CHARACTERISATION OF RESISTANCE TO POWDERY MILDEW (*Erysiphe graminis* f. sp. *tritici* E. Marchal) IN SOME INDIAN AND EXOTIC WHEATS

THESIS

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TO
MY ADORABLE
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CERTIFICATE I

Certified that the thesis entitled, "Characterization of resistance to powdery mildew (*Erysiphe graminis* f.sp.*tritici* E. Marchal) in some Indian and exotic wheats" submitted in partial fulfilment of the requirements for the award of the degree of Master of Science in Plant Pathology of Himachal Pradesh Krishi Vishvavidyalaya, Palampur, is the bonafide research work carried out by Mr. Rax Paul son of Sh. Kura Ram under my supervision and that no part of this thesis has been submitted for any other degree.

The assistance and help received during the course of this investigation have been fully acknowledged.

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CERTIFICATE II

This is to certify that the thesis entitled, "Characterisation of resistance to powdery mildew (*Erysiphe graminis* f.sp. *tritici* E. Marchal) in some Indian and exotic wheats" submitted by **Sh. Rax Paul** (Admission No. A91-30-18) in partial fulfilment of the requirements for the degree of **Master of Science (Agriculture)** in the subject **Plant Pathology** has been approved by the Examining Committee after an Oral Examination of the same in collaboration with the External Examiner.

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CONTENTS

Chapter No.	Title	Page
1	INTRODUCTION	1 - 4
2	REVIEW OF LITERATURE	5 - 24
3	MATERIALS AND METHODS	25 - 45
4	EXPERIMENTAL RESULTS	46 - 71
5	DISCUSSION	72 - 93
6	SUMMARY	94 - 97
	REFERENCES	i - xii

LIST OF TABLES

Table No.	Title	Page
1	Isolates of Erysiphe graminis f.sp. tritici collected from different agroclimatic zones of Himachal Pradesh.	26
2	Near-isogenic lines/cultivars of wheat used for characterisation of Erysiphe graminis f.sp. tritici isolates.	29
3	Infection types used to characterize reaction types.	31
4	List of cultivars/lines used in the present study and their pedigree.	34-40
5	Avirulence/virulence formuale of the pathotypes of Erysiphe graminis f.sp.tritici used for postulation of resistance genes in 140 selected wheat stocks.	41
6	Virulence pattern of conidial isolates of Erysiphe graminis f.sp. tritici on differential lines having known powdery mildew resistance genes.	47
7	Virulence pattern of ascosporic isolates of Erysiphe graminis f.sp. tritici on differential lines.	48
8	Relationship of virulence with frequency of occurrence of different pathotypes of Erysiphe graminis f.sp. tritici	50
9	Virulence frequency (%) of Erysiphe graminis f.sp. tritici populations during different dates in 1993 at Palampur.	52
10	Virulence frequency (%) of Erysiphe graminis f.sp. tritici populations at different locations in Himachal Pradesh.	53
11	Actual and theoretical frequency of association of different genes for virulence in Erysiphe graminis f.sp. tritici.	55

Table No.	Title	Page
12	Reaction of 140 wheat cvs/genotypes to eight pathotypes of Erysiphe graminis f.sp. tritici and their groupings.	57-58
13	Probable powdery mildew resistance genes in 140 genetic stocks classified into XII groups.	59
14	Conidial germination, germ tube elongation, appressorial formation, elongated secondary hyphae production and haustorial development by Erysiphe graminis f.sp. tritici pathotype 4 on wheat genotypes.	68
15	Incubation period, latent period, colony number/cm^2, colony size, sporulation index and sporulation capacity of Erysiphe graminis f.sp. tritici pathotype 4 on primary leaves of susceptible and resistant wheat genotypes.	70

LIST OF PLATES

PLATE I — Infection process of *Erysiphe graminis* f.sp. *tritici* pathotype 4 on cultivar Agra local.

PLATE II — Infection process of *Erysiphe graminis* f.sp. *tritici* pathotype 4 on genotypes Sappo and Kronjuwel.

PLATE III — Infection process of *Erysiphe graminis* f.sp. *tritici* pathotype 4 on genotype TP 114.

INTRODUCTION

1. INTRODUCTION

The genetic potential of wheat has been realized to a great extent and further increase in the production and stability of yield mainly depends upon the adoption of improved cultivation technology and plugging of various losses caused by weeds, insect pests and diseases. Among all these constraints, diseases take a fairly heavy toll annually, and can lead to famine conditions in epidemic year (Joshi et al., 1976).

Among various diseases, powdery mildew caused by Erysiphe graminis f.sp. tritici E. Marchal (Egt) ranks third in many parts of the world and leads to considerable losses (Goel et al.,,1986). Sometime 40-50 percent reduction in potential grain harvest have been reported (Wiese, 1977). The disease was shown to cause 12%, 27% and 24% losses in mid west, east and south east U.S.A. on recently developed cultivars, some of which possess known genes for powdery mildew resistance (Leath and Heun, 1990). In U.K. powdery mildew results in loss of 0.23 million tonnes of grains annually (Cook et al.,,1991). Similarly, heavy losses due to this disease have been reported from Canada, Newzealand and several other European countries (Moseman et al.,,1984). Earlier powdery mildew was considered as a disease of areas with high rainfall and semicontinental climate, but with changing crop practices it has attained economic importance in drier areas of the world also (Bennett, 1984). In India,

the disease is limited to the northern and southern hills and sporadic epidemics have also been reported from the plains (Goel et al., 1986). However, with the cultivation of high yielding dwarf varieties under high fertility conditions, incidence of the disease has increased partially in foot hills and valley areas of north India. It is envisaged that with the extension of irrigation facilities and use of excessive doses of fertilizers particularly nitrogen under intensive cultivation, yield losses due to this disease will increase in future.

