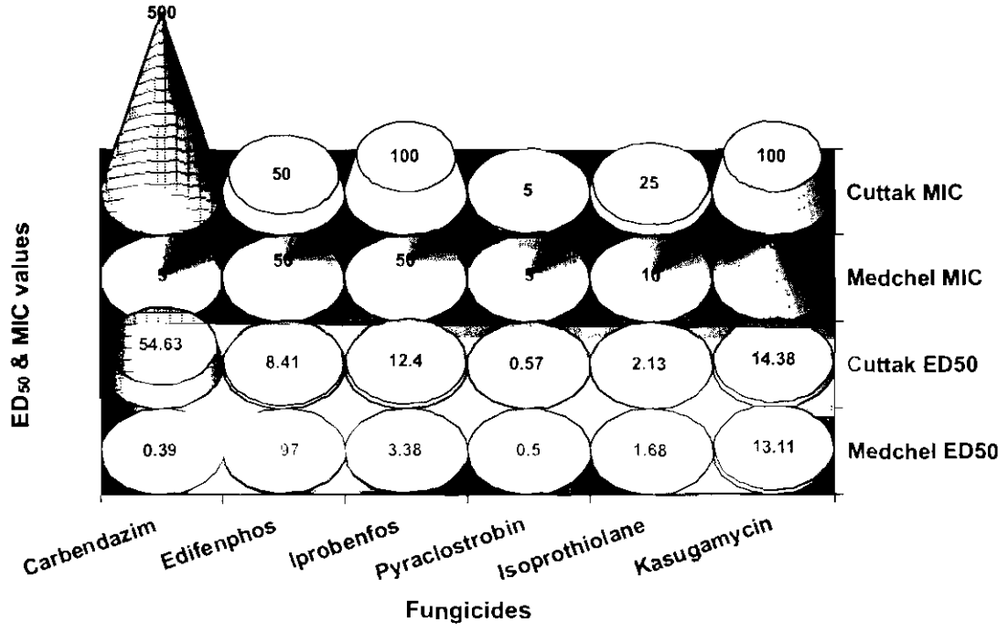
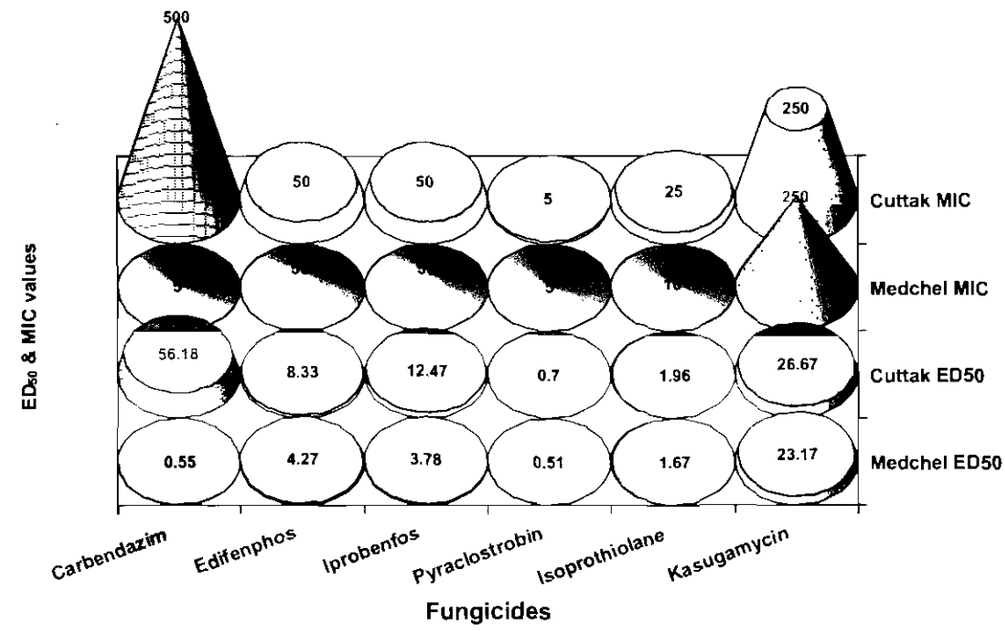


Fig. 29: Cross resistance of Carbendazim resistant (Cuttak) and sensitive (Medchel) isolates to other blasticides (*Spore germination*)



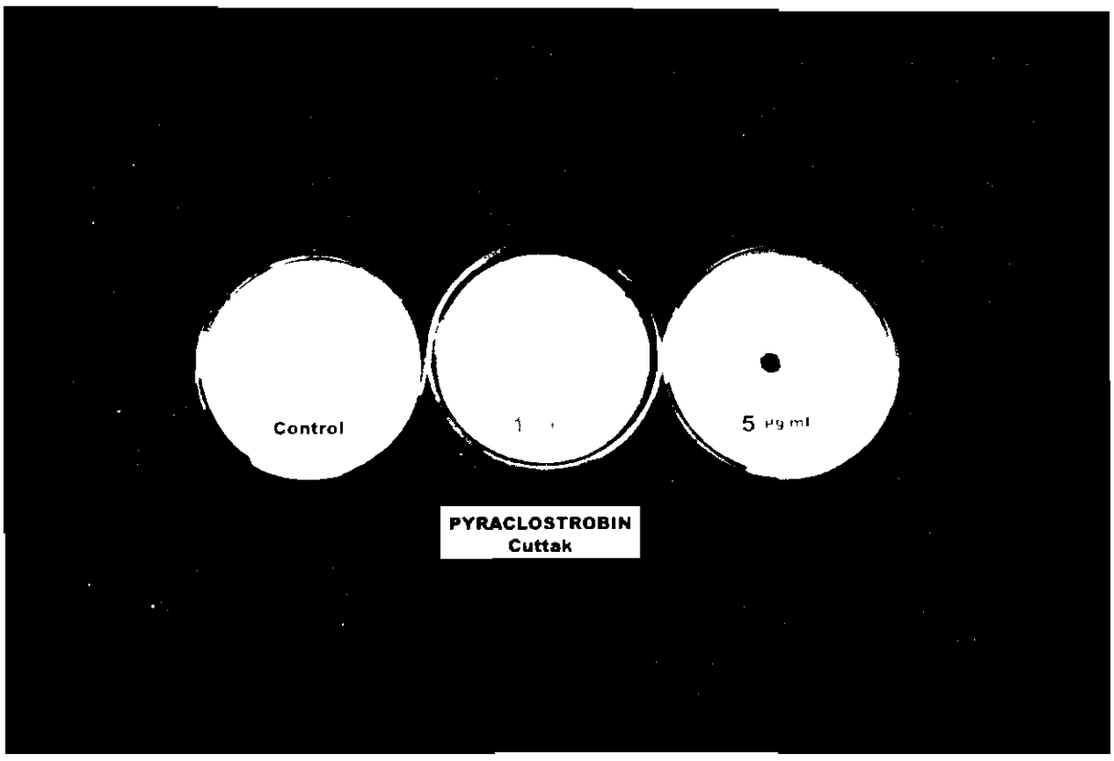


Plate 40: Inhibition of mycelial growth of carbendazim resistant isolate (Cuttak) at different concentrations of pyraclostrobin

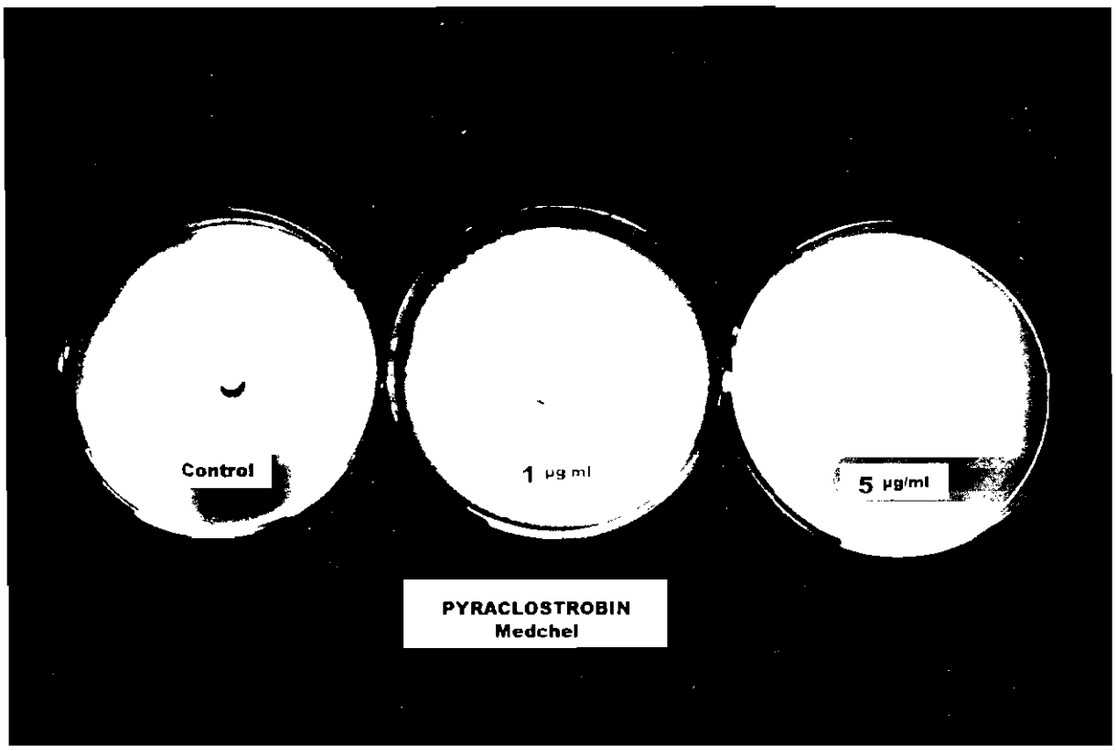


Plate 41: Inhibition of mycelial growth of carbendazim sensitive (Medchel) isolate at different concentrations of pyraclostrobin

4.7.4 *Cross resistance of Cuttak isolate to isoprothiolane*

The ED_{50} values for mycelial growth of carbendazim resistant and sensitive isolates were 2.13 and 1.68 µg/ml, respectively, while they were 1.96 and 1.67 µg/ml, respectively, for spore germination. The mycelial growth and spore germination of both resistant and sensitive isolates were completely inhibited (MIC) at 25 µg/ml indicating that resistance to isoprothiolane was unaltered when compared to its sensitive isolate (Table 32).

4.7.5 Cross resistance of Cuttak isolate to kasugamycin

The ED_{50} values for mycelial growth of the resistant and sensitive isolates were 14.38 and 13.11 µg/ml, respectively, while, they were 26.67 and 23.17 µg/ml respectively, for spore germination. The mycelial growth was completely inhibited at the concentration of 100 µg/ml, where as the spore germination was inhibited completely at 250 µg/ml in both resistant and sensitive isolates (Table 32).

4.8 CROSS RESISTANCE OF EDIFENPHOS RESISTANT ISOLATE TO OTHER FUNGICIDES

Edifenphos resistant isolate (Mandya) of *P. grisea* was tested for cross-tolerance to other fungicides *viz.*, carbendazim, iprobenfos, pyraclostrobin, isoprothiolane and kasugamycin. Cross-resistance experiments were conducted by taking the most sensitive isolate (Pondicherry) and comparing its response with the Mandya isolate. Both the isolates were grown on fungicide amended oat meal agar containing different

concentrations *viz.*, 0, 1, 5, 10, 25, 50, 100, 250, 500 and 1000 µg/ml of above mentioned fungicides. The colony diameter was recorded at different concentrations and per cent mycelial growth inhibition was calculated for each treatment in comparison with untreated control plates. Similarly, per cent inhibition of spore germination was also recorded. ED_{50} and MIC values of each fungicide for edifenphos resistant and sensitive isolates were derived from the dose response curves (Table 33).

4.8.1 Cross resistance of Mandya isolate to iprobenfos

Data presented in table 33 show that the ED_{50} values for mycelial growth of edifenphos resistant and sensitive isolates were 26.90 and 0.66 µg/ml, respectively, while they were 27.55 and 0.68 µg/ml, respectively for spore germination. The MIC value of resistant isolate was 250 µg/ml for both mycelial growth and spore germination, while it was 5 µg/ml for the sensitive isolate, indicating that the Mandya isolate has developed cross-resistance to iprobenfos.

4.8.2 Cross resistance of Mandya isolate to carbendazim

The ED_{50} values for mycelial growth of edifenphos resistant and sensitive isolates were 13.10 and 7.91 µg/ml, respectively, while they were 13.51 and 7.92 µg/ml, respectively, for spore germination. The MIC value for mycelial growth and spore germination of both the resistant and sensitive isolates was 100 µg/ml indicating that the cross resistance of edifenphos resistant isolate was partial when compared to its sensitive isolate.

Table 33: Cross resistance of Edifenphos resistant and sensitive isolates to other blasticides

S. No.	Name of the Blasticide	Resistant isolate (Mandya)				Sensitive isolate (Pondicherry)			
		Mycelial growth		Spore germination		Mycelial growth		Spore germination	
		ED_{50} (µg/ml)	MIC (µg/ml)	ED_{50} (µg/ml)	MIC (µg/ml)	ED_{50} (µg/ml)	MIC (µg/ml)	ED_{50} (µg/ml)	MIC (µg/ml)
1	Edifenphos	30.56	250	32.35	250	0.73	5	0.77	5
2	Iprobenfos	26.90	250	27.55	250	0.66	5	0.68	5
3	Carbendazim	13.10	250	13.51	100	7.91	100	7.92	100
4	Pyraclostrobin	0.66	5	0.67	5	0.56	5	0.74	5
5	Isoprothiolane	18.7	250	19.7	250	0.74	5	0.83	5
6	Kasugamycin	14.21	100	24.44	250	14.74	100	24.71	250

Fig. 30: Cross resistance of Edifenphos resistant (Mandya) and sensitive (Pondicherry) isolates to other blasticides (Mycelial growth method)

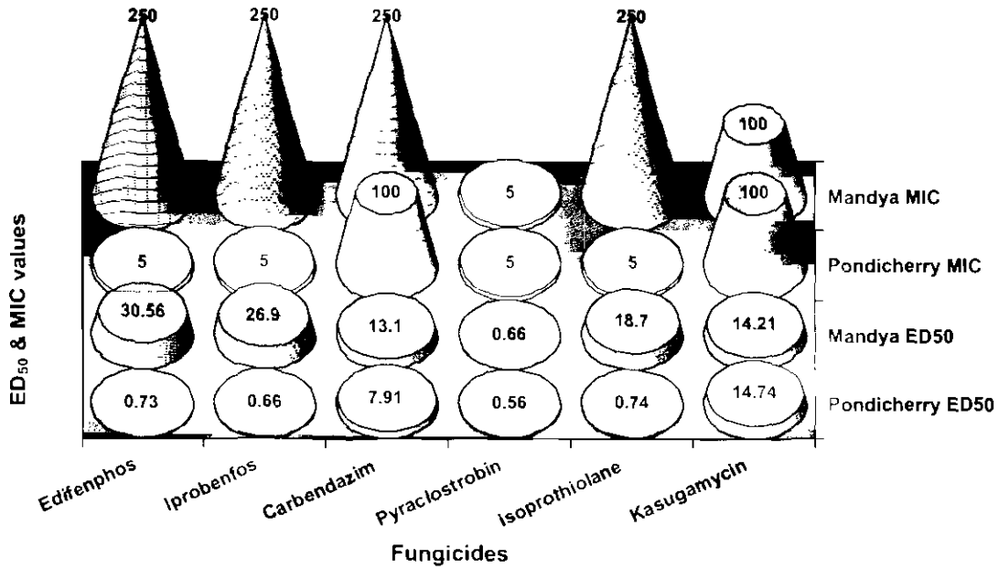
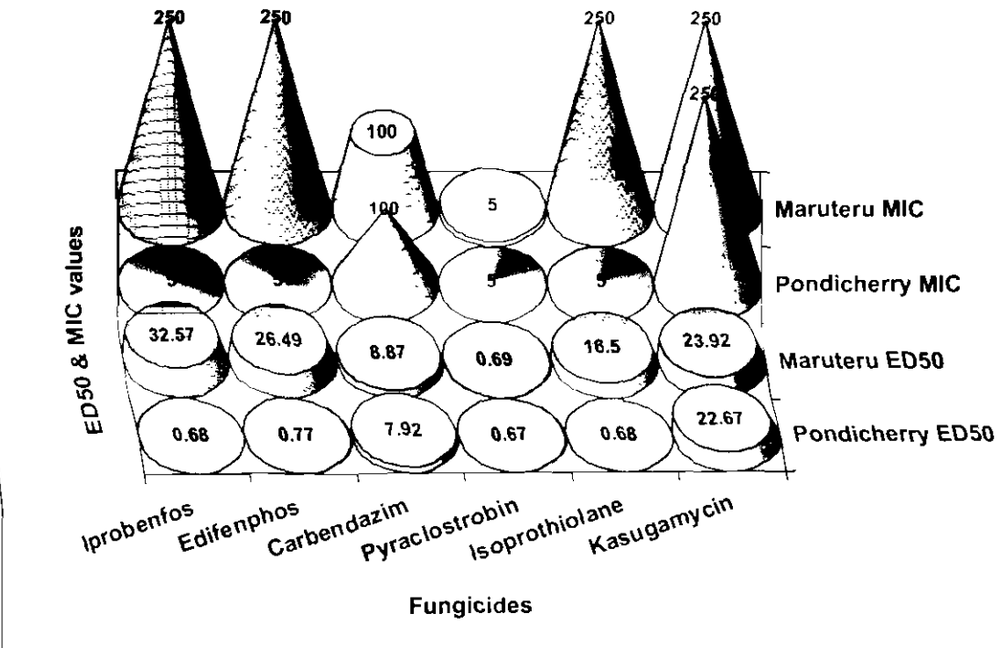


Fig. 31: Cross resistance of Iprobefos resistant (Maruteru) and sensitive (Pondicherry) isolates to other blasticides (Spore germination)



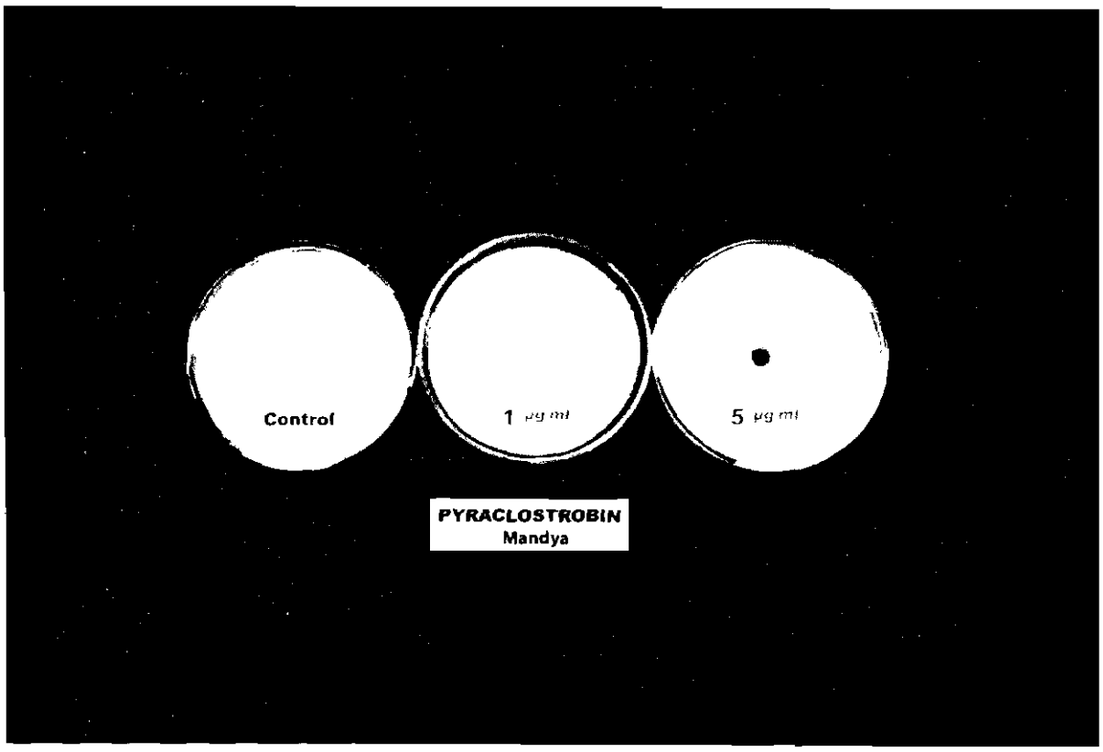


Plate 42: *Inhibition of mycelial growth of edifenphos resistant isolate (Mandya) at* different concentrations of pyraclostrobin

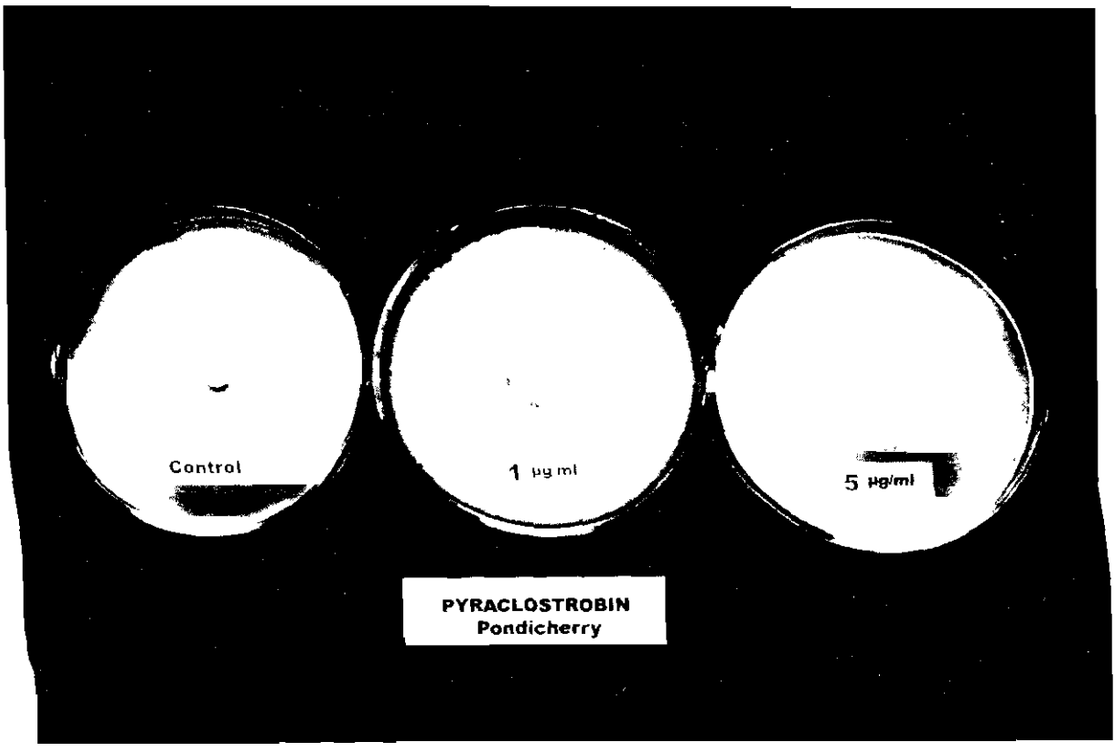


Plate 43: Inhibition of mycelial growth of edifenphos sensitive (Pondicherry) isolate *at different concentrations of pyraclostrobin*

4.8.3 Cross resistance of Mandya isolate to pyraclostrobin

Data presented in table 33 reveal that the ED_{50} values of edifenphos resistant and sensitive isolates towards pyraclostrobin were 0.66 and 0.56 µg/ml, respectively for mycelial growth. Where as, the ED_{50} values were 0.67 and 0.74 µg/ml, respectively for spore germination. The mycelial growth and spore germination of both resistant and sensitive isolates were completely inhibited (MIC) at 5 µg/ml indicating that resistance of the edifenphos resistant isolate to pyraclostrobin was unaltered when compared to its sensitive isolate (Plates 42 & 43).

4.8.4 Cross resistance of Mandya isolate to isoprothiolane

The ED_{50} values for mycelial growth of edifenphos resistant and sensitive isolates were 18.7 and 0.74 µg/ml, respectively, while they were 19.7 and 0.83 µg/ml, respectively for spore germination. The MIC value of resistant isolate was 250 µg/ml for both mycelial growth and spore germination, while it was 5 µg/ml for the sensitive isolate, indicating that the Mandya isolate has developed cross-resistance to isoprothiolane also (Table 33).

4.8.5 Cross resistance of Mandya isolate to kasugamycin

Data presented in Table 33 indicate that the ED_{50} values for mycelial growth of the resistant and sensitive isolates were 14.21 and 14.74 µg/ml, while they were 24.44 and 24.71 µg/ml for spore germination. The mycelial growth of both the isolates was completely inhibited at 100 µg/ml, but the spore germination was inhibited completely at 250 µg/ml.

4.9 CROSS RESISTANCE OF IPROBENFOS RESISTANT ISOLATE TO OTHER FUNGICIDES

The iprobenfos resistant isolate (Maruteru) of *P. grisea* was tested for cross-tolerance to other fungicides *viz.*, carbendazim, edifenphos, pyraclostrobin, isoprothiolane and kasugamycin. Cross-resistance experiments were conducted by taking the most sensitive isolate (Pondicherry) and comparing its response with the Maruteru isolate. Both the isolates were grown on fungicide amended oat meal agar containing different concentrations *viz.*, 0, 1, 5, 10, 25, 50, 100, 250, 500 and 1000 µg/ml of above mentioned fungicides. The colony diameter was recorded in different concentrations and per cent mycelial growth inhibition was calculated for each treatment in comparison with untreated control plates. Similarly, per cent inhibition of spore germination was also recorded. ED_{50} and MIC values of each fungicide for iprobenfos resistant and sensitive isolates were derived from the dose response curves (Table 34).

4.9.1 Cross resistance of Maruteru isolate to edifenphos

Data presented in table 34 show that the ED_{50} values for mycelial growth of iprobenfos resistant and sensitive isolates were 25.64 and 0.73 µg/ml, while they were 26.49 and 0.77 µg/ml, respectively for spore germination. The MIC value of resistant isolate was 250 µg/ml for both mycelial growth and spore germination, while the sensitive isolate was completely inhibited at 5 µg/ml indicating that the Maruteru isolate has developed cross-resistance to edifenphos.

Table 34: Cross resistance of Iprobenfos resistant and sensitive isolates to other blasticides

S. No.	Name of the Blasticide	Resistant isolate (Maruteru)				Sensitive isolate (Pondicherry)			
		Mycelial growth		Spore germination		Mycelial growth		Spore germination	
		ED_{50} (µg/ml)	MIC (µg/ml)	ED_{50} (µg/ml)	MIC (µg/ml)	ED_{50} (µg/ml)	MIC (µg/ml)	ED_{50} (µg/ml)	MIC (µg/ml)
1	Iprobenfos	31.85	250	32.57	250	0.66	5	0.68	5
2	Edifenphos	25.64	250	26.49	250	0.73	5	0.77	5
3	Carbendazim	7.79	100	8.87	100	7.91	100	7.92	100
4	Pyraclostrobin	0.55	5	0.69	5	0.66	5	0.67	5
5	Isoprothiolane	14.9	250	16.5	250	0.65	5	0.68	5
6	Kasugamycin	15.43	100	23.92	250	14.94	100	22.67	250

4.9.2 Cross resistance of Maruteru isolate to carbendazim

The ED_{50} values for mycelial growth of iprobenfos resistant and sensitive isolates were 7.79 and 7.91 µg/ml, respectively, while they were 8.87 and 7.92 µg/ml, respectively for spore germination. The mycelial growth and spore germination were completely inhibited (MIC) at 100 µg/ml in the both the resistant and sensitive isolates.

4.9.3 Cross resistance of Maruteru isolate to pyraclostrobin

Data presented in table 34 reveal that the ED_{50} values for mycelial growth of iprobenfos resistant and sensitive isolates were 0.55 and 0.66 µg/ml, respectively, while they were 0.69 and 0.67 µg/ml, respectively, for spore germination. The mycelial growth and spore germination of both the resistant and sensitive isolates were completely inhibited (MIC) at 5 µg/ml indicating that the sensitivity of the iprobenfos resistant isolate to pyraclostrobin was unaltered when compared to its sensitive isolate.

4.9.4 Cross resistance of Maruteru isolate to isoprothiolane

The ED_{50} values for mycelial growth of edifenphos resistant and sensitive isolates were 14.9 and 0.65 µg/ml, respectively, while they were 16.5 and 0.68 µg/ml, respectively for spore germination. The MIC value of resistant isolate was 250 µg/ml for both mycelial growth and spore germination, while it was 5 µg/ml for the sensitive isolate, indicating that the Maruteru isolate has developed cross-resistance to isoprothiolane also (Table 34).

4.9.5 Cross resistance of Maruteru isolate to kasugamycin

Data presented in Table 34 indicate that the ED_{50} values for mycelial growth of the resistant and sensitive isolates were 15.43 and 14.94 µg/ml, while they were 23.92 and 22.67 µg/ml for spore germination. The mycelial growth of both the resistant and sensitive isolates was completely inhibited at 100 µg/ml. However, the spore germination was inhibited completely at 250 µg/ml.

4.10 PERSISTENCE OF ACQUIRED RESISTANCE IN THE RESISTANT ISOLATES

The isolates of *P. grisea viz.* Cuttak, Mandya and Maruteru that developed resistance to fungicides *viz.* carbendazim, edifenphos and iprobenfos respectively, were grown in oat meal agar medium for 12 successive generations to study the persistence of acquired resistance. All the three isolates were again cultured after 12 generations in their respective fungicide amended oat meal agar containing different concentrations *viz.*, 0, 1, 5, 10, 25, 50, 100, 250, 500 and 1000 µg/ml. The colony diameter was recorded in different concentrations and per cent growth inhibition was calculated in each treatment by comparison with control plates. Similarly, per cent inhibition of spore germination was also recorded. ED_{50} and MIC values of each resistant isolate were derived from the dose response curves (Table 35).

4.10.1 Carbendazim resistant isolate (Cuttak)

The data presented in Table 35 show that the ED_{50} values of the Cuttak isolate for mycelial growth were 54.63 and 36.66 µg/ml before and after the transfer,

Table 35: Persistence of acquired resistance in the resistant isolates after 12 serial transfers on OMA medium

S. No.	Name of the isolate	Acquired Resistance against Fungicide	Mycelial growth (poisoned food)				Spore germination			
			ED_{50} (µg/ml)		MIC (µg/ml)		ED_{50} (µg/ml)		MIC (µg/ml)	
			Before	After	Before	After	Before	After	Before	After
1	Cuttak	Carbendazim	54.63	36.66	500	250	56.18	29.74	500	250
2	Mandya	Edifenphos	30.56	25.48	250	250	32.35	25.69	250	250
3	Maruteru	Iprobenfos	31.85	31.80	250	250	32.57	29.35	250	250

Fig. 32: Persistence of acquired resistance in the resistant isolates after 12 serial transfers on OMA medium (Mycelial growth)

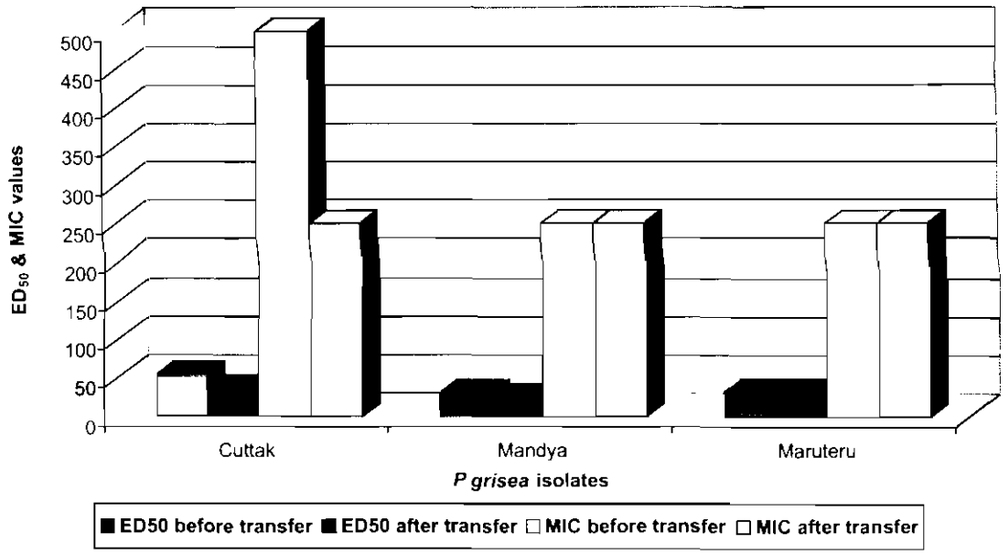
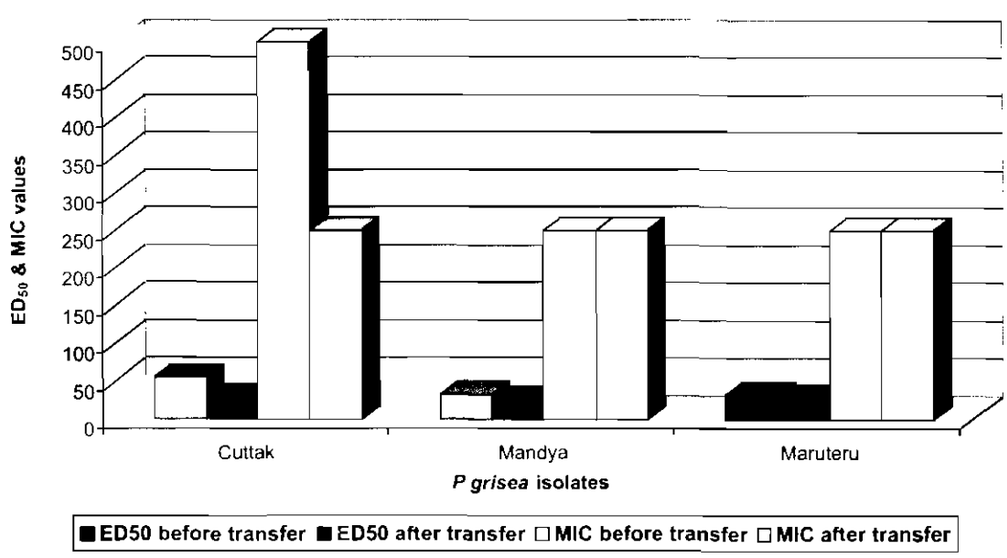


Fig. 33: Persistence of acquired resistance in the resistant isolates after 12 serial transfers on OMA medium (Spore germination)



respectively. Where as, MIC values for mycelial growth of this isolate were 500 µg/ml before the transfer and 250 µg/ml after the transfer. In case of spore germination, the ED_{50} values were 56.18 and 29.74 µg/ml before and after the transfer, respectively. Where as, MIC values of this carbendazim resistant isolate for spore germination were 500 µg/ml before the transfer and 250 µg/ml after the transfer.

4.10.2 Edifenphos resistant isolate (Mandya)

The data furnished in Table 35 show that the ED_{50} values of the Mandya isolate were 30.56 and 25.48 µg/ml before and after the transfer respectively, for mycelial growth. Where as, MIC value for mycelial growth of this isolate was 250 µg/ml both before and after the transfer. In case of spore germination, the ED_{50} values were 32.35 and 25.69 µg/ml before and after the transfer, respectively. Where as, MIC value of this edifenphos resistant isolate for spore germination also was 250 µg/ml both before and after the transfer.

4.10.3 Iprobenfos resistant isolate (Maruteru)

The data given in Table 35 reveal that the ED_{50} values of the Maruteru isolate were 31.85 and 31.80 µg/ml before and after the transfer respectively, for mycelial growth. Where as, MIC value of this isolate for mycelial growth was 250 µg/ml both before and after the transfer. In case of spore germination, the ED_{50} values were 32.57 and 29.35 µg/ml before and after the transfer, respectively. Where as, MIC value of this edifenphos resistant isolate for spore germination also was 250 µg/ml both before and after the transfer.

4.11 FUNGICIDE RESISTANCE MANAGEMENT

Experiments were conducted on resistance management in *P. grisea* both in the laboratory and glass house. Fungicides with varied modes of action *viz.* kasugamycin 2 L (amino acids and protein synthesis), propiconazole 25 EC (sterol biosynthesis in membranes), mancozeb 75 WP (multi-site contact activity), tricyclazole 75 WP (Melanin biosynthesis in cell wall) and BTH 50 WG (host plant defense induction) were used in combination with carbendazim (mitosis and cell division), edifenphos and iprobenfos (lipid and membrane synthesis) to study their efficacy against Cuttak (carbendazim resistant), Mandya (edifenphos resistant) and Maruteru (iprobenfos resistant) isolates, respectively. All the three resistant isolates were grown on fungicide amended oat meal agar containing different concentrations *viz.*, 0, 1, 5, 10, 25, 50, 100, 250, 500 and 1000 µg/ml of the above mentioned fungicides. The colony diameter was recorded at different concentrations and per cent mycelial growth inhibition was calculated for each treatment in comparison with untreated control plates. Similarly, per cent inhibition of spore germination was also recorded. ED_{50} and MIC values of each fungicide/combination were derived from the dose response curves. The efficacy of different fungicide combinations on leaf blast severity was assayed in the glass house against Cuttak (carbendazim resistant), Mandya (edifenphos resistant) and Maruteru (iprobenfos resistant) isolates. The results were presented in the Tables 36, 37 and 38.