The disease can be easily and effectively managed without much cost with the cultivation of resistant cultivars (Bennett, 1984). This requires identification and characterisation of resistant sources against the prevailing virulences of the pathogen, and their exploitation in breeding programme for the development of such resistant cultivars (Moseman et al., 1984). This approach has been followed in many European, American and Australian countries to combat the disease (Bennett, 1984). However, in India the main work has been done to identify resistant sources (Upadhyay et al., 1972; Kochumadhavan et al., 1985 and Basandrai & Sharma, 1990) and a few scattered reports are available on pathogenic variation (Goel et al., 1986; Sharma et al., 1989; Sharma and Singh, 1990c; Sharma et al., 1990 and Kanwar, 1993). The virulence spectrum of the pathogen is reported to change very frequently (Wolfe and Schwarzbach,

1978; Bennett, 1984) and the resistant varieties become susceptible after a few years of their commercial cultivation. Hence, information on the presence and frequency of virulence genes in prevalent pathogen populations is necessary to replace ineffective resistance genes with genes that are currently useful and to improve the durability of resistance.

Identified resistance sources must be characterized in terms of diversity of genes for resistance for their proper exploitation in the breeding programme. The infection-type matching technique makes it possible to propose hypothetical genotypes of a set of cultivars, if their reaction to a set of pathogen cultures of specific virulence pattern is known (Heun and Fischbeck, 1987a, b). It provides evidence for the diversity of resistance and postulation of resistance genes, facilitating their exploitation in breeding programme.

The information pertaining to the components of resistance will help in better understanding of the potential resistance barriers, operating in the genotypes against the pathogen. The potential barriers of race-specific resistance based on conidial germination, elongated secondary hyphae formation, epidermal and mesophyll cell hypersensitivity and sporulation have been reported in near-isogenic lines in the background of Chancellor having genes Pm1 through Pm4,

an unidentified gene Pm(Ma) derived from cv. Michigan Amber, pm5 in the background of cv. Chinese Spring and in several Triticum timopheevii and T. arariticum lines (Hyde and Colhoun, 1975; Nashaat and Moore, 1991). Uptill now genes upto Pm17 have been identified, however, no information is available on components of resistance in these cvs. Keeping the above points in view, the present investigations were undertaken with the following objectives:

1. To work out the virulence pattern of the populations of E. graminis f. sp. tritici from different agro-climatic regions of Himachal Pradesh

2. To identify race-specific powdery mildew resistance genes in some Indian and exotic wheats

3. To study the components of resistance in selected genotypes against specific cultures of the pathogen.

REVIEW OF LITERATURE

2. REVIEW OF LITERATURE

2.1 Physiological Specialization: Erysiphe graminis f.sp. tritici (Egt), causal organism of wheat powdery mildew, is a highly variable fungus. The pioneer work on pathogenic variation dates back to 1930 and 1933, when Waterhouse and Mains, reported races in Australian and American wheat powdery mildew populations. Thereafter, a lot of work was carried out in different countries.

Newton and Cherewick (1947) identified three physiological races tentatively numbered as 3,4 and 5 from Canada, using the differential set consisting of Huron, Norka, Axminister and Chul. In south eastern U.S.A., Lowther (1950) detected nine races out of 40 collections, using cvs Axminister, Ulka, Chul, Hope and Normandie as differentials. Power and Sando (1957) reported virulence genes complimentary to the resistance gene Mlt and Mlu. Power et al. (1959) reported a new pathogenic strain 2A attacking cv Asosan.

In Federal Republic of Germany, Rosentiel (1938) differentiated pathogen populations into six distinct physiological races. Schlichtling (1939) reported six races using cvs Dixon, Huron and Illions as differentials. Nover (1957) differentiated 10 physiological races on cvs Salzminde, Stamm 14/44, Red Fern, Axminister, Normandie, Halle Stamm 13471 and Schwazer perscher.

Vallega and Cenoz (1941) differentiated three physiological races on varieties Chul and Sonora in Argentina.

In Scandanavian countries, 22 races were identified out of 366 field samples collected from Sweden, Denmark and Norwey (Leijerstam, 1962). Genotype CI 12633 was resistant to all the races and cvs Salzminde 14/44, Halle Stamm 13471 and Weihenstephom M1 were the useful sources of resistance. Cvs Ulka, Norka, Hope, Chul and Aurora were discarded as differentials.

Wolfe (1965) identified 13 races based on a set of nine genotypes in north western Europe, using detached leaf technique on benzimidazole solution. Further using the same set, 15 races were reported (Wolfe, 1967).

In Italy, out of 185 samples, races 15,3,27,16 and 1 were the most widespread (Grasso et al., 1966). Subsequently, during the year 1967, 12 and 18 races were identified out of 36 conidial and 45 cleistothecial samples respectively (Grasso et al., 1967). Ceoloni and Vallega (1978) reported virulence v4 on cvs Khapli, Yuma and Valgerado having gene Pm4.

It is evident from the studies conducted in different countries that wheat powdery mildew populations are pathogenically variable. Due to use of variable types

of differentials by different workers in several countries, the exact nature of comparable race flora cannot be ascertained McIntosh and Baker (1966) emphasized the need for development of a uniform differential set. Briggle (1969) established near-isogenic lines, having single powdery mildew resistance genes in the background of cv Chancellor which were accepted as differential set.

In Hungary, the work on physiological specialization was done by Szunics and associates. Szunics (1969) reported that among 17 races, races 16 (22.98%), 18 (14.76%) and 19 (13.12%) were most predominent and race 34 was most virulent Commonly grown cv Benzostaya 1 was susceptible to all the races. Subsequently, races 3,9,0 were most frequent on wheat, and a new race "X" was most aggressive (Szunics and Szunics, 1972). Races from Kavkaz and Aurora were more aggressive (Szunics and Szunics, 1974). Among 56 races identified out of 1717 Egt cultures, isolated during 1970-71 to 1982-83, races 46,47,48,52,53,66,80 and 81 were recorded for the first time. Most of the races were avirulent on genes Pm4 and Pm2+Mld, whereas, cvs Kavkaz and Aurora (Pm8) lost their resistance to new virulences (Szunics and Szunics, 1984). During 1987, 14 new races were identified (Szunics and Szunics, 1987).

Klein et al. (1976) reported races 2,3,4,7,15,17 and 18 of wheat mildew and Haller et al. (1980) reported races 2,3,4,8,15,17,25,27 and 53 as the most frequent races in German Democratic Republic.