4.11.1 Efficacy of different fungicide combinations on carbendazim resistant isolate

The results presented in Table 36 reveal spectacular differences between *in vitro* and *in vivo* performance of different fungicides and their combinations. Tricyclazole 75 WP that was found completely ineffective in inhibiting both mycelial growth and spore germination *in vitro* had shown the best performance *in vivo* in

controlling leaf blast severity. Conversely, mancozeb 75 WP that was found the most effective fungicide in inhibiting both mycelial growth and spore germination *in vitro* has shown dismal performance *in vivo* in controlling leaf blast severity. The effects of different fungicides and their combinations on mycelial growth, spore germination and leaf blast severity are presented below-

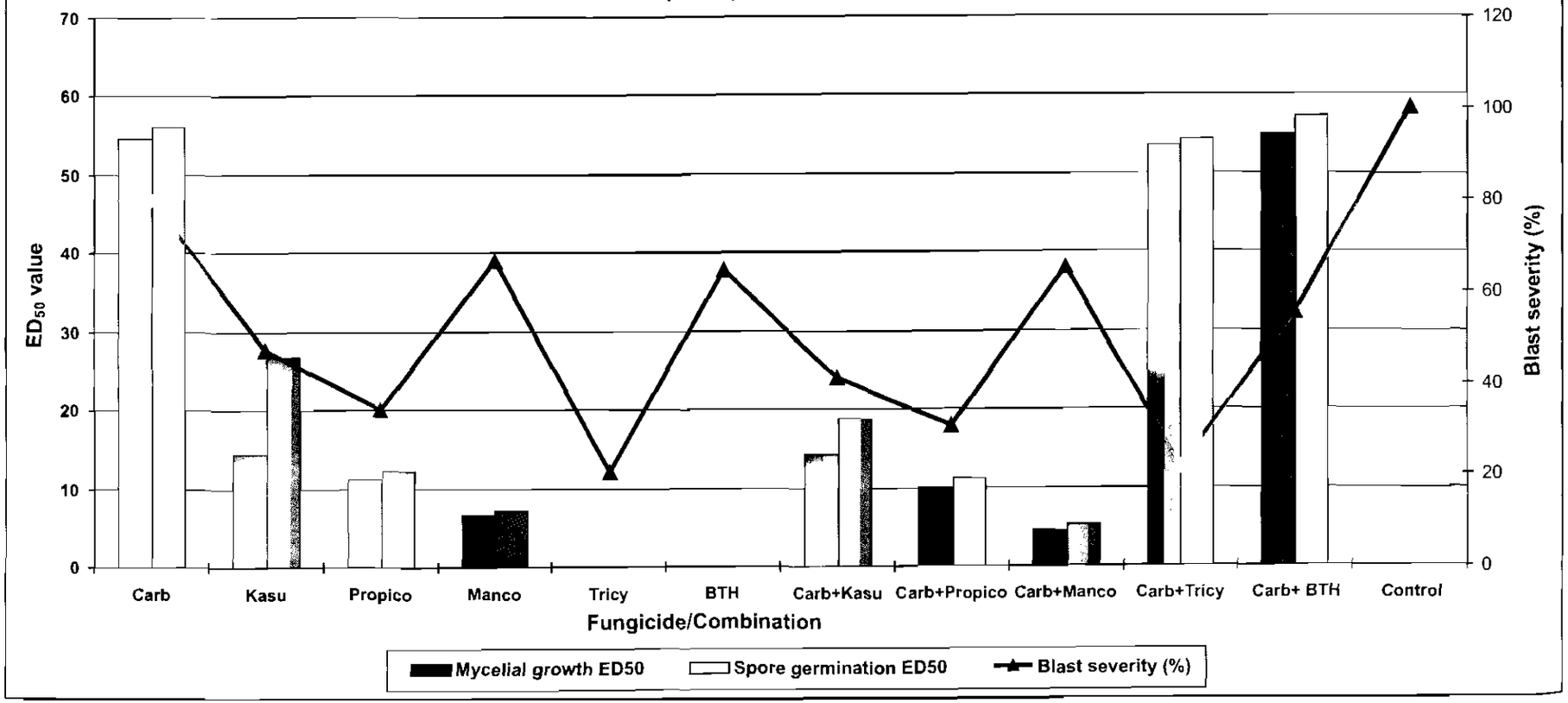
4.11.1.1 Mycelial growth

The ED_{50} values for mycelial growth ranged between 54.80 µg/ml (carbendazim 50 WP + BTH 50 WG) and 4.55 µg/ml (carbendazim 50 WP + mancozeb 75 WP) while the ED_{50} for carbendazim 50 WP alone was 54.63 µg/ml. The MIC values ranged between 500 µg/ml (carbendazim 50 WP + tricyclazole 75 WP; carbendazim 50 WP + BTH 50 WG) and 50 µg/ml (carbendazim 50 WP + mancozeb 75 WP; mancozeb 75 WP) while the MIC value for carbendazim 50 WP alone was 500 µg/ml. The ED_{50} values were reduced significantly from 54.63 µg/ml (carbendazim 50 WP alone) to 14.19 µg/ml, 10.17 µg/ml and 4.55 µg/ml due to the combination of carbendazim 50 WP with kasugamycin 2L, propiconazole 25 EC and mancozeb 75 WP, respectively. However, the combination of carbendazim 50 WP with tricyclazole 75 WP (53.54 µg/ml) and BTH 50 WG (54.80 µg/ml) had not reduced the ED_{50} values significantly when compared to carbendazim alone (54.63 µg/ml). The MIC values were reduced significantly from 500 µg/ml (carbendazim 50 WP alone) to 100 µg/ml, 100 µg/ml and 50 µg/ml due the combination of carbendazim 50 WP with kasugamycin 2L, propiconazole 25 EC and mancozeb 75 WP, respectively. However, there was no change in MIC values due to the combination of carbendazim 50 WP with either tricyclazole 75 WP or BTH 50 WG (500 µg/ml) compared to carbendazim 50 WP alone (500 µg/ml).

Table 36: Efficacy of different fungicide combinations on Carbendazim resistant isolate both *in vitro* and *in vivo*

S. No.	Name of the fungicide/combination	Mycelial growth		Spore germination		Leaf blast severity (%)
		ED_{50} (µg/ml)	MIC (µg/ml)	ED_{50} (µg/ml)	MIC (µg/ml)	
1	Carbendazim 50 WP	54.63	500	56.18	500	80.33 (63.69)
2	Kasugamycin 2 L	14.42	100	26.69	250	47.33 (43.47)
3	Propiconazole 25 EC	11.27	100	12.23	100	34.33 (35.67)
4	Mancozeb 75 WP	6.72	50	7.15	50	66.67 (54.74)
5	Tricyclazole 75 WP	-	-	-	-	20.67 (27.01)
6	BTH 50 WG	-	-	-	-	64.67 (53.54)
7	Carbendazim 50 WP + Kasugamycin 2 L	14.19	100	18.75	100	41.00 (39.81)
8	Carbendazim 50 WP + Propiconazole 25 EC	10.17	100	11.12	100	30.67 (33.62)
9	Carbendazim 50 WP + Mancozeb 75 WP	4.55	50	5.17	50	65.33 (53.93)
10	Carbendazim 50 WP + Tricyclazole 75 WP	53.54	500	54.32	500	21.67 (27.73)
11	Carbendazim 50 WP + BTH 50 WG	54.80	500	57.15	500	55.33 (48.06)
12	Untreated Control	-	-	-	-	100.00 (90.00)
SEM ±						0.68
CD (P=0.05)						2.01
CD (P=0.01)						2.61

Fig. 34: Efficacy of different fungicide combinations on carbendazim resistant isolate both in vitro and in vivo (ED_{50})



4.11.1.2 Spore germination

The ED_{50} values for spore germination ranged between 57.15 µg/ml (carbendazim 50 WP + BTH 50 WG) and 5.17 µg/ml (carbendazim 50 WP + mancozeb 75 WP) while the ED_{50} for carbendazim 50 WP alone was 56.18 µg/ml. The MIC values ranged between 500 µg/ml (carbendazim 50 WP + tricyclazole 75 WP; carbendazim 50 WP + BTH 50 WG) and 50 µg/ml (carbendazim 50 WP + mancozeb 75 WP; mancozeb 75 WP) while the MIC value for carbendazim 50 WP alone was 500 µg/ml. The ED_{50} values were reduced significantly from 56.18 µg/ml (carbendazim 50 WP alone) to 18.75 µg/ml, 11.12 µg/ml and 5.17 µg/ml due to the combination of carbendazim 50 WP with kasugamycin 2L, propiconazole 25 EC and mancozeb 75 WP, respectively. However, the combination of carbendazim 50 WP with tricyclazole 75 WP (54.32 µg/ml) and BTH 50 WG (57.15 µg/ml) had not reduced the ED_{50} values significantly compared to carbendazim alone (56.18 µg/ml). The MIC values were reduced significantly from 500 µg/ml (carbendazim 50 WP alone) to 100 µg/ml, 100 µg/ml and 50 µg/ml due the combination of carbendazim 50 WP with kasugamycin 2L, propiconazole 25 EC and mancozeb 75 WP, respectively. However, there was no change in MIC values due to the combination of carbendazim 50 WP with either tricyclazole 75 WP or BTH 50 WG (500 µg/ml) compared to carbendazim 50 WP alone (500 µg/ml).

4.11.1.3 Leaf blast severity

All the chemicals and their combinations were found significantly superior to the untreated control in reducing the leaf blast severity, where the per cent leaf blast severity ranged *between 20.67 and 100 in tricyclazole 75 WP alone and untreated* control, respectively. The combination of carbendazim 50 WP with any other chemical significantly reduced the leaf blast severity when compared to carbendazim alone.

Tricyclazole 75 WP recorded the lowest leaf blast severity of 20.67 per cent. However, its combination with carbendazim 50 WP (21.67%) had not shown any significant difference. There was no significant difference between mancozeb 75 WP alone (66.67%) and carbendazim 50 WP + mancozeb 75 WP (65.33%). Conversely, there was significant reduction in leaf blast severity between kasugamycin 2L (47.33%) and carbendazim 50 WP + kasugamycin 2L (41%); propiconazole 25 EC (34.33%) and carbendazim 50 WP + propiconazole 25 EC (30.67%); BTH 50 WG (64.67%) and carbendazim 50 WP + BTH 50 WG (55.33%), respectively.

4.11.2 Efficacy of different fungicide combinations on edifenphos resistant isolate

The results presented in the table 37 reveal spectacular differences between *in vitro* and *in vivo* performance of different fungicides and their combinations. Tricyclazole 75 WP that was found completely ineffective in inhibiting both mycelial growth and spore germination *in vitro* had shown the best performance *in vivo* in controlling leaf blast severity. Conversely, mancozeb 75 WP that was found the most effective fungicide in inhibiting both mycelial growth and spore germination *in vitro* has shown the least performance *in vivo* in controlling leaf blast severity. The effects of different fungicides and their combinations on mycelial growth, spore germination and leaf blast severity are presented below-

4.11.2.1 Mycelial growth

The ED_{50} values for mycelial growth ranged between 32.85 µg/ml (edifenphos 50 EC + BTH 50 WG) and 5.05 µg/ml (edifenphos 50 EC + mancozeb 75 WP) while the ED_{50} for edifenphos 50 EC alone was 30.56 µg/ml. The MIC values

ranged between 250 µg/ml (edifenphos 50 EC + tricyclazole 75 WP; edifenphos 50 EC + BTH 50 WG) and 50 µg/ml (edifenphos 50 EC + mancozeb 75 WP; mancozeb 75 WP) while the MIC value for edifenphos 50 EC alone was 250 µg/ml. The ED_{50} values were reduced significantly from 30.56 µg/ml (edifenphos 50 EC) to 13.79 µg/ml, 11.39 µg/ml and 5.05 µg/ml due to the combination of edifenphos 50 EC with kasugamycin 2L, propiconazole 25 EC and mancozeb 75 WP, respectively. However, the combination of edifenphos 50 EC with tricyclazole 75 WP (30.94 µg/ml) and BTH 50 WG (32.85 µg/ml) had not reduced the ED_{50} values significantly compared to edifenphos alone (30.56 µg/ml). The MIC values were reduced significantly from 250 µg/ml (edifenphos 50 EC) to 100 µg/ml, 100 µg/ml and 50 µg/ml due the combination of edifenphos 50 EC with kasugamycin 2L, propiconazole 25 EC and mancozeb 75 WP, respectively. However, there was no change in MIC values due to the combination of edifenphos 50 EC with either tricyclazole 75 WP or BTH 50 WG (250 µg/ml) compared to edifenphos 50 EC alone (250 µg/ml).

4.11.2.2 Spore germination

The ED_{50} values for spore germination ranged between 32.62 µg/ml (edifenphos 50 EC + BTH 50 WG) and 6.17 µg/ml (edifenphos 50 EC + mancozeb 75 WP) while the ED_{50} for edifenphos alone was 32.35 µg/ml. The MIC values ranged between 250 µg/ml (edifenphos 50 EC + kasugamycin 2L; edifenphos 50 EC + tricyclazole 75 WP; edifenphos 50 EC + BTH 50 WG; kasugamycin 2L) and 50 µg/ml (edifenphos 50 EC + mancozeb 75 WP; mancozeb 75 WP) while the MIC value for edifenphos 50 EC alone was 250 µg/ml. The ED_{50} values were reduced significantly from 32.35 µg/ml (edifenphos 50 EC) to 24.18 µg/ml, 11.88 µg/ml and 6.17 µg/ml due to the

Table 37: Efficacy of different fungicide combinations on edifenphos resistant isolate both *in vitro* and *in vivo*

S. No.	Name of the fungicide/combination	Mycelial growth		Spore germination		Leaf blast severity (%)
		ED_{50} (µg/ml)	MIC (µg/ml)	ED_{50} (µg/ml)	MIC (µg/ml)	
1	Edifenphos 50 EC	30.56	250	32.35	250	66.33 (54.54)
2	Kasugamycin 2 L	14.22	100	24.56	250	50.00 (45.00)
3	Propiconazole 25 EC	12.40	100	13.79	100	35.67 (36.67)
4	Mancozeb 75 WP	6.64	50	7.78	50	69.67 (56.58)
5	Tricyclazole 75 WP	-	-	-	-	22.33 (28.18)
6	BTH 50 WG	-	-	-	-	64.00 (53.14)
7	Edifenphos 50 EC + Kasugamycin 2 L	13.79	100	24.18	250	40.00 (39.23)
8	Edifenphos 50 EC + Propiconazole 25 EC	11.39	100	11.88	100	30.67 (33.62)
9	Edifenphos 50 EC + Mancozeb 75 WP	5.05	50	6.17	50	66.67 (54.74)
10	Edifenphos 50 EC + Tricyclazole 75 WP	30.94	250	31.93	250	21.67 (27.74)
11	Edifenphos 50 EC + BTH 50 WG	32.85	250	32.62	250	51.00 (45.57)
12	Untreated Control	-	-	-	-	100.00 (90.00)
SEM ±						0.67
CD (P=0.05)						1.98
CD (P=0.01)						2.57

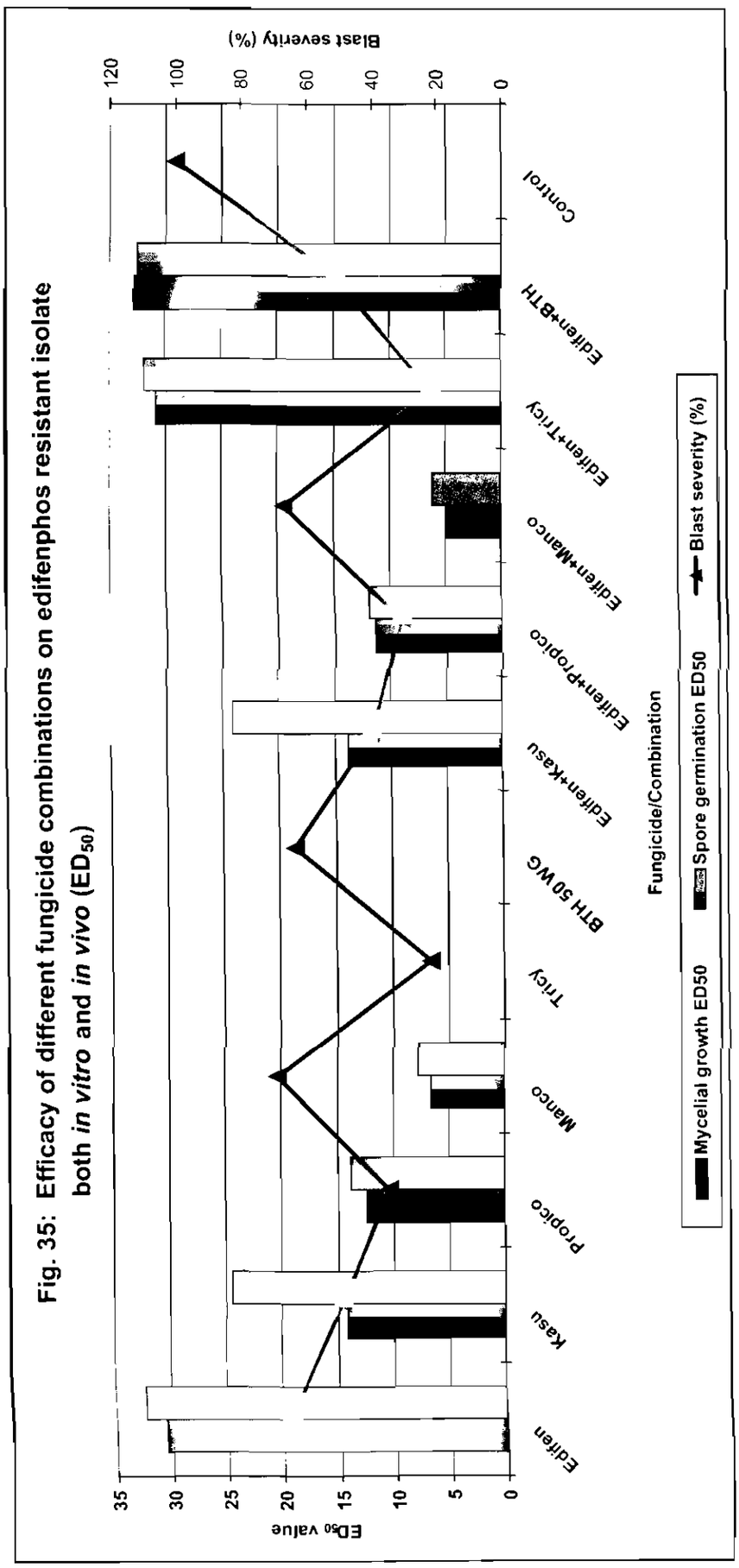


Fig. 35: Efficacy of different fungicide combinations on edifenphos resistant isolate both *in vitro* and *in vivo* (ED_{50})

combination of edifenphos 50 EC with kasugamycin 2L, propiconazole 25 EC and mancozeb 75 WP, respectively. However, the combination of edifenphos 50 EC with tricyclazole 75 WP (31.93 µg/ml) and BTH 50 WG (32.62 µg/ml) had not reduced the ED_{50} values significantly compared to edifenphos alone (32.35 µg/ml). The MIC values were reduced significantly from 250 µg/ml (edifenphos 50 EC alone) to 100 µg/ml and 50 µg/ml due the combination of edifenphos 50 EC with propiconazole 25 EC and mancozeb 75 WP, respectively. However, there was no change in MIC values due to the combination of edifenphos 50 EC with kasugamycin 2L, tricyclazole 75 WP or BTH 50 WG (250 µg/ml) compared to edifenphos 50 EC alone (250 µg/ml).

4.11.2.3. Leaf blast severity

All the chemicals and their combinations were found significantly superior to the untreated control in reducing the leaf blast severity where the per cent leaf blast severity ranged between 21.67 and 100 in edifenphos 50 EC + tricyclazole 75 WP and untreated control, respectively. The combination of edifenphos 50 EC with any other chemical significantly reduced the leaf blast severity except with mancozeb 75 WP. Edifenphos 50 EC in combination with tricyclazole 75 WP recorded the lowest leaf blast severity of 21.67 per cent. However, there was no significant difference between tricyclazole 75 WP alone and its combination with edifenphos 50 EC. But, there was significant reduction in leaf blast severity between kasugamycin 2L (50%) and edifenphos 50 EC + kasugamycin 2L (40%); propiconazole 25 EC (35.67%) and edifenphos 50 EC + propiconazole 25 EC (30.67%); BTH 50 WG (64%) and edifenphos 50 EC + BTH 50 WG (51%), respectively. Conversely, there was no significant difference between mancozeb 75 WP alone (69.67%) and edifenphos 50 EC + mancozeb 75 WP (66.67%).

4.11.3 Efficacy of different fungicide combinations on iprobenfos resistant isolate

The results presented in Table 38 reveal spectacular differences between *in vitro* and *in vivo* performance of different fungicides and their combinations. Tricyclazole 75 WP that was found completely ineffective in inhibiting both mycelial growth and spore germination *in vitro* had shown the best performance *in vivo* in controlling leaf blast severity. Conversely, mancozeb 75 WP that was found the most effective fungicide in inhibiting both mycelial growth and spore germination *in vitro* has shown the least performance *in vivo* in controlling leaf blast severity. The effects of different fungicides and their combinations on mycelial growth, spore germination and leaf blast severity are presented below-

4.11.3.1 Mycelial growth

The ED_{50} values for mycelial growth ranged between 32.36 µg/ml (iprobenfos 48 EC + BTH 50 WG) and 4.62 µg/ml (iprobenfos 48 EC + mancozeb 75 WP) while the ED_{50} for iprobenfos 48 EC alone was 31.85 µg/ml. The MIC values ranged between 250 µg/ml (iprobenfos 48 EC + tricyclazole 75 WP; iprobenfos 48 EC + BTH 50 WG) and 50 µg/ml (iprobenfos 48 EC + mancozeb 75 WP; mancozeb 75 WP) while the MIC value for iprobenfos 48 EC alone was 250 µg/ml. The ED_{50} values were reduced significantly from 31.85 µg/ml (iprobenfos 48 EC) to 14.65 µg/ml, 11.60 µg/ml and 4.62 µg/ml due to the combination of iprobenfos 48 EC with kasugamycin 2L, propiconazole 25 EC and mancozeb 75 WP, respectively. However, the combination of iprobenfos 48 EC with tricyclazole 75 WP (31.56 µg/ml) and BTH 50 WG (32.36 µg/ml) had not reduced the ED_{50} values significantly compared to iprobenfos alone (31.85 µg/ml). The MIC values were reduced significantly from 250 µg/ml (iprobenfos 48 EC)

Table 38: Efficacy of different fungicide combinations on iprobenfos resistant isolate both *in vitro* and *in vivo*

S. No.	Name of the fungicide/combination	Mycelial growth		Spore germination		Leaf blast severity (%)
		ED_{50} (µg/ml)	MIC (µg/ml)	ED_{50} (µg/ml)	MIC (µg/ml)	
1	Iprobenfos 48 EC	31.85	250	32.57	250	62.67 (52.34)
2	Kasugamycin 2 L	15.07	100	23.61	250	49.33 (44.62)
3	Propiconazole 25 EC	12.74	100	13.95	100	33.67 (35.47)
4	Mancozeb 75 WP	6.37	50	7.81	50	70.00 (56.79)
5	Tricyclazole 75 WP	-	-	-	-	20.33 (26.80)
6	BTH 50 WG	-	-	-	-	61.00 (51.36)
7	Iprobenfos 48 EC + Kasugamycin 2 L	14.65	100	23.36	250	36.33 (37.06)
8	Iprobenfos 48 EC + Propiconazole 25 EC	11.60	100	11.69	100	31.00 (33.83)
9	Iprobenfos 48 EC + Mancozeb 75 WP	4.62	50	5.24	50	61.67 (51.75)
10	Iprobenfos 48 EC + Tricyclazole 75 WP	31.56	250	32.34	250	21.00 (27.27)
11	Iprobenfos 48 EC + BTH 50 WG	32.36	250	33.50	250	45.76 (42.53)
12	Untreated Control	-	-	-	-	100.00 (90.00)
SEM ±						0.55
CD (P=0.05)						1.61
CD (P=0.01)						2.10

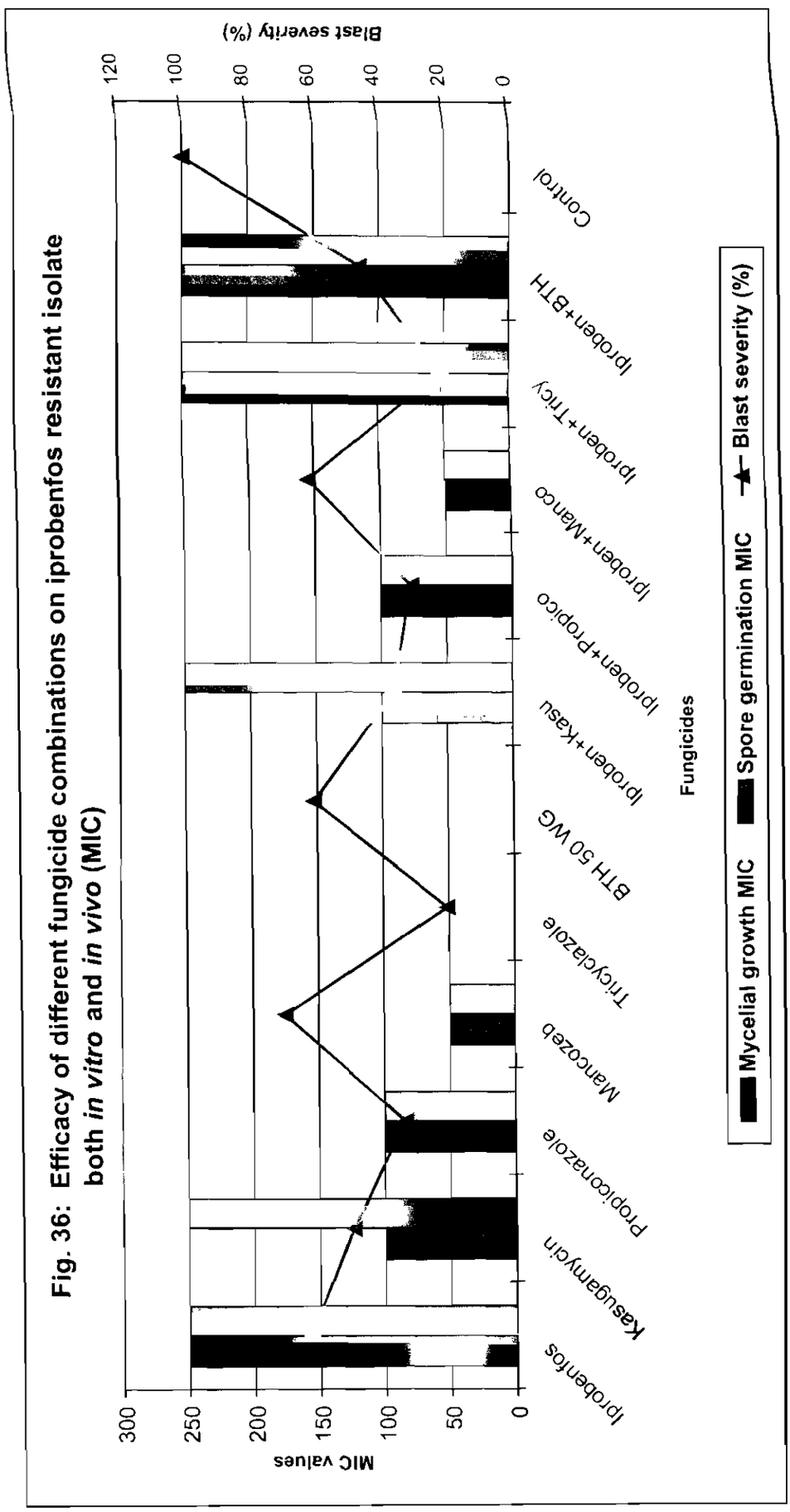


Fig. 36: Efficacy of different fungicide combinations on iprobenfos resistant isolate both *in vitro* and *in vivo* (MIC)

to 100 µg/ml, 100 µg/ml and 50 µg/ml due the combination of iprobenfos 48 EC with kasugamycin 2L, propiconazole 25 EC and mancozeb 75 WP, respectively. However, there was no change in MIC values due to the combination of iprobenfos 48 EC with either tricyclazole 75 WP or BTH 50 WG (250 µg/ml) compared to iprobenfos 48 EC alone (250 µg/ml).

4.11.3.2 Spore germination

The ED_{50} values for spore germination ranged between 33.50 µg/ml (iprobenfos EC + BTH 50 WG) and 5.24 µg/ml (iprobenfos 48 EC + mancozeb 75 WP) while the ED_{50} for iprobenfos 48 alone was 32.57 µg/ml. The MIC values ranged between 250 µg/ml (iprobenfos 48 EC + kasugamycin 2L; iprobenfos 48 EC + tricyclazole 75 WP; iprobenfos 48 EC + BTH 50 WG; kasugamycin 2L) and 50 µg/ml (iprobenfos 48 EC + mancozeb 75 WP; mancozeb 75 WP) while the MIC value for iprobenfos 48 EC alone was 250 µg/ml. The ED_{50} values were reduced significantly from 32.57 µg/ml (iprobenfos 48 EC) to 23.36 µg/ml, 11.69 µg/ml and 5.24 µg/ml due to the combination of iprobenfos 48 EC with kasugamycin 2L, propiconazole 25 EC and mancozeb 75 WP, respectively. However, the combination of iprobenfos 48 EC with tricyclazole 75 WP (32.34 µg/ml) and BTH 50 WG (33.50 µg/ml) had not reduced the ED_{50} values significantly compared to iprobenfos 48 EC alone (32.57 µg/ml). The MIC values were reduced significantly from 250 µg/ml (iprobenfos 48 EC alone) to 100 µg/ml and 50 µg/ml due the combination of iprobenfos 48 EC with propiconazole 25 EC and mancozeb 75 WP, respectively. However, there was no change in MIC values due to the combination of iprobenfos 48 EC with kasugamycin 2L, tricyclazole 75 WP or BTH 50 WG (250 µg/ml) compared to iprobenfos 48 EC alone (250 µg/ml).

4.11.3.3 Leaf blast severity

All the chemicals and their combinations were found significantly superior to the untreated control in reducing the leaf blast severity where the per cent leaf blast severity ranged between 20.33 and 100 in tricyclazole 75 WP alone and untreated control, respectively. The combination of iprobenfos 48 EC with any other chemical significantly reduced the leaf blast severity except with mancozeb 75 WP. Tricyclazole 75 WP alone recorded the lowest leaf blast severity of 20.33 per cent. However, there was no significant difference between tricyclazole 75 WP alone and its combination with iprobenfos 48 EC (21%). But, there was significant reduction in leaf blast severity between kasugamycin 2L (49.33%) and iprobenfos 48 EC + kasugamycin 2L (36.33%); BTH 50 WG (61%) and iprobenfos 48 EC + BTH 50 WG (45.76%), respectively. Conversely, there was no significant difference between propiconazole 25 EC (33.67%) and iprobenfos 48 EC + propiconazole 25 EC (31%). However, the combination of iprobenfos 48 EC with mancozeb 75 WP (61.67%) was found significantly superior to mancozeb 75 WP alone (70%).

Discussion

A3

Chapter V

DISCUSSION

The results of present investigation *viz.* resistance exhibited by different rice cultivars against blast disease; the *in vitro* efficacy of different SAR inducing chemicals as well as fungicides against *Pyricularia grisea*; efficacy of SAR chemicals in inducing systemic resistance in rice plants against blast disease; performance of various fungicides against rice blast both in the nursery and main field; fungicide resistance and cross resistance present in various isolates collected different rice growing regions of India; persistence of acquired resistance in resistant strains and finally the fungicide resistance management strategies are discussed here under -

5.1 EVALUATION OF RICE CULTIVARS FOR RESISTANCE AGAINST BLAST DISEASE

Different rice cultivars were evaluated for blast disease resistance against Rajendranagar isolate of *Pyricularia grisea* during the years 2002 and 2003 in Uniform Blast Nursery by using 0-9 scale of Standard Evaluation System developed by the International Rice Research Institute in order to use few of them for SAR studies. The cultivars were categorized into 5 groups *viz.* highly resistant (0-1 score), resistant (2-3 score), moderately resistant/susceptible (4-5 score), susceptible (6-7 score) and highly susceptible (8-9 score) based on their reaction to the blast disease. A total of 145 rice cultivars were evaluated, out of which 46 were highly resistant, 32 were resistant, 15 were moderately resistant/susceptible, 19 were susceptible and 33 were highly susceptible.

Saifulla and Manjunatha (1995), Sharma and Ghimire (1996), Singh and Dodan (1996), Rao and Reddy (2004), Sengultuvel *et al.* (2006) and Tripathi (2006) have also done similar kind of evaluation. Majority of the cultivars used by them were different from those used in the present investigation. Saifulla and Manjunatha (1995) have reported HR-12 as highly susceptible while Tripathi (2006) reported the varieties Govind and Jaya as resistant. The present investigation also agrees with the report of the above authors. Singh and Dodan (1996) screened 175 rice genotypes during 1993-94 for resistance against rice blast in Haryana. Among them, 19 genotypes were resistant to leaf blast and 10 were resistant to neck blast.

Rao and Reddy (2004) evaluated ten new plant types (NPTs) of rice for their disease resistance along with IR-64 and Rasi as resistant controls and HR-12 as susceptible control in the Uniform Blast Nursery (UBN) using 0-9 scale of Standard Evaluation System (SES, 1996) developed by IRRI. Among the NPTs, 5 were resistant, 3 were moderately resistant and 2 were reported as susceptible. Tripathi (2006) screened 12 rice cultivars *viz.* Indrajal, Vandana, Gurmatia, Basmati, Govinda, Jaya, Kalinga, Dehula, IR-64, Ajan, Lochai and Newari. Higher degree of resistance was observed in the cultivars *viz.* Kalinga, Vandana, IR-64 and Govinda while Gurmatia, Indrajal and Dehula were found susceptible.

5.2 INDUCTION OF SYSTEMIC ACQUIRED DISEASE RESISTANCE IN RICE AGAINST P. GRISEA

Three varieties *viz.* Rasi, Swarnadhan and HR-12 were selected randomly under highly resistant (0 scale), moderately resistant or susceptible (4 scale) and highly susceptible (9 scale) categories, respectively to induce systemic acquired resistance using chemicals.

5.2.1 *In vitro* efficacy of SAR inducing chemicals against *P. grisea*

Salicylic acid, BTH, sodium selenite, ferric chloride and zinc sulphate were tested at different concentrations *viz.* 1 M, 10^{-1} M, 10^{-2} M, 10^{-3} M, 10^{-4} M and 10^{-5} M by employing 'Inhibition Zone Technique'. All the five chemicals formed inhibition zones at 1 M, 10^{-1} M and 10^{-2} M while sodium selenite and ferric chloride formed inhibition zones at 10^{-3} M also. None of the chemicals formed inhibition zones at 10^{-5} M while sodium selenite alone formed inhibition zone at 10^{-4} M (Fig. 4).

Any chemical that induces systemic resistance in the crop plants should have no fungitoxicity at its effective concentration. When Bhattacharyya and Roy (1998) tested sodium selenite at 10^{-5} M, zinc sulphate at 10^{-4} M, lithium sulphate at 10^{-4} M, calcium nitrate at 10^{-2} M, sodium fluoride at 10^{-3} M and ferric chloride at 10^{-4} M by poisoned food technique, the results showed no *in vitro* fungitoxicity against *Rhizoctonia solani*. In the present study, sodium selenite, ferric chloride and zinc sulphate did not show any inhibitory effect on mycelial growth *in vitro* at 10^{-5} M, 10^{-4} M and 10^{-3} M, respectively. Senapaty *et al.* (2000) tested 21 non-conventional chemicals against *P. grisea in vitro* by poisoned food technique and concluded that cadmium chloride (10^{-4} M), ferric chloride (10^{-4} M), lithium sulphate (10^{-3} M), magnesium sulphate (10^{-3} M), potassium chloride (10^{-3} M), sodium fluoride (10^{-3} M), sodium molybdate (10^{-4} M), sodium silicate (10^{-4} M) and zinc sulphate (10^{-4} M) showed no *in vitro* fungitoxicity at their effective concentrations.

In the present study, zinc sulphate and ferric chloride have shown no *in vitro* fungitoxicity against *P. grisea* at 10^{-4} M and 10^{-3} M concentrations, respectively.

Senapaty *et al.* reported zinc sulphate non-toxic at 10^{-4} M concentration, but the present study reveal that zinc sulphate did not show any inhibitory effect on mycelial growth of *P. grisea* at 10^{-3} M concentration itself. Similarly, Bhattacharyya and Roy (1998) reported zinc sulphate as non-toxic at 10^{-4} M concentration on *Rhizoctonia solani.*

Dann *et al.* (1998) reported that 2,6-dichloro isonicotonic acid (INA) and benzothiodiazole (BTH) did not result in observable pathotoxicity *in vitro* or inhibit the growth of *Sclerotinia sclerotiorum.* SA did not inhibit mycelial growth of *Pythium aphanidermatum* at 100-200 µg/ml *in vitro*, but higher levels inhibited mycelial growth. Ishii *et al.* (1999) reported that there is no remarkable activity of BTH on mycelial growth and conidial germination of many fungi like *Cladosporium cucumerinum, Colletotrichum lagenarium, Fusarium oxysporum* f.sp. *cucumerinum, Corynespora cassicola, Alternaria alternata* and *Botrytis cinerea.* In the present study, both salicylic acid and BTH did not show any inhibitory effect on mycelial growth of *P. grisea* at 10^{-3} M concentration which was in agreement with the work of above authors.

5.2.2 Effect of SAR inducing chemicals on seed germination

The per cent seed germination was significantly lower when treated with sodium selenite (10^{-5} M) i.e. 80.46, 84.38 and 80.83 in HR-12, Swarnadhan and Rasi cultivars, respectively (Fig. 5). Untreated control had recorded the highest per cent seed germination of 97.92 and 98.63 in HR-12 and Swarnadhan, respectively. Zinc sulphate 10^{-3} M had recorded the highest per cent seed germination (96.56%) in Rasi; but, it was statistically on par with untreated control (96.39%).

Seed treatment with chemicals often results in considerable effect on seed germination. Peratla *et al.* (2000) reported a reduction in germination of alfalfa seed due to increased concentrations of metal ions viz. cadmium (Cd), chromium (Cr), copper (Cu) and nickel (Ni), but not zinc (Zn). There was significant reduction in seed germination with cadmium and chromium at 10 ppm while it was 20 ppm for copper and nickel. But zinc did not significantly reduce seed germination even at a concentration of 40 ppm. Claire *et al.* (1991) obtained similar results in a study using nickel and other heavy metals on cabbage, lettuce, millets, radish, turnip and wheat. Flavia *et al.* (2003) reported inhibition of seed germination in soybean crop due to seed treatment with ferulic acid, coumarin and naringenin. In the present study, sodium selenite (10^{-5} M) showed significant inhibition of seed germination in all the three varieties. Zinc sulphate (10^{-3} M) has given highest per cent seed germination in Rasi while untreated control recorded highest germination in HR-12 and Swarnadhan.