In Czechoslovakia, Mraz (1972) identified 10 and 12 races in 1965 and 1966, respectively. Races 18 and 33 were most predominent during both years. Races 13 and 29 were identified during the year 1969 and race 23 in 1970. Race 3 was most frequent followed by 18, 0,4,21,34 and 15 (Mraz,1972) and three more races N_1, N_2, N_3 were recorded subsequently (Mraz, 1980).

In Russia, races 14 and 16 were the most widespread out of five races identified during 1969 (Rigna & Krivchenko,1971), whereas during 1972 races 14 and 16 were most widespread (Rodigina & Alita, 1972). Out of 8 races identified in Ukranine, races 52,50 and 18 were predominent (Kol' Nobritskii, 1980). In Slovakia, races 4,2,52,57,18, 46 and 32 were most widespread (Solc and Paulech, 1977), and among 47 races of Egt identified during the years 1979-81, races 2,4,9,26,27,35,44,50 were predominent and races 33,43,46,47,51, X and X_2 were highly virulent (Gromáshevs'ka, 1983). Further, (Koller, 1984) reported that among the 16 races identified, races 4 and 18 were the predominent.

In Poland, out of 14 races detected during 1968-69, race 3 was widespread, (Blaszczak, 1973). Out of 40 races identified among 789 isolates during 1973-75, races 15 and 4 were most frequent during the years 1974 and 1975,respectively, whereas, races 4,13,27 and 96 were most virulent (Blaszczak et al., 1976). According to Woznick (1974), out of 19 races identified during 1971-72, race 46 was most predominent.

During the years 1976-77 and 1978-79, out of 29 races identified, races 4,27,35,46,52,53 and 56 occured in all seasons and race 46 was the most common and races 59, 35 and 46 were the widespread. Cv. Halle Stamm 13471 was resistant to all the races except 51 (Wozniak and Strezembicka, 1982).

Kwiatkowski (1989) reported that out of 50 races identified during 1982-86, races 2,4,27,46,57 and 67 were most frequent, and 36,38,73 and 82 were the new records. All the races were categorized into 7 virulence groups. Lines with genes Pm4b, Pm2, Pm6, Mld and Mlha were the most effective.

Out of 31 races of Egt identified in Bonsia and Herzegovina states in Yugoslavia races 13 and 16 were predominent and race 41 was most virulent (Spenhar and Vlahoric, 1978). Koric (1984) reported that out of 18 races identified on wheat,race 75 was most dominant, whereas, race 64 and isolates F and G were new records. Stojanovic et al. (1990) identified 70 physiologic races of Egt in Yugoslavia.

In Japan,OKu et al. (1987) identified 20 races among field isolates collected during 1962, 1967 and 1984-1986, using cvs. Axminister X Cc8, Ulka X Cc8, Asosan X Cc8, Chul X Cc8, Khapli X Cc8, CS/Hope and Sapporoharukomogi as differentials.

Wu et al. (1983) grouped 151 strains of Egt, collected from Jiangsu province in China, into 16 physiologic races based on their pathogenic differences on eight differential

hosts. All races were virulent on cultivars grown in Kweichew in China. In India, scanty and localized information is available about race spectrum of Egt. Arya (1962) reported three races (3,4 and X) from ten isolates collected from Jodhpur, using differential set proposed by Mains (1933). Prabhu and Prasada (1963) indicated the prevalence of different races of Egt in Shimla and Wellington based on the variable reaction on seven wheat genotypes, however, identity of the races was not established.

Singh and Sood (1977) grouped eight Egt isolates collected from Kangra valley in Himachal Pradesh in two categories defined as culture 1 & 2 based on their reaction on near-isogenic lines (Pm1-Pm4). One culture was virulent on genes Pm3b and Pm3c (culture 1) and the other on genes Pm3b, Pm3c and Pm4 (culture 2).

Sharma et al. (1990) classified 20 single spore isolates of Egt collected from Punjab and Himachal Pradesh into nine virulence combinations based on their reaction on near-isolgenic-lines in the background of cv Chancellor having genes Pm1 - Pm4 and Pm(Ma), and genotypes possessing genes Pm5, Pm6, Pm7 and Pm8. All the pathotypes were virulent on genes Pm3c pm5 and Pm(Ma) and avirulent on genes Pm1 and Pm4a.

Sharma and Singh (1990c) identified 14 and 5 races from 37 conidial,12 ascosporic isolates, respectively using the differential set of Sharma et al. (1990), excluding line having gene pm5. Most virulent races 3,12, and 13 were distributed

were more virulent than races from ascospores. Races with very narrow and high virulence were either non-existant or rare.

Using the same differential set, Kanwar (1993) identified 26 and 14 pathotypes among 63 conidial and 21 ascosporic isolates collected from different agroclimatic areas of Himachal Pradesh. Pathotype 23, with virulence on eight powdery mildew resistant genes was most virulent and pathotype 11 with virulence on Pm(Ma) only was least virulent, but most prevalent pathotype. Gene Pm1 was effective against all pathotypes, whereas virulence on genes Pm2 and Pm6 was rare.

2.2 Virulence Structure of E. graminis f.sp. tritici (Egt) Populations:

Wolfe and Schwarzbach (1975) emphasised that instead of identifying a physiologic race based on the reaction of an isolate to a set of differential varieties, relative frequency of virulence genes in pathogen population in a particular region towards known powdery mildew resistance genes should be monitored. In many countries virulences expressed in relation to host resistance genes have been reported.

Ceoloni and Vallega (1978) reported virulence (v_4), in an Italian isolate of E. graminis tritici on genotypes having resistance gene Pm4. Negulexu et al. (1979) reported that out of seven resistance genes, Pm6 was effective and Pm4 partially effective in Romania.

Royer et al. (1984) distinguished single colony isolates of Egt collected from Central Peinsylvania (USA) during 1976-78

and 1980, on 9 near-isogenic lines in background of cv Chancellor having known powdery mildew resistance gene (Pmx). Virulence was rare on lines having genes Pm1, Pm3a and Pm3b. Associated virulence (P) for genes (Pm2, Pm5), (Pm3c, Pm5) and (Pm3c, PMA) was quite common and genotype P_2, P_5 was most prevalent during 1976-78, whereas, in 1980 virulences P 3c and P MA occured in combination with genotype P_2, P_5 resulting in increased racial complexity. In North Carolina, virulences were detected against all the known powdery mildew genes i.e. Pm1, Pm3, Pm2(+), Pm3a, Pm3b, Pm3c, Pm4, Pm6, Pm7, Pm8 and Michigan Amber using mobile nursery technique (Leath and Murphy, 1985).

A mobile nursery comprising of 11 near-isogenic lines having known single genes for resistance (Pm1, Pm2, Pm3a, Pm3b, Pm3c, Pm4 and pm5) was exposed to the field inoculum in south Ontario, Canada. Virulence pattern of wheat powdery mildew populations was different in various regions (Bailey and McNeill, 1985).

The survey of 10 regions in south Ontario, Canada during the years 1983, 1984 and 1985 depicted general virulence formula (effective/ineffective host genes) as /Ma, 1,2,3a, 3b, 3c, 4 ,5. Gene Pm3a was effective at most of sites over three year period. Single pustule isolates provided better estimate of the occurence of genes than collections of mass cultures (Menzies and MacNeill,1986). Virulence frequency of Egt for the years 1986 and 1987 in six regions of S. Ontario, was expressed as virulence formula (effective/ineffective genes) MA/1,2,3a,3b,4,

8. The race composition was stable over the survey period. Genes Pm3a and Pm3b were effective at more than 50% of sites in both years of survey. Virulence for genes Pm(Ma)+Pm4, Pm(MA)+Pm3c, and Pm2 + Pm5 was found associated.

In Jiangsu state of China, Wu and Liu (1983) observed that genes Pm2 and Pm4 were very effective against the local Egt populations. Lu (1986) analysed virulence frequencies of Egt in 5 locations in Jiangsu province by inoculations of wheat leaf segments and using a mobile nursery. Virulence genes were detected against 21 differential cvs. The virulence frequency of genes v_1, v_{3a}, v_{3b}, v_{3c}, v_1+v_2, v_5, vsumai 3 and v-78194-2-1 were very high (av 75%) and adult plants with corresponding resistant genes were susceptible in the field. Whereas, virulence frequencies of genes v_2, v_4, v_{775-1}, c v-Ning 8026, v-783-4684, v-Lovring 231, v-Xingmai 9 and v-Alondra "S" were low (av 1-20%) and the adult plants were resistant. Virulence of genes v_{2+4b}, v VPM9, v AH 688 $F_7$8N were very low (av. $\angle$0.2%) and the adult plants were immune. Out of 67 isolates of E. graminis tritici, collected in Sichuan province (China), cultivars carrying frequency of virulence genes v_1 and v_5 was 90%, whereas, the frequency of v_8, v_{2x}, v_{Ba} and v_{kg} were 65.7, 7.5, 4.5 and 16.4%, respectively. Cultivars Baimain 3 (Pm2x), 81-7241 (Ba) and KenGui-A (KG) remained highly resistant (Li and Huang, 1991). Of the 64 strains of E. graminis, collected from autumn sown commercial wheat cultivars and varieties with known powdery mildew resistance genes, 59.38-87.5% of isolates were virulent

on Axminister x Cc^8, Asosan x Cc^8, Chul x Cc^8, Sonora x Cc^8 and Hope, 6.25-18.75% strains were virulent for Baimain 3, Kenguia 1, CI 12632 and Maris Huntsman and virulence frequency for Ulka x Cc^8, Luo fulin 10 and Gaojiasno was 32.81, 45.31 and 39.06%, respectively. Most of 18 commercial cultivars were susceptible. Race 0 was the most frequent (15.61%) followed by race 5 (12.5%) (Wang et al., 1990). Li and Huang (1992) reported that out of 85 isolates of E. graminis tritici collected from 11 wheat cvs and isogenic-lines from Sichuan province in China, virulence frequency of genes v_1, v_{3b}, v_{3c} and v_5 was highest (90%) followed by v_{3a}, v_7, v_8, v_2 and v_6. Combinations of v_2+ v_6, v_2+v_{3a}, v_2 + v_{4b}, v_{4b} + v_6 and v_{4a}+v_8 were more frequent indicating that wheat cultivars containing corresponding resistance genes were highly susceptible. In all 37.6 and 47.1% strains were virulent on 7 and 8 genes, respectively.

According to Lesovoi and Kol,Nlobritskii (1980) genes Pm1, Pm2, Pm3a, Pm3b, Pm3c, Pm4, pm5, M1r were not effective against the predominant races in Ukraine. Some of the races viz. 35,52,16 were highly virulent on these genes. Gene Pm4 was highly resistant gene, followed by genes Pm2+Pm7 and Pm2+Pm6.

In Switzerland, Streckeisen and Fried (1985) detected 43 pathotypes out of 162 isolates of E. graminis using a differential set, consisting of 24 genotypes of United Kingdom and German origin. No isolate could overcome the resistance of CWW 1645/5 and line 623/65 was resistant to 86.4% isolates.

Resistance of Sappo (Pm2+Pm4b) and Walter (Pm2+Pm6) was overcome for the first time.

Limpert et al. (1987) observed regional differences in virulence frequencies of single colony progenies of Egt using wheat differentials with powdery mildew resistance genes Pm2, Pm4b, Pm8, Mli, Pm2+Pm6, Pm4b+Pm8 and Pm2+Pm4b+Pm8. Virulence frequency on genes Pm2, Pm2+Pm6 and Pm4b was higher in England and it decreased to east whereas reverse was true for gene Pm8. Virulence was generally high in Mli and to the genotypes combined it was low.

Hovmoller (1987) worked out virulence structure of wheat mildew population in north west Europe using mobile nurseries. Commercial wheat cvs Karka (Mli), Disponent (Pm8) and Longbow (Pm2) were susceptible to 50% isolates and Sleipner (Pm2+Pm8+?) was highly resistant.