5.2.3 Effect of SAR inducing chemicals on leaf blast severity

In the susceptible variety HR-12, BTH 10^{-3} M was superior in minimizing the leaf blast severity followed by salicylic acid 10^{-3} M. Sodium selenite 10^{-5} M and ferric chloride 10^{-4} M had shown some SAR inducing potential. The SAR inducing effect of zinc sulphate 10^{-3} M was negligible (Fig. 6, 7, 8 & 9). The SAR inducing chemicals applied as seed treatment followed by either one or two foliar sprays were found significantly superior to that of seed treatment alone. However, the 2nd spray did not show any significant effect in inducing any additional systemic resistance (Fig. 10, 11, 12 & 13).

In the moderately resistant variety Swarnadhan, BTH 10^{-3} M and salicylic acid 10^{-3} M were more effective in reducing the leaf blast severity, while the effect of ferric chloride 10^{-4} M, sodium selenite 10^{-5} M and zinc sulphate 10^{-3} M was found negligible (Fig. 6, 7, 8 & 9). The SAR inducing capabilities of the chemicals did not differ with the method and number of applications *viz.* seed treatment alone, seed treatment followed by either one or two foliar sprays. In the highly resistant variety Rasi, disease incidence was observed neither in untreated control nor in the chemical treatments and hence the SAR inducing capacity could not be assessed in this variety against blast disease. This is in agreement with the statement of Kessmann *et al.* (1994) that induced resistance is not the creation of resistance where there is none, but the activation of latent resistance mechanisms.

Bhattacharyya and Roy (1998) tested 21 non-conventional chemicals each at two concentrations in wet seed treatment, root-dip treatment and spray treatment against sheath blight of rice and concluded that sodium selenite at 10^{-5} M, zinc sulphate at 10^{-4} M, lithium sulphate at 10^{-4} M, calcium nitrate at 10^{-2} M, sodium fluoride at 10^{-3} M and ferric chloride at 10^{-4} M could check the disease in excised rice leaves as well as in standing plants. Highest efficacy was recorded when the combination of all three methods was used stage by stage. Manandhar *et al.* (1998) reported that ferric chloride, dipotassium hydrogen phosphate and salicylic acid significantly reduced rice blast disease severity when applied as either soil drench or as foliar spray but not as seed treatment. Neck blast incidence was significantly reduced by all 3 chemicals when applied as foliar spray at least 2 times from the seedling to the heading stage, while

greatest reduction was obtained with ferric chloride. In the present study also ferric chloride 10^{-4} M and zinc sulphate 10^{-3} M were found effective only when compared to the untreated control while salicylic acid 10^{-3} M showed better efficacy in reducing the leaf blast severity in both HR-12 and Swarnadhan.

Treatment of rice seeds with BTH enhanced SAR to *P. grisea* at 3 to 4 leaf stage of rice seedlings (Ge-Xiuchun *et al.*, 1999 and Schweizer *et al.*, 1999). Foliar application of BTH at tillering induced systemic resistance in wheat against both powdery mildew and speckled leaf blotch. BTH treatment at the end of tillering provided better protection than at mid tillering stage (Stadnik and Buchenauer, 1999). Tosi *et al.* (1999) reported that BTH applied as a soil drench and foliar spray was shown to provide good protection against *Plasmopara helianthi* in sunflower plants. In the present investigation also BTH 10^{-3} M was effective in reducing leaf blast severity in both HR-12 and Swarnadhan.

5.2.4 Effect of SAR inducing chemicals on disease parameters

There was no disease incidence in Rasi. The incubation period ranged between 3.13 days (untreated control) and 3.58 days (salicylic acid 10^{-3} M) in HR-12 (Fig. 14 and 15), while it varied between 5.29 days (untreated control) and 9.46 days (BTH 10^{-3} M) in Swarnadhan (Fig. 16 & 17). In HR-12, the lesion size ranged between 3.36 mm^2 (salicylic acid 10^{-3} M) and 5.88 mm^2 (untreated control), while in Swarnadhan, the lesion size ranged between 1.95 mm^2 (BTH 10^{-3} M) and 4.40 mm^2 (untreated control). The number of lesions/leaf ranged between 6.67 (BTH 10^{-3} M) and 12.29 (untreated control) in HR-12, while it varied between 2.79 (BTH 10^{-3} M) and 8.71

(untreated control). The number of conidia per mm^2 of lesion ranged between 1337.17 (BTH 10^{-3} M) and 2031.79 (untreated control) in HR-12, while it ranged between 805.88 (BTH 10^{-3} M) and 1219 (untreated control) in Swarnadhan.

BTH treatment (100 µg a.i./ml) caused significant reduction in the number of lesions in pea following challenge inoculation with *Mycosphaerella pinodes* compared to untreated control (Dann and Deverall, 2000). According to Oostendrop *et al.* (2001) BTH translocates systemically in plants and can take the place of salicylic acid in the natural SAR signal pathway inducing the same spectrum of resistance. In the present study also BTH at 10^{-3} M and salicylic acid 10^{-3} M induced SAR in Swarnadhan and HR-12. Kamalakannan *et al.* (2001) tested the effect of barium chloride, cadmium chloride, ferric chloride, mercuric chloride and nickel nitrate at 10^{-3} and 10^{-5} M on mycelial growth, biomass production and spore germination of *P. grisea in vitro*. Foliar spray and seed treatment at 10^{-3} M concentration of all the heavy metal salts provided moderate to substantial protection to rice seedlings at different stages. In rice, ferric chloride at 10^{-4} M induced SAR as shown by reduced lesion size, number of lesions/leaf and number of conidia/mm^2 of the lesion. Colson and Deverall (2000) reported significant reduction in number of lesions on sunflower leaves when treated with BTH against *Alternaria macrospora.*

5.3 EFFICACY OF DIFFERENT FUNGICIDES AGAINST P. GRISEA

Investigations were carried out to know the efficacy of different fungicides both *in vitro* and *in vivo*. The *in vitro* experiment was carried out by employing 'inhibition zone technique' while the field efficacy was tested both in the nursery and main field by using the fungicides as seed treatment as well as foliar sprays. The

fungicides that could not check the growth of the pathogen *in vitro* had given the best control in the field both as seed treatment as well as foliar spray. Similarly, some fungicides that were found very effective in inhibiting the fungus *in vitro* had shown dismal performance in the field. Conversely, a few fungicides have shown equal performance both *in vitro* and *in vivo*. Also, the fungicides that were ineffective as seed treatment had shown good performance as foliar sprays, while the fungicides that has given best performance as seed treatment were also found equally good as foliar sprays.

5.3.1 *In vitro* efficacy of different fungicides against *P. grisea*

Among the fungicides tested, the combination of carbendazim and epoxiconazole (25 EC) was found the most effective *in vitro* followed by carbendazim (50 WP) alone. Inhibition of mycelial growth was observed at 1 µg/ml for both carbendazim + epoxiconazole 25 EC (8.66 mm) and carbendazim 50 WP alone (4.66 mm) with an average inhibition zone of 25.33 mm and 20.63 mm, respectively, while pyroquilon 50 WP, tricyclazole 75 WP and fenoxanil 20 SC had failed to inhibit mycelial growth even up to the concentration of 1000 µg/ml (Fig. 18).

Inhibition of mycelial growth was seen at 1 µg/ml for azoxystrobin 25 SC (5.66 mm), pyraclostrobin 25 EC (4.66 mm) and RIL010 25 SC (2.66 mm), while it was 5 µg/ml for propiconazole + difenconazole 30 EC (3.66 mm), edifenphos 50 EC (3.33 mm), isoprothiolane 40 EC (3 mm), propiconazole 25 EC (2.66 mm) and iprobenfos 48 EC (1.66 mm). Formation of inhibition zone started at 10 µg/ml for epoxiconazole 7.5 EC (1.33 mm), kasugamycin 2L (0.66 mm) and difenconazole 25 EC (0.33 mm) while, it was 25 µg/ml for diniconazole 25 WP (1.33 mm). The zone of inhibition had

progressively been increasing with increased concentration for all the fungicides except pyroquilon 50 WP, tricyclazole 75 WP and fenoxanil 20 SC where no inhibition zones were observed at all concentrations.

The combination of different fungicides yielded better performance over any of the individual fungicides. Inhibition of mycelial growth has started at 10 μg/ml for epoxiconazole 7.5 EC while its combination with carbendazim inhibited mycelial growth at 1 μg/ml itself. Similarly for difenconazole 25 EC, inhibition of mycelial growth has stated at 10 μg/ml while its combination with propiconazole 25 EC started inhibiting mycelial growth at 5 μg/ml itself. However, combination of carbendazim with epoxiconazole and propiconazole with difenconazole had also started inhibiting mycelial growth at concentrations similar to that of carbendazim alone (1 μg/ml) and propiconazole alone (5 μg/ml), respectively. But the combination fungicides formed bigger inhibition zones as compared to the individual fungicides *viz.* carbendazim and propiconazole at the same concentration. The difference in the inhibition zones between two consecutive concentrations has been consistently decreasing as the concentration increased (Fig. 18).

Among the fungicides tested, the highest *in vitro* efficacy was shown by those fungicides that affect mitosis and cell division (carbendazim 50 WP) followed by the ones that disrupt respiration (azoxystrobin 25 SC, pyraclostrobin 25 EC and RIL010 25 SC). The target site for carbendazim 50 WP is β-tubulin assembly in mitosis while the target site for strobilurins *viz.* azoxystrobin, pyraclostrobin and RIL 010 is cytochrome bc_1 at Q_O site of complex III. On the other hand, melanin biosynthesis inhibitors *viz.* pyroquilon 50 WP, tricyclazole 75 WP and fenoxanil 20 SC have failed to inhibit

mycelial growth *in vitro*. However, the site of action for pyroquilon and tricyclazole is reductase in melanin biosynthesis where as the site of action for fenoxanil is dehydratase in melanin biosynthesis (Plate 44).

The next best control was offered by edifenphos 50 EC, iprobenfos 48 EC and isoprothiolane 40 EC whose mode of action is lipid and membrane synthesis. Sterol biosynthesis inhibitors *viz.* propiconazole 25 EC, epoxiconazole 7.5 EC, difenconazole 25 EC and diniconazole 25 WP were next in the order of their *in vitro* efficacy. The target site of action for edifenphos, iprobenfos and isoprothiolane is phospholipid biosynthesis and methyl transferase while it was C_{14} – demethylase in sterol biosynthesis for propiconazole, epoxiconazole, difenconazole and diniconazole. These fungicides are otherwise known as DMI-fungicides i.e. De-Methylation Inhibitors that encompass all the triazole group including the above. The *in vitro* efficacy of kasugamycin was found as good as DMI-fungicides, but this antifungal antibiotic acts on amino acid and protein synthesis.

Kapooria and Hairwadzi (1982) studied the effect of carbendazim *in vitro* on *P. grisea* at different concentrations *viz.* 100, 200, 400, 600, 800 and 1000 µg/ml and concluded that inhibition of growth had started at 800 µg/ml when the fungicide was added to the solidified medium while inhibition of growth was started at 400 µg/ml itself when the fungicide was incorporated into the molten medium. Hossain and Kulkarni (2001b) said that iprobenfos, propiconazole and carbendazim were the most effective fungicide *in vitro* followed by hexaconazole and tricyclazole at 500 ppm. In the present study, the inhibition of growth was started at 1 µg/ml for carbendazim while it was 5 µg/ml for iprobenfos and propiconazole with Rajendranagar isolate of *P. grisea*. The disparity in growth inhibition might be attributed to the differential sensitivity of the *P. grisea* isolates employed in the investigation.

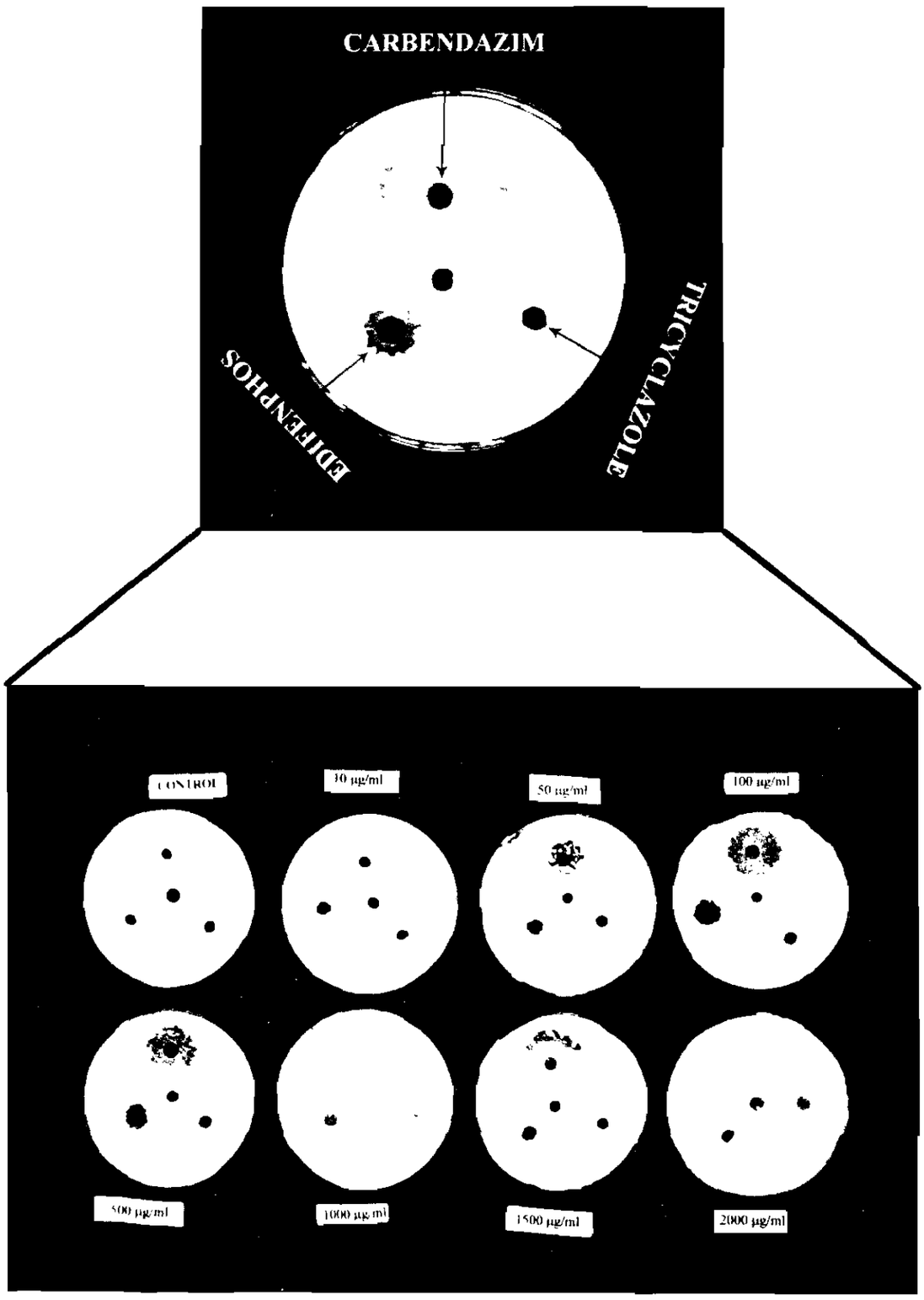


Plate 44: Comparative inhibition of mycelial growth of *P. grisea* by carbendazim 50 WP, edifenphos 50 EC and tricyclazole 75 WP at different concentrations

El Kazzaz *et al.* (1990) reported that edifenphos and iprobenfos inhibited the growth of *P. grisea, in vitro* at 75 and 100 ppm, respectively, while the minimum inhibitory concentration of tricyclazole and pyroquilon were 500 and 700 ppm, respectively. Viswanathan and Narayanasamy (1992) reported that both tricyclazole and mancozeb effectively inhibited spore germination, germ tube elongation, mycelial growth and sporulation of *P. grisea in vitro* at 500 ppm. Kim *et al.* (2000) evaluated three known melanin biosynthesis inhibitors namely KC 10017, tricyclazole and carpropamid against *P. grisea, in vitro* and concluded that all three MBI fungicides did not show any inhibitory effect on fungal growth. Pyroquilon and tricyclazole did not show any inhibitory effect on mycelial growth even up to 1000 µg/ml in the present study which is in agreement with Kim *et al.* (2000), while it contradicts with El Kazzaz et al. (1990) and Viswanathan and Narayanasamy (1992). In another study, Rashmi and Singh (1999) reported that carpropamid, a melanin biosynthesis inhibitor exhibited a little effect on *in vitro* mycelial growth and no effect on spore germination and appressoria formation. The present study also had shown that all the three melanin biosynthesis inhibitors *viz.* pyroquilon 50 WP, tricyclazole 75 WP and fenoxanil 20 SC failed to inhibit the mycelial growth of *P. grisea* in the laboratory.

5.3.2 Efficacy of fungicides as seed treatment against rice blast in the nursery

Blast disease can affect rice at all growth stages from seedling to harvesting. The disease can be devastating at any stage of the crop if left uncontrolled particularly in the susceptible cultivars. Seedling phase of rice is the most vulnerable stage for blast incidence since there will be less silica deposit in the tender leaves. Transplanted seedlings from infected nurseries are the most potential source (infection

foci) for rapid spread of the disease in the main field. Also, inoculum might be carried through seed material collected from the blast infected fields. Management of blast disease in the nursery offers several advantages like using minimum quantity of chemical for seed treatment; easy to detect and control the disease in small area of nursery and transplanting healthy seedlings in the main field will naturally delay disease establishment and development which in tern reduce the total number of sprays for blast control. Hence, seed treatment is the most economical and effective method of blast disease control in the nursery.

5.3.2.1 Effect of seed treatment on seed germination

Seed germination tests were conducted to know whether there was any effect of fungicides on seed germination. The per cent seed germination ranged between 86.33 and 95.16 (Fig. 20). It was also found that the per cent seed germination of different treatments was not consistent between the years 2002 and 2003. Goulart (1991) studied the effect of captan, totylfluanid, ethyltrianol, iprodione and thiram as seed treatment against *P. grisea* and reported that none of the treatments improved seed germination. Sader *et al.* (1990) studied the effect of seed treatment with quintozene, benomyl, carbendazim, thiram, captafol and captan on paddy seed germination and found no effect of fungicides on seed germination except quintozene. From the present results, it appears that seed treatment with fungicides under investigation neither increased nor decreased the per cent seed germination of the variety HR-12 and the difference was found non-significant with reference to untreated control (90.83%).

5.3.2.2 Effect of seed treatment on the appearance of initial disease symptoms

It took three days for the appearance of initial disease symptoms in the untreated control. Ten out of seventeen fungicides tested had delayed the appearance of initial disease symptoms ranging between 1 and 11.16 days while the rest of fungicides were not at all successful in delaying the disease incidence (Fig. 19). Pyroquilon 50 WP was found to be the best fungicide in delaying the disease appearance for maximum number of days (11.16 days) followed by azoxystrobin 25 SC (10.83 days), pyraclostrobin 25 EC (10.16 days), RIL 010 25 SC (8.33 days), tricyclazole 75 WP (6.16 days) and fenoxanil 20 SC (4 days). The other fungicides *viz.* carbendazim 50 WP (1.66 days), propiconazole 25 EC (1.5 days), carbendazim + epoxiconazole 25 EC (1.16 days) and isoprothiolane 40 EC (1 day) had marginally been successful in delaying the disease appearance. The remaining fungicides *viz.* iprobenfos 48 EC, kasugamycin 2L, difenconazole + propiconazole 30 EC, epoxiconazole 7.5 EC, edifenphos 50 EC, difenconazole 25 EC and diniconazole 25 WP were found ineffective in delaying initial disease development.