Stojanovic and Ponos (1990) analysed 375 isolates of the Erysiphe graminis f.sp. tritici, collected from different wheat varieties in 76 localities in South Eastern Yugoslavia, on isogenic lines having genes Pm1, Pm2, Pm3a, Pm3b, Pm3c and Pm4a. In all, 38 virulence gene combinations were detected and with majority of isolates had 3-4 virulence genes. Isolates with virulence formulae 2,3b, 4a/1,3a,3c; 1,2,3b,4a/3a,3c and 1,3b,4b/2,3a,8 were most common.

Stojanovic et al.(1991) worked out virulence pattern of 735 isolates during the year 1988-1989 in Serbia (Yugoslavia). Virulence genes 1,2,3a,5,8/4a and 1,2,3a,3c,5,6,8/3b; 5,6,8/3b,4a were dominent in 1988 and 1989,

respectively. Allele v-3b and v-4a were least frequent. Most pathotypes had 5-8 virulent genes.

In India, Upadhyay and Kumar (1974) evaluated near-isogenic powdery mildew resistant lines having genes Pm1 through Pm4 at Shimla, Dhaulakuan and Wellington. Gene Pm2 (CI 12632 x Cc^8 and Ulka x Cc^8) was resistant at all the locations. In north India, strains were avirulent on genes Pm3a, Pm3b and Pm3c whereas in Nilgiri hills on Pm1, Pm3b and Pm4.

Saharan et al. (1981) found that all the collections of Egt from Himachal Pradesh were virulent on resistant genes Pm3a and Pm3c. Virulence was abundant on gene Pm2 but was absent on genes Pm1 and Pm4. Sharma and Singh (1990d) determined the virulence structure of E. graminis f.sp.tritici populations during 1986 in different areas of Himachal Pradesh with the help of mobile nurseries. Virulence was most common on genes Pm(Ma)+Pm7 whereas virulence on gene Pm1 was rare and no virulence was trapped on genes Pm2, Pm4 and Pm6.

Kanwar (1993) determined virulence structure of Egt populations in Himachal Pradesh during the year 1991 and 1992. Virulence was quite frequent on genes Pm3a, Pm3c, Pm5, Pm7, Pm8 and Pm(Ma) and no virulence was detected on gene Pm4a. Virulence was rare on genes Pm1, Pm2 and Pm6. Combination of gene Pm1 with Pm2, Pm3a, Pm3b, Pm3c, Pm4a, Pm5, Pm6, Pm7, Pm8 and Pm(Ma) and genes Pm2 with Pm4a, Pm2 with Pm6, Pm2 with Pm8, Pm4a with Pm8, Pm6 with Pm8, Pm7 with Pm8 were expected to be effective against prevailing virulences in the State.

2.3 Characterisation of Resistance:

Gene-for-gene concept (Flor, 1942) makes it possible to propose hypothetical genotypes of a set of cultivars if their infection pattern to a set of pathogen cultures with known genes for virulence is available (Person 1959; Browder 1973). The technique is known as infection-type matching technique and has been used by a number of workers in wheat-*E. graminis tritici* system (Heun and Fischbeck, 1987a, 1987b; Hovmöller, 1989; Leath and Heun, 1990).

Pugsley and Carter (1953), without knowing the concept of infection-type-matching technique, grouped 12 wheat varieties into 3 groups based on their reaction with races P and P-1 of *E. graminis* f.sp. *tritici*. Pugsley (1961) used this technique to identify six different powdery mildew resistant genes, designated as *Mla*, *Mlb*, *Mle*, *Mls*, *Mlt* and *Mlu* in wheat.

Wolfe (1965) grouped 26 wheat lines into 10 different groups, which formed the basis for the identification of races of powdery mildew in United Kingdom and several other European countries.

Based on the infection-type matching technique, Sebastian *et al.* (1983) attributed the resistance of lines IL-72-2919-1 and Va-66-54-10 to gene *Pm3a*. Moseman *et al.* (1984) evaluated powdery mildew resistance in 233 accession of *T. dicoccoides* based on their reaction to four Egt cultures having virulences corresponding to most of the known resistance genes.

Heun and Fischbeck (1987a) identified race specific powdery mildew resistance genes in 23 spring wheat varieties

and lines using 19 isolates having diverse virulence pattern. Most of the varieties carried single resistance gene whereas, resistance in five and three varieties was found to be governed by combinations of two and three genes, respectively.

Heun and Fischbeck (1987b) postulated powdery mildew resistance genes in 59 winter wheat cultivars using 11 isolates of E. graminis tritici. Twenty seven genotypes were susceptible to all isolates and remaining 32 genotypeswere categorised into ten groups involving 6 of the 12 major genes analysed. The largest group of 16 cultivars had gene Mli, and 4 other cultivars had Mli combined with other resistance genes. Resistance of varieties Kronjuwel and two breeding lines was controlled by gene Pm4b. Resistance pattern of Mli and Pm5 were similar and it is suggested that the genes are identical.

Leath and Heun (1990) postulated powdery mildew resistance genes in 22 softred winter wheats based on gene-for-gene concept and pedigree analysis. In some stocks resistance was attributed to genes Pm3a, pm5, and Pm6, whereas, other cultivars carried no known resistance gene.

Lutz et al. (1992) specified powdery mildew resistance genes in 24 Czechoslovakian wheats. In most of the winter wheats genes Pm4b, pm5, Pm8 were responsible for resistance, and in few cultivars resistance was attributed to combination of two or three genes. Spring wheat cultivars lacked major genes for resistance. Gene Mlk was responsible for resistance in one variety whereas in two varieties the resistance was governed by

gene Mlk in combination with gene pm5. In variety Sylva, genes Pm1, Pm5 and an unidentified gene was responsible for the resistance.

In India, very limited work has been done on this aspect. Sharma et al. (1993) postulated powdery mildew resistance genes in 75 wheat stocks using nine diverse cultures of E. graminis tritici. In most of the stocks resistance was governed by genes Pm3a, Pm3b and some other unidentified gene(s) Basandrai et al.(1991) postulated powdery mildew resistance genesin 26 Indian and exotic wheats, using five cultures with diverse virulence pattern.

Kanwar (1993) postulated powdery mildew resistance genes in 65 wheat genotypes based on their differential reaction to six cultures of Egt with diverse virulence pattern. In more than 60% genotypes, gene Pm8 individually or in combination with some other gene(s) was found to be responsible for resistance. Presence of gene Pm8 was further confirmed by postulation of leaf rust resistance gene Lr 26 which is closely linked with gene Pm8.

3.4 Components of Resistance:

Components of race-specific incompatible reaction to Erysiphe graminis f.sp. tritici (Egt) have been widely investigated in wheat (Hyde and Colhoun 1975; Nashaat and Moore, 1991). In general, resistance was attributed to the morphological characters of the host i.e. number of surface hairs, cutical thickness, papilla deposition epidermal cell wall thickness and inability of fungus to penetrate host cells, haustorial abortion

or restricted haustorial development in host epidermal cell, necrosis of tissues surrounding or underlying infected cell or cells and suppression of sporulation.

According to Ghemawat (1969) adult plant resistance in five winter wheat cvs susceptible to Egt race 50 was expressed at three stages i.e. inhibition in penetration of primary germ tube and formation of papilla, abortion of haustoria in apparently normal host cells and hypersensitive reaction. Accordingly, resistance in variety 5772 was attributed to 73% less primary germ tube penetration, 5% abortion of haustoria and 11% hypersensitive reaction whereas in susceptible cv Vermillion, there was only 3.6% less penetration, 0.5% abortion of haustoria and 0.9% hypersensitive reaction.

Hyde and Colhoun (1975) studied infection process of E. graminis tritici in compatible and incompatible race-variety combinations. With the incompatible host-parasite combinations involving host genes Pm4a and Mle resistance expressed as necrosis of cells and abortion of the developing haustorium, was observed shortly after penetration of the host epidermal cell. Incompatible P2/Pm2 (Ulka x Cc^8) reaction was expressed as necrosis of the mesophyll cells underlying the infection court. In P3a/Pm3a (involving line CI 14120 and isolate W9/30) and P3b/Pm3b (involving line Chul x Cc^8 and isolate W71/178) gene combinations, majority of the pathogenic growth was inhibited prior to elongated secondary hyphae formation due to hypersensitive reaction of epidermal cells. The parasitic units which escaped this stage were inhibited due to

necrosis of tissues underlying infection court. Pathogen development in gene combination P3c/Pm3c (involving lines CI 14122 and TP 160/1-3 and isolate W9/30) was restricted due to formation of low number of elongated secondary hyphae. Incompatible reaction in Michigan Amber x Cc^{8} to isolate W9/30 expressed as epidermal cell hypersensitivity and necrosis of tissues underlying parasitic unit. In cv CS/Hope and line CS/H7B having gene pm5, sporulation was less.

Wu et al. (1985) attributed variable powdery muldew resistance in 16 wheat varieties to delay in early haustorial development, slow increase in rate of haustorial formation, reduction in number and size of haustoria and rapid distortion and disintigration of haustoria. More haustoria were found in seedlings and young plants than in older plants.

Iliev (1989) attributed adult plant resistance in cultivars Kennya Page, 5517AS-5-IP3 and 77A12PI-1 to the formation of lesser number of secondary hyphae as compared to susceptible check.

Donchev and Iliev (1987) attributed non- specific resistance in genotypes 5517AS-5-IP3 and Sadovoi to slow and late spore germination, occurring as late as 72 hrs after inoculations. Spore germination was highest in Sadovska Ranozreika 4 and lowest in Kenya page, the least and the most resistant varieties, respectively. Most of the germinated conidiospores formed appresoria irrespective of resistance of genotype.

Nashaat and Moore (1991) studied expression of components of powdery mildew resistance, 72 hrs after inoculations in some T. timopheevii and T. araticum lines to investigate efficiency of the sequence of barriers (cutical, papilla, epidermal cell hypersensitivity and hypersensitive necrotic reaction of mesophyll cells underlying infection area) to fungal penetration and development. T. arariticum and T. timopheevii exhibited papilla mediated resistance, whereas, T. arariticum showed epidermal hypersensitivity also. On lines CI 12644 and T. timopheevii conidial production of E. graminis isolate CHI was substantially reduced. It was concluded that T. timopheevii may possess resistance gene(s) in addition to already transferred and expressed in hexaploid derivative CI 12633, particularly, those conditioning the hypersensitive necrotic reaction of mesophyll cells surrounding infection area.

In addition to race-specific hypersensitive resistance, some genotypes may exhibit reduced growth and reproduction of pathogen and such type of resistance has been referred to as rate reducing resistance, which has been reported to be governed by polygenes (Vanderplank, 1963). However, Clifford (1975) suggested that polygenes are archaic major genes, which have lost their large effects through evolution of virulence in pathogen, but exhibit residual or "ghost effect". According to Nelson (1981) race-specific and race non-specific resistance is merely an expression of different actions of same gene/s in different genetic background and or when exposed to different races of pathogen. Such type of quantitative resistance is

detectable in cultivars which either have no identified qualitative resistance or in which qualitative resistance has been matched.

Shaner (1973) considered latent period, number of colonies/area, average colony size, sporulation index as components of slow mildewing resistance. He reported that size and number of conidial chains per unit area of colony were less on cv. Knox than on susceptible cv Vermillion.

According to Martin and Ellingboe (1976), lines with gene Pm4a on inoculation with isolate MS-2, virulent on gene Pm4a exhibited slow growth of the parasite which was expressed as production of less number of infection sites as compared with line having no powdery mildew resistant gene.

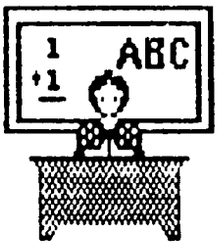
Nass et al. (1981) reported that out of six near-isogenic winter wheat lines, having known powdery mildew resistance genes, lines with genes Pm3c, Pm4 and Pm(Ma) showed potential residual effects against an isolate virulent on these genes, which was expressed as production of less number of sporulating colonies as compared with cv Chancellor. However, no such statistical significant residual effects were obtained for resistance genes Pm2, Pm2+ and pm5.