The fungicides that were found most effective in delaying the appearance of blast lesions belong to either melanin biosynthesis inhibitors (pyroquilon, tricyclazole and fenoxanil) or the strobilurins (azoxystrobin, pyraclostrobin and RIL 010) groups. The degree of protection offered by the three melanin biosynthesis inhibitors also varied considerably i.e. pyroquilon was significantly superior to fenoxanil. However, the performance of strobilurin fungicides was found more or less on par with one another. The remaining fungicides were proved to be ineffective as seed treatment except carbendazim 50 WP and propiconazole 25 EC. Peterson *et al.* (1990) found that

tricyclazole was effective as a preventive treatment for rice blast control while Greer and Webster (2001) reported azoxystrobin as most effective fungicide when used as a protectant prior to infection. Clough and Godfrey (1998) also suggested the preventive use of azoxystrobin rather than curative use. The present results are in agreement with the above authors with respect to melanin biosynthesis inhibitors and also the strobilurin fungicides.

It was also observed that delay in the appearance of initial disease symptoms was comparatively low during the year 2003. The delay in disease appearance ranged between 2 and 13.33 days during 2002 while it ranged between 1 and 9 days during 2003. This difference might be due to the congenial atmospheric temperature and relative humidity coupled with rainy days in the year 2003 compared to the dry weather that prevailed during the year 2002 even though sprinklers and polythene covers were used to build inoculum pressure during both the years.

5.3.2.3 Effect of seed treatment on leaf blast severity

Pyroquilon 50 WP was found the best fungicide with 6.66 per cent leaf blast severity at 20 days after inoculation (DAI) followed by azoxystrobin 25 SC and pyraclostrobin 25 EC with 7.33 and 8.33 per cent disease severity, respectively when compared to cent per cent disease severity in the untreated control (Fig. 20). The next best protection was offered by RIL010 25 SC, tricyclazole 75 WP and fenoxanil 20 SC with 11.16, 14.16 and 24.99 per cent disease severity, respectively. Carbendazim 50 WP and propiconazole 25 EC were comparatively good with 32.49 and 39.99 per cent disease

severity, respectively. The remaining fungicides were not good enough as seed treatment when compared to their superior counterparts even though they performed far better than the untreated control.

The appearance of initial disease symptoms and progress of leaf blast was very much delayed by both melanin biosynthesis inhibitors (MBIs) and strobilurins, while the disease appearance was very early and the disease progress was rapid in the untreated control. There was considerable variation in leaf blast control among the MBIs where as the strobilurins exhibited more or less consistent performance with in the group. This difference in MBIs in blast control as seed treatment might be attributed to their chemical structure even though the mode of action is inhibition of melanin biosynthesis in the fungal cell wall. Pyroquilon, tricyclazole and fenoxanil belong to pyrroloquinolinone, triazolobenzotriazole and propionamide groups, respectively. More over, pyroquilon and tricyclazole act on reductase in melanin biosynthesis while fenoxanil act on dehydratase in melanin biosynthesis. Among strobilurins, azoxystrobin and RIL010 belong to methoxy-acrylates group while pyraclostrobin belong to methoxy-carbamates group.

Carbendazim 50 WP which was found effective as seed treatment after MBIs and strobilurins belong to benzimidazole group inhibits β-tubulin assembly in mitosis. Leaf blast incidence had started early (3 DAI) in all the fungicides of triazole group (difenconazole 25 EC, diniconazole 25 WP, epoxiconazole 7.5 EC and propiconazole 25 EC). However, the disease progress was relatively slow with propiconazole 25 EC while it was very rapid with diniconazole 25 WP. The performance

of lipids and membrane synthesis inhibitors such as edifenphos 50 EC, iprobenfos 48 EC and isoprothiolane 40 EC as seed treatment against rice blast was moderate to minimum. The effect of kasugamycin 2L, an antifungal antibiotic that inhibits protein synthesis was also found minimal in reducing blast severity in the nursery as seed treatment.

Saifulla and Seshadri (1992) suggested that seed treatment with pyroquilon and tricyclazole (4 g/kg seed) was effective in controlling *P. grisea* in rice nurseries. Prabhu and Filippi (1993) reported that pyroquilon at the rate of 4 g/kg seed kept leaf blast severities below 5 per cent at 38 days after sowing in susceptible rice cultivars. Again, Filippi and Prabhu (1997) said that seed treatment with pyroquilon at 4 g a.i./kg seed suppressed rice blast severity by an average of 28.6 per cent. Rodriguez *et al.* (1993) showed that seed treatment with carboxin (2.5 g/kg seed), benomyl (2.5 g/kg seed), edifenphos (2 ml/kg seed), kasugamycin (2 ml/kg seed) and tricyclazole (2 g/kg seed) reduced leaf blast at least by 50 per cent when compared to untreated control. The present results are in agreement with the above authors.

5.3.3 Efficacy of fungicides as foliar spray in the nursery

The rice seedlings in the nursery were inoculated with Rajendranagar isolate of *P. grisea* on 15th day after sowing. Fungicides were sprayed in all the experimental plots immediately after the appearance of initial disease symptoms as per the treatments. The per cent leaf blast severity was non-significant among the treatments including untreated control before spraying the fungicides. The fungicides were sprayed only once and the disease progress was recorded till leaf blast severity in the untreated

control reached hundred per cent (20 DAI) at 5 day interval. The reasons for high disease incidence and rapid progress in the nursery beds were, high relative humidity created by using water sprinklers; covering the nursery beds with polythene covers over night and tenderness of the leaves. The results on the efficacy of fungicides as foliar spray are compared with those of seed treatment as well as the *in vitro* inhibition of mycelial growth. Pooled data of both the treatments *viz.* foliar spray and seed treatment was used for the comparison in the discussion.

The per cent leaf blast severity had progressed from 1.66 to 9.99, 4.49 to 18.83 and 4.65 to 25.65 between 5 DAI and 20 DAI in the plots treated with pyroquilon 50 WP, tricyclazole 75 WP and fenoxanil 20 SC, respectively (Fig. 21). The leaf blast severity was comparatively low in case of seed treatments as compared to foliar spray i.e. 6.66 and 14.16 against 9.99 and 18.83 per cent with pyroquilon 50 WP and tricyclazole 50 WP, respectively at 20 DAI or when the leaf blast severity in the untreated control reached hundred per cent. However, the leaf blast severity was found almost equal in case of fenoxanil 20 SC i.e. 24.99 against 25.65 per cent in case of seed treatment and foliar spray, respectively. But, pyroquilon 50 WP, tricyclazole 75 WP and fenoxanil 20 SC completely failed to inhibit mycelial growth of the fungus *in vitro* even up to the concentration of 1000 µg/ml. Thus, there is no comparison between the *in vitro* and *in vivo* performance of these three melanin biosynthesis inhibiting fungicides.

Similarly, the per cent leaf blast severity had progressed from 1.99 to 13.49, 2.49 to 15.16 and 2.49 to 17.16 between 5 DAI and 20 DAI in the plots treated with azoxystrobin 25 SC, RIL010 25 SC and pyraclostrobin 25 EC, respectively (Fig. 21). However, the leaf blast severity was comparatively low in case of seed treatments as

compared to the foliar spray i.e. 7.33, 11.16 and 8.33 against 13.49, 15.16 and 17.16 per cent with azoxystrobin 25 EC, RIL010 25 SC and pyraclostrobin 25 EC, respectively at 20 DAI or when the leaf blast severity in the untreated control reached hundred per cent (Fig. 20). The *in vitro* inhibition of mycelial growth by these three strobilurin fungicides was also found excellent indicating a better correlation between their *in vitro* and *in vivo* performance.

The per cent leaf blast severity had progressed from 5.33 to 27.99, 5.83 to 32.16 and 6.16 to 32.83 between 5 DAI and 20 DAI in the plots treated with iprobenfos 48 EC, isoprothiolane 40 EC and edifenphos 50 EC, respectively (Fig. 21). However, the leaf blast severity was very high in case of seed treatments as compared to the foliar spray i.e. 49, 46.66 and 62.49 against 27.99, 32.16 and 32.83 with iprobenfos 48 EC, isoprothiolane 40 EC and edifenphos 50 EC, respectively at 20 DAI or when the leaf blast severity in the untreated control reached hundred per cent (Fig. 20). The *in vitro* inhibition of mycelial growth by these three fungicides was found very good, indicating that iprobenfos 48 EC, isoprothiolane 40 EC and edifenphos 50 EC had effectively carried their *in vitro* efficacy to *in vivo* also. But, they had shown better efficacy when applied as foliar spray rather than seed treatment.

The per cent leaf blast severity had progressed from 6.49 to 34.99 between 5 DAI to 20 DAI in the plots treated with kasugamycin 2L applied as foliar spray (Fig. 21). However, the leaf blast severity was comparatively high in case of seed treatment i.e. 51.66 against 34.99 per cent in foliar spray (Fig. 20) at 20 DAI. It indicates that kasugamycin 2L had suppressed leaf blast more effectively as curative foliar spray rather than preventive seed treatment. Kasugamycin 2L had effectively inhibited mycelial growth of the fungus. Hence, kasugamycin 2L had effectively carried its *in vitro* efficacy to the field, but as preventive foliar spray rather then curative seed treatment.

The per cent leaf blast severity had progressed from 9.33 to 39.16 between 5 DAI and 20 DAI in the plots treated with carbendazim 50 WP as foliar spray (Fig. 21). However, the leaf blast severity was comparatively low in case of seed treatment i.e. 32.49 against 39.16 per cent in foliar spray (Fig. 20). It indicates carbendazim 50 WP had shown greater efficacy as seed treatment when compared to foliar spray. Surprisingly, carbendazim 50 WP had shown the highest inhibition of mycelial growth than any other individual fungicide under investigation. But, its efficacy in controlling leaf blast was found very poor as compared to strobilurins and melanin biosynthesis inhibitors. Thus, the carbendazim 50 WP which showed excellent *in vitro* efficacy has recorded poor performance in suppressing leaf blast disease *in vivo*.

In case of propiconazole 25 EC, epoxiconazole 7.5 EC, difenconazole 25 EC and diniconazole 25 WP, the per cent leaf blast had progressed from 6 to 33.83, 11.16 to 51.99, 12.33 to 55.33 and 14.49 to 65.16, respectively between 5 DAI and 20 DAI when applied as foliar spray (Fig. 21). But the leaf blast severity was comparatively high in case seed treatments as compared to foliar spray i.e. 39.99, 59.16, 65.83 and 86.66 against 33.83, 51.99, 55.33 and 65.16 with propiconazole 25 EC, epoxiconazole 7.5 EC, difenconazole 25 EC and diniconazole 25 WP, respectively at 20 DAI or when the leaf blast severity in untreated control reached hundred per cent (Fig. 20). Even though all these four chemicals belong to triazole group, there was marked difference among fungicides with in the group i.e. the *in vivo* efficacy of propiconazole 25 EC (33.83%) was almost double to that of diniconazole (65.16%). However, propiconazole 25 EC, epoxiconazole 7.5 EC, difenconazole 25 EC and diniconazole 25 WP had effectively inhibited mycelial growth of the fungus *in vitro*. But, the performance of these four chemicals was very poor in the field as compared to those of strobilurins, melanin biosynthesis inhibitors and phosphoro-thiolates.

The per cent leaf blast severity had progressed from 7.83 to 39.99 and 8.49 to 43.16 between 5 DAI and 20 DAI in the plots treated with difenconazole + propiconazole 30 EC and carbendazim + epoxiconazole 25 EC, respectively. The leaf blast severity in the case of difenconazole + propiconazole 30 EC and carbendazim + epoxiconazole 25 EC at 20 DAI (when the leaf blast severity in the untreated control reached hundred per cent) was high when compared to propiconazole 25 EC (39.99% against 33.83%) and carbendazim 50 WP (43.16% against 39.16%) i.e. propiconazole 25 EC and carbendazim 50 WP performed better individually as compared to the combination. Conversely, the leaf blast severity in the case of difenconazole + propiconazole 30 EC and carbendazim + epoxiconazole 25 EC at 20 DAI was low when compared to difenconazole 25 EC (39.99% against 55.33%) and epoxiconazole 7.5 EC (43.16% against 51.99%) i.e. the combination of fungicides performed better as compared to difenconazole 25 EC and epoxiconazole 7.5 EC, individually. The combination of carbendazim and epoxiconazole had excellently inhibited *in vitro* mycelial growth of the fungus, but its *in vivo* performance was very poor. Similarly, the combination of difenconazole and propiconazole was also found effective *in vitro*, but failed to show the same *in vivo*.

Naidu and Reddy (1989) reported that carbendazim, edifenphos, pyroquilon, isoprothiolane, iprobenfos and thiophanate-methyl significantly reduced leaf blast on a susceptible rice cultivar IR-50. Application of isoprothiolane, edifenphos, iprobenfos, kasugamycin, blasticidin-S or ferimzone to rice plants after the appearance of rice blast lesions inhibited lesion enlargement and spore production. A linear relationship was found between days after application of each fungicide and the area of lesion during a particular period (Ishiguro *et al.*, 1992). According to Mustaq Ahmad and Ahmad (1992), edifenphos was the best fungicide in the field followed by iprobenfos, pyroquilon

and kasugamycin both in controlling leaf and neck blast and in increasing the grain yield. Paul *et al.* (1993) said that tricyclazole gave more effective control when compared to iprobenfos, carbendazim, metiram, edifenphos and pyroquilon.

5.3.4 Efficacy of fungicide sprays against rice blast in the main field

Leaf blast incidence and disease severity in the main field were low as compared to the nursery beds even though the plots were sprayed with spore suspensions of *P. grisea* in addition to natural inoculum. The progress of blast disease was also slow and it had seldom reached hundred per cent as in nursery beds. The reasons for low blast incidence and slow progress in the main field were dry weather and hardness in the leaves as compared to the nursery.

The supremacy of pyroquilon 50 WP was inadvertently continued in controlling both leaf blast severity and neck blast incidence. The same trend was also continued in increasing the total grain yield while reducing the quantity of ill-filled grain. The performance of other two melanin biosynthesis inhibitors *viz.* tricyclazole 75 WP and fenoxanil 20 SC was next only to the strobilurin fungicides *viz.* azoxystrobin 25 SC, pyraclostrobin 25 EC and RIL 010 25 SC. There were 2.66 per cent of lesioned necks with out any broken ones in the plots treated with tricyclazole 75 WP while fenoxanil 20 SC produced 4 per cent lesioned necks and 1.66 per cent broken necks. Conversely, pyroquilon 50 WP had produced neither lesioned necks nor broken necks against 21.66 per cent lesioned necks and 17.33 per cent broken necks in the untreated control. The highest grain yield of 2.9 t/ha was obtained from the plots treated with pyroquilon 50 WP as against 1.8 t/ha from the untreated control (Fig. 22, 23 & 24) plots.

The strobilurin fungicides *viz.* azoxystrobin 25 SC, pyraclostrobin 25 EC and RIL 010 25 SC had shown the most consistent performance in reducing both leaf blast and neck blast incidence while increasing the total grain yield. The strobilurins were proved as the best fungicides for blast disease management after melanin biosynthesis inhibitor pyroquilon 50 WP. However, the other two melanin biosynthesis inhibitors *viz.* tricyclazole 75 WP and fenoxanil 20 SC fell below the strobilurins not only in containing the leaf and neck blast but also in increasing the total grain yield (Fig. 22, 23 & 24).

Iprobenfos 48 EC, isoprothiolane 40 EC and edifenphos 50 EC had shown better efficacy against rice blast after strobilurins and MBIs which was reflected by total grain yield of 2.6 t/ha, 2.6 t/ha and 2.5 t/ha, respectively. Isoprothiolane 40 EC had produced less number of lesioned necks i.e. 2.5 per cent as against 4.99 and 5.49 per cent in iprobenfos 48 EC and edifenphos 50 EC, respectively. However, there were no broken necks in the plots treated with these fungicides (Fig. 22, 23 & 24).

Kasugamycin 2L and carbendazim 50 WP were next in the other of their efficacy followed by the combination fungicides *viz.* difenconazole + propiconazole 30 EC and carbendazim + epoxiconazole 25 EC. However, the performance of combination fungicides was superior to difenconazole 25 EC and epoxiconazole 7.5 EC, but inferior to propiconazole 25 EC and carbendazim 50 WP. The field efficacy of difenconazole 25 EC, epoxiconazole 7.5 EC and diniconazole 25 WP was not up to the mark even though they had suppressed rice blast far better compared to untreated control (Fig. 22, 23 & 24).

Awoderu (1990) reported yield loss up to 58.8 per cent which was attributed to the neck blast. Filippi and Prabhu (1997) found significant variation in yield because of both leaf blast and panicle blast. The losses in grain yield in the rice cultivars *viz.* IAC 202, Caiapo, Rio Paraniba and Araguaia due to panicle blast were 44.8, 27.4, 24.4 and 18.2 per cent, respectively (Prabhu *et al.*, 2003). Similarly, Condole *et al.* (1999) studied the effect of panicle blast on the physical properties and milling quality of rice and concluded that the occurrence of unfilled, chalky and fissured kernels in diseased samples were higher by 30, 21, and 7 per cent, respectively than in blast free samples. Blast reduced kernel bulk density by 140 kg/m^3 and head rice recovery by 12 per cent. The moisture content of rough rice in blast infected panicles was lower than the average moisture content of rough rice from blast free samples. In the present investigation incidence of neck blast has greater correlation with yield reduction compared to leaf blast. This may be due to the fact that rice plants can sustain the loss of certain leaf area with out significant reduction in the grain yield at peak vegetative phase. Infected necks hinder the flow of food material to the grain and hence bear a direct impact on the grain yield and its quality. Lesioned necks produced significant quantities of ill-filled grain while the broken necks resulted in complete chaffy grain.

Moletti *et al.* (1988) concluded that pyroquilon (1.5 kg/ha) applied twice gave good results against leaf blast followed by tricyclazole at 0.75 kg/ha. But, tricyclazole gave good control of neck blast followed by pyroquilon. In the present study pyroquilon 50 WP at 1 kg/ha applied twice was superior to tricyclazole 75 WP at 0.60 kg/ha in controlling both leaf blast and neck blast. Naidu and Reddy (1989) reported that

carbendazim, edifenphos, pyroquilon, isoprothiolane, iprobenfos and thiophanate-methyl reduced leaf blast but not neck blast on a susceptible rice cultivar IR-50 when applied as foliar spray at tillering, panicle initiation and flowering stages. In the present investigation the fungicides that were found effective in controlling leaf blast were equally good in controlling neck blast also in the highly susceptible cultivar HR-12. Fenoxanil 20 SC was comparatively poor in controlling neck blast among the MBI fungicides while isoprothiolane 40 EC exhibited better control of neck blast compared to leaf blast. Tricyclazole was the most effective compared to benomyl, edifenphos and thiabendazole in reducing the neck blast though the differences between grain yields were not statistically significant (Okhovot, 1989). In the present investigation also tricyclazole was superior to edifenphos while benomyl and thiabendazole were not included in the study.

According to Mustaq Ahmad and Ahmad (1992), edifenphos was the best fungicide in the field followed by iprobenfos, pyroquilon and kasugamycin both in controlling leaf and neck blast and in increasing the grain yield. In the present study pyroquilon 50 WP exhibited far better efficacy compared to edifenphos 50 EC while iprobenfos 48 EC and kasugamycin 2 L were found equivalent to edifenphos in controlling both leaf and neck blast. Paul *et al.* (1993) said that tricyclazole gave effective control when compared to iprobenfos, carbendazim, metiram, edifenphos and pyroquilon. But in present investigation pyroquilon 50 WP was the most effective and superior to tricyclazole 75 WP and it agrees with Paul *et al.* (1993) regarding the superiority of tricyclazole over iprobenfos, edifenphos and carbendazim. Ram Singh *et al.* (1994)

concluded that tricyclazole and propiconazole were the most effective in reducing neck blast and increasing grain yield. In the present investigation also propiconazole 25 EC exhibited good amount of control that was comparable to edifenphos 50 EC, kasugamycin 2 L and carbendazim 50 WP while tricyclazole 75 WP was more effective than propiconazole 25 EC in controlling both leaf and neck blast.

Saifulla (1994) tested tricyclazole, blasticidin, carbendazim, cyproconazole, chlorothalonil, bitertenol, propiconazole and hexaconazole for blast disease control using the susceptible variety Mandya Vijaya and concluded that the best disease control and yield were achieved with tricyclazole and blasticidin. Tirmali and Patil (2000) reported that tricyclazole showed superior performance over propineb, carpropamid, fluquinconazole, prochloraz and epoxiconazole which reduced neck blast incidence by 50.06% and increased grain yield by 89.13% while epoxiconazole (12.5 SC) was the next best fungicide with 41.3% increase in the grain yield. But in the present investigation, out of seventeen fungicides tested epoxiconazole 7.5 EC stood last but one position in controlling both leaf and neck blast; however, the other fungicides used by Tirmali and Patil (2000) were not included in the present study.

Becerra and Tosquicy (2001) reported that the best control of rice blast disease and the highest grain yield (4.43 t/ha) were obtained with azoxystrobin at the rate of 0.6 l/ha on the susceptible rice cultivar Milagro Filipino Depurodo in Mexico. Similarly, Cortesi and Giuditta (2003) reported that both strobilurins and tricyclazole were highly effective against leaf blast and neck blast by reducing incidence and disease severity by 90-100% and 75-90%, respectively. They reported that the difference in

efficacy between two treatments of strobilurins and one treatment of tricyclazole at 450 g/ha was not significant. They also stated that there was no significant improvement in efficacy of tricyclazole (225 g/ha) in combination with propiconazole (125 g/ha) when compared to tricyclazole (225 g/ha) alone. It was suggested that two sprays at late booting and at heading stages with azoxystrobin or trifloxystrobin at 250 and 125 g/ha, respectively or one treatment with tricyclazole at 450 g/ha followed by one treatment with strobilurin when severe rice blast epidemics are predicted. In the present investigation also the performance of strobilurins *viz.* azoxystrobin, RIL 010 and pyraclostrobin was excellent which can be compared to the best blasticides currently available in the market.

5.4 RELATIVE SENSITIVITY OF P. GRISEA ISOLATES TO DIFFERENT FUNGICIDES

Rice blast samples were collected from 12 different parts of India including Andhra Pradesh. Single spore isolations of *P. grisea* were made to test their sensitivity towards some commonly used fungicides in the field for blast control. All the isolates were tested *in vitro* for their sensitivity towards carbendazim, edifenphos and iprobenfos at different concentrations *viz.* 0, 1, 5, 10, 25, 50, 100, 250, 500 and 1000 µg/ml. Inhibition of mycelial growth as well as spore germination were taken as criterion for sensitivity studies. Rice blast samples were also collected from three different locations in Andhra Pradesh *viz.* Tirupathi, Naidupeta and Kadapa to know the base line sensitivity as these isolates were collected from the rice fields that had not received any fungicide sprays.

5.4.1 Relative sensitivity of *P. grisea* isolates to carbendazim *in vitro*

The results showed a highly varied response of different isolates towards carbendazim with ED_{50} values ranging between 0.39 – 54.53 µg/ml and 0.55 – 56.18 µg/ml for mycelial growth and spore germination, respectively (Fig. 25). The MIC values also ranged between 5 and 500 µg/ml for both mycelial growth and spore germination. Cuttak isolate recorded the highest ED_{50} values of 54.63 and 56.18 µg/ml for mycelial growth and spore germination, respectively against 0.39 and 0.55 µg/ml, respectively by Medchel isolate. Similarly, Cuttak isolate had recorded the maximum MIC value of 500 µg/ml against a minimum of 5 µg/ml by the Medchel isolate (Plates 34 & 35). Where as the mean base line sensitivity for carbendazim was 0.54 and 0.70 µg/ml for mycelial growth and spore germination, respectively, with a MIC of 5 µg/ml.

Reddy *et al.* (1980) reported carbendazim resistance in *Gloeosporium ampelophagum* for the first time in India and stated that an isolate of *G. ampelophagum* developed 4-fold (500-2000 µg/ml) resistance after training *in vitro*. The mechanism of resistance to carbendazim was demonstrated by Tripathi and Schlosser in 1982. They found that the uptake of MBC was significantly less in resistant strains of *Botrytis cinerea* when compared to the susceptible ones. They had suggested reduced uptake and lesser binding of MBC to tubulin-like protein contributed towards MBC resistance. Mohiotto (1985) reported carbendazim and benomyl tolerant isolates of *Cercosporidium personatum* could grow well in oat meal agar containing 100 µg/ml, while sensitive isolates were inhibited at 1 µg/ml. Similarly several scientists reported carbendazim resistant isolates of *G. ampelophagum* from the vine yards of Punjab state in India (Kumar and Thind, 1992; Thind *et al.*, 1994 and Mohan and Thind, 1995). Carbendazim

resistance was also reported in *Venturia inequalis* by Gupta and Sharma (1996) while Mingguo and Xiu (2001) reported carbendazim resistance in *Fusarium geaminearuni.*

Georgopoulos (1977) opined that the grape vine powdery mildew pathogen, *Uncinula necator* with several spore generations and infection cycles per year generally develops resistance more rapidly. Hemantha Kumar *et al.* (1998) suggested that the reduced efficacy of carbendazim against *G. ampelophagum* might be due to usage of high doses and post symptoms sprays that exerted selection pressure on the pathogen. Hemantha Kumar (2003) also indicated that high selection pressure of carbendazim and related fungicides lead to evolution of carbendazim resistant population of *U. nacator* in grape growing areas of Andhra Pradesh. In the present study, the Malan isolate also developed high degree of tolerance towards carbendazim which was indicated by its MIC value (500 µg/ml) at par with Cuttak isolate. Similarly, the other isolates *viz.* Nellore, Gerua, Mandya, Almora, Pondicherry, Maruteru, Kapurthala, Rajendranagar and Pattambi had also developed different degrees of tolerance towards carbendazim with the minimum inhibitory concentrations ranging between 250 and 10 µg/ml. The differential sensitivity of the isolates towards carbendazim indicates the magnitude of selection pressure exerted by carbendazim and/or related fungicides used for blast control in the respective areas. More over, discontinued use of carbendazim and related fungicides reduces carbendazim resistant populations in the nature.

5.4.2 Relative sensitivity of *P. grisea* isolates to edifenphos *in vitro*

The investigations revealed a highly varied response of different isolates towards edifenphos with ED_{50} values ranging between 0.73 – 30.56 µg/ml and 0.77 – 12.35 µg/ml for mycelial growth and spore germination, respectively (Fig. 26). The MIC

values also ranged between 5 and 250 µg/ml for both mycelial growth and spore germination. Mandya isolate recorded the highest ED_{50} values of 30.56 and 32.35 µg/ml for mycelial growth and spore germination, respectively against 0.73 and 0.77 µg/ml, respectively by Pondicherry isolate. Similarly, Mandya isolate had recorded the maximum MIC value of 250 µg/ml against a minimum of 5 µg/ml by the Pondicherry isolate (Plates 36 & 37). Where as the mean base line sensitivity for edifenphos was 0.52 and 0.67 µg/ml for mycelial growth and spore germination, respectively, with a MIC of 5 µg/ml.

Rose Maria (1987) reported that *P. grisea* acquired tolerance to the fungicide edifenphos just by one exposure. *P. grisea* adopted 33 times the ED_{50} value of edifenphos. Yamaguchi (1988) reported kasugamycin and iprobenfos tolerant strains of *P. grisea* in various parts of Japan and also found that IBP tolerant strains were cross tolerant to edifenphos and isoprothiolane. Yaoita and Goh (1990) investigated the effect blasticides on the development of tolerant strains of *P. grisea*. They opined that the blasticides which were related to the development of tolerant strains were iprobenfos, edifenphos, isoprothiolane, kasugamycin and blasticidin S. Peng *et al.* (1993) reported field isolates of *P. grisea* that developed tolerance to isoprothiolane with an average EC_{50} of 17.79 µg/ml compared to 5.71 µg/ml for sensitive isolates. They opined that most tolerant isolates occurred in double harvesting rice areas of Southern China with a frequency of 8.33 per cent. Zhang Shuya *et al.* (2005) obtained an azoxystrobin resistant mutant *viz.* B9-R2 by exposing *P. grisea* to ultraviolet radiation. The amount of conidia and spore germination of B9-R2 was 65.8 and 12.9 per cent lower than the wild parent isolate B9, respectively.

In the present study, the Maruteru isolate also developed high degree of tolerance towards edifenphos which was indicated by its MIC value (250 µg/ml) at par with Mandya isolate. Similarly, the other isolates *viz.* Malan, Rajendranagar, Cuttak, Nellore, Medchel, Pattambi, Gerua and Almora had also developed different degrees of tolerance towards edifenphos with the minimum inhibitory concentrations ranging between 100 and 10 µg/ml. The differential sensitivity of the isolates towards edifenphos indicates the magnitude of selection pressure exerted by edifenphos and/or related fungicides used for blast control in the respective areas.

5.4.3 Relative sensitivity of *P. grisea* isolates to iprobenfos *in vitro*

The investigations revealed a highly varied response of different isolates towards iprobenfos with ED_{50} values ranging between 0.66 – 31.85 µg/ml and 0.68 – 32.57 µg/ml for mycelial growth and spore germination, respectively (Fig. 27). The MIC values also ranged between 5 and 250 µg/ml for both mycelial growth and spore germination. Maruteru isolate recorded the highest ED_{50} values of 31.85 and 32.57 µg/ml for mycelial growth and spore germination, respectively against 0.66 and 0.68 µg/ml, respectively by Pondicherry isolate. Similarly, Maruteru isolate had recorded the maximum MIC value of 250 µg/ml against a minimum of 5 µg/ml by the Pondicherry isolate (Plates 38 & 39). Where as the mean base line sensitivity for edifenphos was 0.51 and 0.66 µg/ml for mycelial growth and spore germination, respectively, with a MIC of 5 µg/ml (Table 31).

Osada and Imouye (1980) observed diminishing control of rice blast disease by IBP when compared to other blasticides. The minimal inhibitory concentration (MIC) value of isolates causing a diminishing effect of IBP in the field were above 40

ppm. Most isolates showed the resistance to IBP *in vitro* also suggesting the occurrence of IBP-resistant strains in that locality. Taga *et al.* (1982) obtained mutants resistant to IBP from wild-type strains of *P. grisea* isolated from finger millet. Osada (1983b) examined the effect of rice cultivars differing in field resistance to blast on population change in resistant strains to Kasugamycin and/or IBP by inoculating the mixture of each strain on the three cultivars Mokoto (ss), Sasamishi ki (s) and Toyonishiki (r) and observed no difference among the three rice cultivars with regard to isolation frequency of resistant strains to kasugamycin and iprobenfos. Yamaguchi (1988) reported kasugamycin and iprobenfos tolerant strains of *P. grisea* in various parts of Japan and also found that kasugamycin tolerant strains were cross tolerant to blasticidin S, IBP tolerant strains to edifenphos and isoprothiolane. Yaoita and Goh (1990) investigated the effect of blasticides on the development of tolerant strains of *P. grisea*. They opined that the blasticides which were related to the development of tolerant strains were iprobenfos, edifenphos, isoprothiolane, kasugamycin and blasticidin S.

In the present study, the Mandya isolate also developed high degree of tolerance towards iprobenfos which was indicated by its MIC value (250 µg/ml) at par with Maruteru isolate. Similarly, the other isolates *viz.* Malan, Rajendranagar, Cuttak, Nellore, Medchel, Gerua, Pattambi, Kapurthala and Almora had also developed different degrees of tolerance towards iprobenfos with the minimum inhibitory concentrations ranging between 100 and 10 µg/ml. The differential sensitivity of the isolates towards iprobenfos indicates the magnitude of selection pressure exerted by iprobenfos and/or related fungicides used for blast control in the respective areas.

5.5 CROSS RESISTANCE OF CARBENDAZIM, EDIFENPHOS AND IPROBENPHOS RESISTANT ISOLATES TO OTHER FUNGICIDES

Cross resistance among microorganisms occurs when resistance to a chemical is conferred to another chemical mediated by the same genetic factor (Delp, 1980). If a mutation affects sensitivity to one toxicant and not to another, the two compounds are said to be uncorrelated in terms of cross resistance, a positive correlation exists when the mutant is less sensitive or less resistant to both fungicides than the wild type strain (Georgopoulos, 1982). If the mutant is more resistant than the wild type to the one fungicide and more sensitive than wild type to the other, such a phenomenon is called negatively correlated cross resistance.

The results obtained clearly indicate that development of resistance towards carbendazim had no correlation with the fungicides *viz.* pyraclostrobin and kasugamycin as there was no change in the MIC values of both carbendazim resistant and sensitive isolates *viz.* Cuttak and Medchel, respectively. The minimum inhibitory concentration of pyraclostrobin was 5 µg/ml while it was 250 µg/ml for kasugamycin for both mycelial growth and spore germination (Plates 28 & 29). The carbendazim resistant isolate had shown partial resistance towards iprobenfos, isoprothiolane and edifenphos. The ED_{50} values of edifenphos, iprobenfos and isoprothiolane were high in the resistant isolate as compared to the sensitive isolate. But, there was no change in the MIC values (50 µg/ml) of edifenphos for both resistant and sensitive isolates while the MIC values of isoprothiolane were slightly higher in the resistant isolate (25 µg/ml) as compared to the sensitive isolate (10 µg/ml). Kasugamycin inhibited the mycelial growth completely at

100 µg/ml while the MIC for spore germination was 250 µg/ml which indicates kasugamycin is a good inhibitor of mycelial growth as compared to spore germination.

The results presented in Tables 33 and 34 reveal that development of resistance towards edifenphos will contribute to the development of positive cross resistance not only to iprobenfos but also to isoprothiolane. Similarly, development of resistance towards iprobenfos will also contribute towards the tolerance of edifenphos and isoprothiolane (Fig. 30 & 31). The MIC values of edifenphos and iprobenfos resistant isolates (250 µg/ml) were very high as compared to those of sensitive isolates (5 µg/ml). Development of resistance to either edifenphos or iprobenfos had no correlation with the fungicides *viz.* pyraclostrobin, kasugamycin and carbendazim as there was no change in the MIC values of both resistant and sensitive isolates. Kasugamycin showed good inhibition of mycelial growth than spore germination.

Brown *et al.* (1984) demonstrated that carbendazim tolerant isolates of *Pseudocercosporella herpotrichoides* were cross tolerant to benomyl, thiophanate-methyl and to a lesser degree to thiobendazole, but not to prochloraz. Benzimidazole resistant isolates of *Septoria tritici* could tolerate upto 1000 µg/ml of MBC, whereas sensitive isolates could tolerate 0.2 – 0.5 µg/ml. Tolerant isolates were cross resistant to thiabendazole and thiophanate methyl (Fisher and Griffen, 1984). Carbendazim tolerant strain of *Botrytis cinerea* from chickpea showed positively correlated cross tolerance with mancozeb, thiram and triadimefon (Uday Bhan and Chatrath, 1994). Mohan and Thind (1995) reported that carbendazim resistant isolates of *G. ampelophagum* showed cross resistance to other formulations of carbendazim but the resistant isolates appear equally sensitive to multi-site inhibitors like captan, mancozeb and Bordeaux mixture.

Taga *et al.* (1982) obtained mutants resistant to IBP from wild-type strains of *P. grisea* isolated from finger millet and were grouped into two classes, highly resistant (HR) and moderately resistant (MR). HR strains grew at 0.2 mM IBP and showed cross resistance to isoprothiolane. In constrast, MR strains did not grow at 0.2 mM IBP and were sensitive to isoprothiolane. Osada (1983a) observed reduced field efficacy of isoprothiolane against rice blast due to the prevalence of iprobenfos resistant strains in the rice ecosystem. Yamaguchi (1988) reported kasugamycin and iprobenfos tolerant strains of *P. grisea* in various parts of Japan and also found that IBP tolerant strains to edifenphos and isoprothiolane. Yaoita and Goh (1990) opined that the blasticides which were related to the development of tolerant strains were iprobenfos, edifenphos, isoprothiolane, kasugamycin and blasticidin S.

In the present study the positive cross resistance among edifenphos, iprobenfos and isoprothiolane might be attributed to similar mode of action of these fungicides i.e. affecting methyl transferase in phospholipid biosysnthesis. Edifenphos and iprobenfos belong to phosphorothiolate group while isoprothiolane belongs to dithiolane group. There is more correlation between edifenphos and iprobenfos as compared to isoprothiolane and edifenphos or iprobenfos. Pyraclostrobin acts on respiration (Quinone Outside Inhibitors); carbendazim affects β-tubulin assembly in mitosis and kasugamycin affects amino acids and protein synthesis. Hence there is no correlation among pyraclostrobin, carbendazim and kasugamycin. Development of partial resistance by carbendazim resistant isolate towards edifenphos and iprobenfos may be due to simultaneous exposure of the pathogen to edifenphos and iprobenfos in the field.

5.6 PERSISTENCE OF ACQUIRED RESISTANCE IN THE RESISTANT ISOLATES

The isolates of *P. grisea viz.* Cuttak, Mandya and Maruteru that developed resistance to fungicides *viz.* carbendazim, edifenphos and iprobenfos respectively, were grown in oat meal agar medium for 12 successive generations to study the persistence of acquired resistance. The current results reveal that ED_{50} value of Cuttak isolate was reduced by 33 and 47 per cent in mycelial growth and spore germination, respectively (Fig. 32 & 33). Where as the minimum inhibitory concentration was reduced by hundred per cent i.e. from 500 µg/ml to 250 µg/ml in Cuttak isolate. The ED_{50} value of Mandya isolate was reduced by 13 and 11 per cent in mycelial growth and spore germination, respectively, while the ED_{50} value of Maruteru isolate was reduced by 1 and 10 per cent in mycelial growth and spore germination, respectively. However, there was no change in the MIC value (250 µg/ml) of both Mandya and Maruteru isolates in either of the methods. The persistence of acquired resistance was comparatively low in fungal spores when compared to the mycelium.