Nelson et al. (1982) obtained two and four gene heterozygote pyramid from different defeated powdery mildew resistance genes. Two gene pyramids Pm3c x Pm4 significantly reduced number of lesions per leaf, while another pyramid Pm4 x Pm(Ma) reduced sporulation per lesion. Four gene

heterozygote pyramids were more effective in reducing lesion number and sporulation.

Sharma et al. (1991) reported that single powdery mildew resistant genes Pm3a, Pm3b, Pm3c, pm5 and Pm(Ma) and their all possible combinations exhibited residual resistance to E. graminis tritici culture Egt 5 virulent on all these genes. Gene Pm3b showed longer latent period and lines with genes Pm3a, Pm3b, Pm4a, pm5 developed less number of colonies and produced less number of spores as compared to susceptible cultivar Agra Local. All gene combinations developed less number of colonies/area, smaller colonies and less number of spores/colony except in case of combination of gene Pm3c x Pm8 and Pm3a x Pm8 in which sporulation was statistically at par with the susceptible check.

Kanwar (1993) studied components of slow mildewing resistance at the seedling and adult plant stage in eight wheat genotypes, showing field resistance. On all the test cultivars, except CPAN 1796, incubation and latent periods were significantly longer than the susceptible variety WL 711 at both seedling and adult plant stages. Number of colonies/leaf on most cultivars were less as compared to susceptible check WL 711 both in seedling and adult plant stages. Colony size was significantly smaller than in susceptible check WL 711 at both seedling and adult plant stages in all the genotypes. Cultivars Sonalika and HS 240 had least sporulation index in the seedling stage whereas cultivars HPW 42 and CPAN 1796 had least sporulation index in adult plant stage.



MATERIALS AND METHODS

3 MATERIALS AND METHODS

The present investigations were carried out on characterisation of resistance against E. graminis f.sp. tritici (Egt), wheat powdery mildew pathogen, in some Indian and exotic wheats. The experimental material consisted of differential set comprising near-isogenic lines in the background of cv Chancellor, having known powdery mildew resistant genes Pm1 through Pm4 and Pm(Ma) (an unidentified gene from cultivar Michigan Amber), gene pm5 in the background cv Chinese Spring, cvs with genes Pm6 to Pm8, and combination of genes Pm2+4b and Pm4b+8, and 140 genotypes of wheat. In addition, 56 isolates of Egt collected from different agroclimatic zones of Himachal Pradesh were also used for virulence characterisation. The experiments were conducted at HPKV, Palampur both in glass house and laboratory.

3.1 Collection, Purification and Maintainance of Isolates of E. graminis tritici

Conidial isolates of powdery mildew (Egt) were collected from various locations in different agroclimatic zones of Himachal Pradesh (Table 1). Naturally infected leaves, ears or both were brought to the laboratory in the air filled polythene bags. Conidial inoculum from the sporulating colonies was transferred to eight days old seedlings of susceptible cultivar Agra local with the help of a sterilized camel hair brush. In plant material with scanty

Table 1. Isolates of *Erysiphe graminis* f.sp. *tritici* collected from different agroclimatic zones of Himachal Pradesh.

Conidial isolates	Place of collection/zone		Variety
1,2,3	Kukumseri	IV	Sonalika
4,5,6	Kukumseri	IV	Aurora
7,8,9,10	Palampur	II	Sonalika
11,12,13	Palampur	II	VL 421
14,15,16	Palampur	II	Unknown
17,18,19	Maranda	II	Sonalika
20,21,22	Palampur (Neugal)	II	Sonalika
24,25	Palampur	II	WL-711
26,27	Dehra	I	Kalyan Sona
28,29,30,23	Dhaulakuan	I	Sonalika
31,32,33,45	Dhaulakuan	I	Unknown
34,35,36	Jaisinghpur	I	Kalyansona
37,38,44	Paisa	I	Unknown
39,40,41,42	Nehran Pukhar	I	Kalyansona
43	Jawalamukhi	I	VL-616
Ascosporic isolates A-K	Dalang Maidan	IV	Unknown

sporulation, conidial production was intensified by incubating plant parts in air filled moistened polythene bags in growth chambers maintained at 22±2°C under artificial 14 hr day length. Multiplication of collected inoculum was carried out on seedlings of susceptible cultivar Agra local.

3.1.1 Raising the Seedlings of Susceptible Cvs: For inoculum multiplication seedlings of cv Agra local were raised in 4" plastic pot (10-15 seeds/pot) filled with a mixture of natural field soil and FYM under spore proof conditions to avoid contamination. Inoculated seedlings were covered with glass chimneys, the open upper end of which was covered with double layered muslin cloth.

3.1.2 Propagation, Purification and Multiplication of Conidial Isolates: Inoculated susceptible seedlings were kept at room temperature (Max. 20±8°C and min. 12±5°C) for 7-8 days and conidia from well spaced sporulating colonies were picked up with the help of sterilized camel hair brush and transferred to seedlings for further propagation. In some cases, conidia were directly transferred from the well spaced sporulating colonies on naturally infected plant parts. Thus 5-10 isolates from a single sample collected from a particular region, were isolated and multiplied on a susceptible cultivar. The isolates were maintained by transferring the inoculum every 20 days to the seedlings of susceptible cultivar. In total, 45 isolates, thus collected from different areas, were maintained for

further studies.

3.1.3 Studies on Ascosporic Isolates: Ascosporic isolates were derived from the cleistothecia collected from Dalang Maidan (Lahaul & Spiti) during September-October 1992. Cleistothecia were placed with the help of an inoculation needle on watch glasses lined with wet blotter papers. Watch glasses were inverted on the open ends of the glass chimneys enclosing four day old seedlings of susceptible cv Agra local. Cleistothecia were kept moist by continuous supply of moisture, maintained by strips of blotter papers dipped in water. Cleistothecia were examined for ascospore formation, starting from fourth day after incubation. Development of fungal colonies on seedlings was also observed starting seven days after the ascospores formation. Fungal colonies were individually transferred for mass inoculum production as described in section 3.1.2. Thus 11 ascospore isolates were obtained and used for further studies.