Viswanathan and Narayanasamy (1991) reported that *P. grisea* had developed increased tolerance towards mancozeb following repeated transfers in fungicide amended media which was lost after five successive generations on fungicide-free media. Rose Maria (1987) reported that *Cochliobolus miyabeanus* and *P. grisea* acquired tolerance to the fungicide edifenphos just by one exposure. *Cochliobolus miyabeanus* and *P. grisea* adopted 11 and 33 times the ED_{50} value of edifenphos, respectively. The acquired tolerance in *C. miyabeanus* was not of permanent nature and

was lost on maintenance in fungicide-free medium, but the acquired tolerance of *P. grisea* was totally retained. However, both fungi lost their sporulating capacity when transferred to fungicide-free media. Similar results were obtained in the present investigation also where carbendazim resistant isolates lost their tolerance very quickly indicating that the resistance development may due to adaptation. But it was not so happened with the edifenphos and iprobenfos resistant isolates even after 12 generations indicating the change in the genes related to resistance development.

5.7 FUNGICIDE RESISTANCE MANAGEMENT

Mixtures of resistance prone fungicides with other fungicides having different mechanisms of action provide one of the best strategies that have proved highly effective to limit the build up of fungicide resistance in the field (Staub and Sozzi, 1983). In the present study, experiments were conducted on resistance management in *P. grisea* both in the laboratory and glass house. Fungicides with varied modes of action *viz.* kasugamycin 2 L (amino acids and protein synthesis), propiconazole 25 EC (sterol biosynthesis in membranes), mancozeb 75 WP (multi-site contact activity), tricyclazole 75 WP (Melanin biosynthesis in cell wall) and BTH 50 WG (host plant defense induction) were used in combination with carbendazim (mitosis and cell division), edifenphos and iprobenfos (lipid and membrane synthesis) to study their efficacy against Cuttak (carbendazim resistant), Mandya (edifenphos resistant) and Maruteru (iprobenfos resistant) isolates, respectively.

The results obtained in the present investigation reveal that tricyclazole 75 WP and BTH 50 WG did not show any inhibitory effect on the fungus *in vitro*

individually, while mancozeb 75 WP showed the highest inhibition of both mycelial growth and spore germination when compared to carbendazim 50 WP, kasugamycin 2L and propiconazole 25 EC, individually. But, tricyclazole 75 WP recorded the lowest blast severity of 20.67 per cent as compared to 66.67 per cent in mancozeb 75 WP and 64.67 per cent in BTH 50 WG (Fig. 34). The combination of carbendazim 50 WP with kasugamycin 2L or propiconazole 25 EC and mancozeb 75 WP reduced the minimum inhibitory concentration from 500 µg/ml to 100, 100 and 50 µg/ml, respectively. The same trend was observed in ED_{50} values also as for as carbendazim was concerned. However, there was no change in the MIC values of kasugamycin 2L, propiconazole 25 EC and mancozeb 75 WP when mixed with carbendazim though there was slight reduction in the ED_{50} values. On the other hand, tricyclazole 75 WP and BTH 50 WG inhibited both mycelial growth and spore germination in combination with carbendazim 50 WP *in vitro*. But, there was no significant reduction in leaf blast severity due to combination of tricyclazole 75 WP and carbendazim 50 WP, while the mixture of BTH 50 WG and carbendazim 50 WP reduced leaf blast severity significantly to 55.33 per cent as compared to 64.67 per cent in BTH 50 WG and 80.33 per cent in carbendazim 50 WP when used individually. There was significant reduction in leaf blast severity due to the combination of kasugamycin 2L, propiconazole 25 EC and mancozeb 75 WP with carbendazim 50 WP (Fig. 34).

The combination of edifenphos 50 EC with kasugamycin 2L or propiconazole 25 EC and mancozeb 75 WP reduced the minimum inhibitory concentration from 250 µg/ml to 100, 100 and 50 µg/ml, respectively (Table 37). The same trend was observed in ED_{50} values of both mycelial growth and spore germination. However, there was no change in the MIC values of kasugamycin 2L, propiconazole 25

EC and mancozeb 75 WP by virtue of their combination with edifenphos. On the other hand, tricyclazole 75 WP and BTH 50 WG inhibited both mycelial growth and spore germination in combination with edifenphos 50 EC *in vitro* (Fig. 35). But, there was no significant reduction in leaf blast severity due to combination of tricyclazole 75 WP and edifenphos 50 EC, while the mixture of BTH 50 WG and edifenphos 50 EC reduced leaf blast severity significantly to 51 per cent as compared to 64 per cent in BTH 50 WG and 66.33 per cent in edifenphos 50 EC when used individually. The reduction in leaf blast severity was significant due to the combination of kasugamycin 2L, propiconazole 25 EC and mancozeb 75 WP with edifenphos 50 EC (Fig. 35).

There was greater similarity between edifenphos and iprobenfos where the combination of iprobenfos 48 EC with kasugamycin 2L or propiconazole 25 EC and mancozeb 75 WP also reduced the minimum inhibitory concentration from 250 µg/ml to 100, 100 and 50 µg/ml, respectively. The same trend was observed in ED_{50} values of both mycelial growth and spore germination also. There was no change in the MIC values of kasugamycin 2L, propiconazole 25 EC and mancozeb 75 WP by virtue of their combination with iprobenfos. On the other hand, tricyclazole 75 WP and BTH 50 WG inhibited both mycelial growth and spore germination in combination with iprobenfos *in vitro* (Fig. 36). But, there was no significant reduction in leaf blast severity due to combination of tricyclazole 75 WP and iprobenfos 48 EC, while the mixture of BTH 50 WG and iprobenfos 48 EC reduced leaf blast severity significantly to 47.76 per cent as compared to 61 per cent in BTH 50 WG and 62.67 per cent in iprobenfos 48 EC when used individually. The reduction in leaf blast severity was significant due to the combination of kasugamycin 2L, propiconazole 25 EC and mancozeb 75 WP with iprobenfos 48 EC (Fig. 36).

Fungicide mixtures were used by several scientists in different crops against various pathogens as an effective strategy to combat fungicidal resistance. Use of mixture of metalaxyl with mancozeb against *Phytophthora infestans* on potato in UK compared to metalaxyl alone in Netherlands and Ireland serve as an ideal example for use of combinations to avoid build up of resistance (Staub and Sozzi, 1983, 1984; Davidse and Dewaard, 1984). Tivoli *et al.* (1986) reported that thiabendazole in combination with imazalil was found to reduce the infection caused by thiabendazole resistant strains of *Fusarium roseum* var. *sambercinum*. *Phytophthora infestans* and *Pythium aphanidermatum*. Application of mixtures of a phenylamide fungicide with mancozeb or mancozeb plus cymoxanil decreased the build up of phenylamide resistance, compared with phenylamide alone (Sanders *et al.*, 1985; Samoucha and Gisi, 1987; Cohen and Samoucha, 1990).

Huang Xing *et al.* (2005) evaluated the efficacy of tricyclazole, prochloraz and their mixture at 2:1, 4:1, 7:1 and 9:1 against rice blast *in vivo*. The 4:1 mixture of tricyclazole and prochloraz had the best control effect against *P. grisea*. The high control effect was due to inhibitory effect on spore formation and hypal growth with EC_{50} value of 0.238 and 0.346 µg/ml, respectively. The mixture also had control effect on wounded leaves and possessed a good residual performance. Present investigation revealed a paradoxical situation where the fungicides that had given the best *in vitro* performance were not so in the field level control. For instance, the multisite contact fungicide mancozeb was the most effective in inhibiting both mycelial growth and spore germination in the laboratory, but its performance in the field was very poor. Conversely, the melanin biosynthesis inhibitor tricyclazole completely failed to inhibit both mycelial

growth and spore germination *in vitro*, but it was the best among the fungicides tested in the field. The performance of BTH, a host plant defense enhancer was moderate in the field even though it does not inhibit the fungal growth *in vitro*.

In the present investigation, propiconazole in combination with carbendazim, edifenphos and iprobenfos exhibited the optimum level of control both *in vitro* and *in vivo* against carbendazim, edifenphos and iprobenfos resistant isolates, respectively. However, mancozeb with tricyclazole may be the best combination to combat resistance of *P. grisea* given their individual laboratory as well as field performances. The modes of action of these two fungicides are quite complementary with each other and hence they may fit well in the resistance management programme.

SALIENT FEATURES OF THE STUDY:

- Salicylic acid and BTH 50 WG at 10^{-3} M concentration can be used to induce Systemic Acquired Resistance in susceptible rice cultivars if they are agronomically desirable.
- For better results with SAR inducing chemicals in rice, seed treatment may be supplemented with a foliar spray.
- Induction of SAR should be used as part Integrated Disease Management rather than a sole strategy since its efficacy is only about 50 per cent.
- Melanin Biosynthesis Inhibitors *viz.* pyroquilon, tricyclazole and fenoxanil were ineffective in inhibiting both mycelial growth and spore germination of *P. grisea* in the laboratory, but were proved excellent in the field for blast control.

- Seed treatment with pyroquilon 50 WP or tricyclazole 75 WP or azoxystrobin 25 SC or pyraclostrobin 25 EC or RIL 010 25 SC or fenoxanil 20 SC at 2g or 2ml per kg seed can effectively control the blast in the nursery.
- Strobilurin fungicides *viz.* azoxystrobin, pyraclostrobin and RIL 010 can be good substitutes for other blasticides like edifenphos, iprobenfos, isoprothiolane, carbendazim etc. where ever the pathogen developed resistance because their intensive field applications.
- Application of propiconazole 25 EC as foliar spray will be more economical in the areas where sheath blight is also a problem since its effectiveness can be comparable to other blasticides such as edifenphos, iprobenfos, kasugamycin and carbendazim (Propiconazole is being used against sheath blight).
- Use of edifenphos, iprobenfos and isoprothiolane may be discontinued in Mandya, Maruteru and Malan areas due the development of resistance strains in the field and also due to cross resistance.
- Seed treatment followed by two foliar sprays, one at maximum tillering stage and the other at panicle emergence stage using melanin biosynthesis inhibitors and strobilurins in tandem may be a good strategy to control leaf blast and neck blast both in the nursery and main field. However, additional applications may be necessary based on climate and disease progress.
- The combination of mancozeb and tricyclazole could be an effective strategy to combat fungicide resistance due their excellent *in vitro* and *in vivo* efficacies, respectively.

FUTURE LINE OF RESEARCH:

The results of present study indicate the need for future research in the following areas –

- Greater emphasis should be given on the development of blast resistant rice varieties using gene transfer technologies.
- More and more chemicals should be tested to identify their SAR inducing potential with a view to integrate them in the Integrated Disease Management options as these chemicals proved less effective when used alone.
- Resistance monitoring in rice blast fungus should be a continuous process while searching for improved and rapid diagnostic methods to detect and monitor fungicide resistance with special reference to high risk fungicides.
- Appropriate techniques should be developed to study the development of resistance against tricyclazole.
- Research on the development of effective and amicable integrated disease management options.

Summary

Chapter VI

SUMMARY

Different rice cultivars were evaluated for blast disease resistance against *Pyricularia grisea* during the years 2002 and 2003 by adapting Uniform Blast Nursery (UBN) pattern developed by the International Rice Research Institute (IRRI). Forty six cultivars were highly resistant (0-1 score), 32 were resistant (2-3 score), 15 were moderately resistant/susceptible (4-5 score), 19 were susceptible (6-7 score) and 33 were highly susceptible (8-9 score). Among them, a highly resistant cultivars viz. Rasi (0), a moderately resistant/susceptible cultivars viz. Swarnadhan (4) and a highly susceptible cultivar namely HR-12 (9) were selected for further studies on systemic acquired resistance.

An experiment was conducted to know the inhibitory effect of systemic acquired resistance (SAR) inducing chemicals against *P. grisea* in the laboratory. Five such chemicals *viz.* salicylic acid, BTH, sodium selenite, ferric chloride and zinc sulphate were tested at different concentrations of 1 M, 10^{-1} M, 10^{-2} M, 10^{-3} M, 10^{-4} M and 10^{-5} M by employing Inhibition Zone Technique. All the five chemicals formed inhibition zones at 1 M, 10^{-1} M and 10^{-2} M while sodium selenite and ferric chloride formed inhibition zones at 10^{-3} M also. None of the chemicals formed inhibition zones at 10^{-5} M while sodium selenite alone formed inhibition zone at 10^{-4} M. Hence, salicylic acid, BTH and zinc sulphate were used at 10^{-3} M to induce systemic resistance, while ferric chloride and sodium selenite were used at 10^{-4} M and 10^{-5} M, respectively in the glass house experiment.

Green house experiments were conducted to assess the effect of selected SAR inducing chemicals for inducing systemic acquired resistance against blast disease in HR-12, Swarnadhan and Rasi cultivars during *Kharif*, 2002 and *Kharif*, 2003. The per cent seed germination was significantly lower when treated with sodium selenite (10^{-5} M) i.e. 80.46, 84.38 and 80.83 in HR-12, Swarnadhan and Rasi cultivars, respectively (Fig. 5). In the susceptible variety HR-12, BTH 10^{-3} M was superior in minimizing the leaf blast severity followed by salicylic acid 10^{-3} M. The SAR inducing chemicals applied as seed treatment followed by either one foliar spray or two foliar sprays were found significantly superior to those of seed treatment alone.

In the moderately resistant variety Swarnadhan, BTH 10^{-3} M and salicylic acid 10^{-3} M were more successful in reducing the leaf blast severity, while the effect of ferric chloride 10^{-4} M, sodium selenite 10^{-5} M and zinc sulphate 10^{-3} M was found negligible. The SAR inducing capabilities of the chemicals did not differ with the method and number of applications *viz.* seed treatment alone, seed treatment followed by either one or two foliar sprays. In the highly resistant variety Rasi, disease incidence was observed neither in untreated control nor in chemical treatments and hence the SAR inducing capacity can not be assessed in Rasi due to its resistance against blast disease.

Among the fungicides tested, the highest *in vitro* efficacy was shown by those fungicides that affect mitosis and cell division (carbendazim 50 WP) followed by the ones that disrupt respiration (azoxystrobin 25 SC, pyraclostrobin 25 EC and RIL010 25 SC). On the other hand, melanin biosynthesis inhibitors *viz.* pyroquilon 50 WP,

tricyclazole 75 WP and fenoxanil 20 SC have failed to inhibit mycelial growth *in vitro*. The next best control was offered by edifenphos 50 EC, iprobenfos 48 EC and isoprothiolane 40 EC whose mode of action is lipid and membrane synthesis. Sterol biosynthesis inhibitors *viz.* propiconazole 25 EC, epoxiconazole 7.5 EC, difenconazole 25 EC and diniconazole 25 WP were next in the order of their *in vitro* efficacy. The *in vitro* efficacy of the antifungal antibiotic kasugamycin was found as good as DMI-fungicides.

In the seed treatment experiments, pyroquilon 50 WP was found the best fungicide with 6.66 per cent leaf blast severity at 20 days after inoculation (DAI) followed by azoxystrobin 25 SC and pyraclostrobin 25 EC with 7.33 and 8.33 per cent disease severity, respectively when compared to cent per cent disease severity in the untreated control (Fig. 20). The next best protection was offered by RIL010 25 SC, tricyclazole 75 WP and fenoxanil 20 SC with 11.16, 14.16 and 24.99 per cent disease severity, respectively. Carbendazim 50 WP and propiconazole 25 EC were comparatively good with 32.49 and 39.99 per cent disease severity, respectively.

In the nursery experiments where the fungicides were used as foliar sprays, all the fungicides tested were found significantly superior to untreated control upto 20 days after inoculation. The efficacy of pyroquilon 50 WP, azoxystrobin 25 SC, RIL 010 25 SC, pyraclostrobin 25 EC and tricyclazole 75 WP was excellent. Fenoxanil 20 EC, iprobenfos 48 EC, isoprothiolane 40 EC, edifenphos 50 EC, propiconazole 25 EC and kasugamycin 2 L were also found superior in controlling leaf blast incidence. Carbendazim 50 WP exhibited good amount of control compared to rest of the fungicides. Diniconazole 25 WP was the least effective of all the fungicides tested even though it was significantly superior over control.

In the main field, the supremacy of pyroquilon 50 WP was inadvertently continued in controlling both leaf blast severity and neck blast incidence. The same trend was also continued in increasing the total grain yield while reducing the quantity of ill-filled grain. The performance of tricyclazole 75 WP and fenoxanil 20 SC was next only to azoxystrobin 25 SC, pyraclostrobin 25 EC and RIL 010 25 SC. There were 2.66 per cent of lesioned necks with out any broken ones in the plots treated with tricyclazole 75 WP while fenoxanil 20 SC produced 4 per cent lesioned necks and 1.66 per cent broken necks. The highest grain yield of 2.9 t/ha was obtained from the plots treated with pyroquilon 50 WP as against 1.8 t/ha from the untreated control plots.

Rice blast samples were collected from 12 different parts of India including Andhra Pradesh. For carbendazim, Cuttak isolate recorded the highest ED_{50} values of 54.63 and 56.18 µg/ml for mycelial growth (poisoned food) and spore germination, respectively. Medchel isolate recorded the lowest ED_{50} values of 0.39 and 0.55 µg/ml for inhibition of mycelial growth (poisoned food) and spore germination, respectively. For edifenphos, Mandya isolate recorded the highest ED_{50} values of 30.56 and 32.35 µg/ml for mycelial growth (poisoned food) and spore germination, respectively. Pondicherry isolate recorded the lowest ED_{50} values of 0.73 and 0.77 µg/ml for mycelial growth and spore germination, respectively. For iprobenfos, Maruteru isolate recorded the highest ED_{50} values of 31.85 and 32.57 µg/ml for mycelial growth and spore germination, respectively. For iprobenfos also, Pondicherry isolate recorded the lowest ED_{50} values of 0.66 and 0.68 µg/ml for mycelial growth and spore germination, respectively.

Development of resistance towards edifenphos has contributed to the development of positive cross resistance not only to iprobenfos but also to isoprothiolane. Development of resistance to either edifenphos or iprobenfos had no correlation with the fungicides *viz.* pyraclostrobin, kasugamycin and carbendazim. Similarly, development of resistance towards carbendazim had no correlation with the fungicides *viz.* pyraclostrobin and kasugamycin. The carbendazim resistant isolate had shown partial resistance towards iprobenfos, isoprothiolane and edifenphos. Kasugamycin inhibited the mycelial growth completely at 100 µg/ml while the MIC for spore germination was 250 µg/ml which indicates kasugamycin is a good inhibitor of mycelial growth as compared to spore germination.

In the resistance management studies, tricyclazole 75 WP and BTH 50 WG did not show any inhibitory effect on the fungus *in vitro* individually, while mancozeb 75 WP showed the highest inhibition of both mycelial growth and spore germination. But, tricyclazole 75 WP recorded the lowest blast severity of 20.67 per cent as compared to 66.67 per cent in mancozeb 75 WP and 64.67 per cent in BTH 50 WG. There was greater similarity between edifenphos and iprobenfos where the combination of iprobenfos 48 EC or edifenphos 50 EC with kasugamycin 2L or propiconazole 25 EC and mancozeb 75 WP reduced the minimum inhibitory concentration from 250 µg/ml to 100, 100 and 50 µg/ml, respectively.

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