3.2 Studies on Virulence Pattern of Different Isolates:

3.2.1 Differential Hostes: Twenty stocks including near-isogenic lines in the background of cv Chancellor and Chinese Spring, and some cvs having single or combination of two powdery mildew resistant genes were employed to study virulence pattern of isolates. Detailed information on these stocks is given in Table 2.

3.2.2 Raising of Seedlings: Seedlings of each differential line/cultivar along with susceptible check Agra local, were

Table 2. Near-isogenic lines/cultivars of wheat used for characterisation of *Erysiphe graminis* f.sp. *tritici* isolates

Near-Isogenic lines/cvs	Gene(s)	Source
Axminister x Cc^8	Pm1	Dr. J.G. Moseman (USA)
Norka x Cc^8	Pm1	"
CI 13638 x Cc^8	Pm1	"
Ulka x Cc^8	Pm2	"
CI 12632 x Cc^8	Pm2	"
Asosan x Cc^8	Pm3a	"
Chul x Cc^8	Pm3b	"
Sonora x Cc^8	Pm3c	"
Khapli x Cc^8	Pm4a	"
Yuma x Cc^8	Pm4a	"
CS/Hope	pm5	Dr.R.A.McIntosh (Australia)
TP 114	Pm6	Dr.J.G. Moseman (USA)
Timgalin	Pm6	"
Transec	Pm7	"
Kavkaz	Pm8	"
Veery	Pm8	"
Aurora	Pm8	"
Michigan Amber	Pm(Ma)	"
Sappo	Pm2+4b	Dr. M. Heun (German)
Kronjuwel	Pm4b+8	"
Agra Local		Local collection from Uttar Pradesh

raised in plastic trays (20 x 15 x 4 cm) filled with field soil and FYM (10:1). Seedlings were kept under spore proof conditions in humidity chambers covered with two layers of muslin cloth, and were irrigated as per need.

3.2.3 Inoculation of Seedlings: Eight days old seedlings of differential set at one leaf stage were inoculated with each isolate by dusting conidia from susceptible cultivar. For obtaining fresh and viable conidia, sporulating seedlings were shaken with a glass rod 24 hr prior to inoculations to dislodge old conidia. Inoculated seedlings were kept in humidity chambers for the development of disease.

Recording of Data: Observation on infection type, for each differential line, were recorded after 7-8 days of inoculation following modified scale of Smith and Blair (1950) given in Table 3.

3.3 Relative Frequency of Virulence Genes in Pathogen Populations:

3.3.1 Mobile Nurseries and Virulence Frequencies: Seedlings of differential set representing the available resistance genes and gene combinations along with the susceptible cv Agra local, were raised in plastic trays as described in section 3.2.2. Seedlings at one leaf stage were exposed, in fields at Palampur, Dhaulakuan and Jawalamukhi for 24 hrs. In some cases, where it was not possible to expose the seedlings of differential cultivars in field, sporulating plant material collected was brought to the

Table 3. Infection types used to characterize reaction types.

Infection type	Description	Reaction category
0	Immune: No infection	Resistant
;	Highly resistant: Slight flecking or necrotic spots	Resistant
1	Resistant: Slight mycelial development, very slight spore production	Resistant
2	Moderately resistant: Moderate development of mycelium slight spore production.	Resistant
3	Moderately susceptible: Moderate development of mycelium and sporulation	Susceptible
4	Very susceptible: Abundant mycelium development and sporulation	Susceptible

Modified after Smith and Blair (1950)

laboratory and shaken over the differential set. The differential set exposed to natural inoculum/dusted with it were brought back to Palampur under spore proof conditions in humidity chambers and incubated till the development of sporulating colonies. The number of colonies were counted on primary leaves of five plants of each differential line. The relative frequency of virulence genes for each resistance gene was converted into percent frequency (Wolfe and Schwarzbach, 1975). The expected frequencies of virulence alleles for specific combination of resistance genes were calculated as follows:

$$\text{Expected frequency of combined virulence on A and B} = \frac{\text{Virulence frequency on A} \times \text{Virulence frequency on B}}{100}$$

The actual and expected frequencies of virulence were compared according to Vander Plank (1984) to determine the virulence dissociation on any particular combination of genes for powdery mildew resistance.

3.4 Postulation of Powdery Mildew Resistance Genes in Wheat Stocks:

For postulation of powdery mildew resistance genes, infection-type matching technique (Browder 1973) was used. Resistance to a particular powdery mildew culture indicated the presence of genes corresponding to avirulence loci in that particular test isolate and susceptibility was indicative of absence of genes corresponding to avirulence loci in that pathotype.

One hundred forty wheat stocks showing field resistance were selected for this study (Table 4). The genotypes were exposed to eight pathotypes of Egt having diverse virulence spectrum. Avirulence/virulence formulae of pathotypes are given in Table 5.

3.4.1 Raising of Wheat Seedlings and Their Inoculation:

Seedlings of the test genotypes were raised in aluminium trays (30 x 20 x 29 cm) filled with field soil under spore proof conditions. In each tray 47 test genotypes and susceptible cultivar Agra local were sown in single rows. The method of raising seedlings, inoculations, incubation of seedlings, recording of data were same as described earlier in section 3.2.

With each test set of genotypes, a differential set was also inoculated to ensure purity of pathotypes. After testing with one pathotype, humidity chambers were thoroughly sterilized with 95% ethanol and kept in sun for two days.

3.5 Components of Resistance:

Eleven cultivars comprising of differential lines having powdery mildew resistant genes Pm1, pm5, Pm6, Pm7, Pm8, (Pm17), Pm2+4b, Pm4b+8 (Table 2), commonly grown cultivars HS 240, HS 295 and susceptible cultivar Agra local were included to examine histopathology of compatibility and incompatibility against Egt pathotype 4 (Table 5).

3.5.1 Raising of Seedlings: Seedlings of each cultivar were raised in 4" diameter pots filled with a mixture of fine

Table 4. List of cultivars/lines used in the present study and their pedigree.

S.No.	Cultivar/ Genotype	Pedigree
1	7ARSN-36	PF 70354/ALD "S"//BOW "S" CM 78565-033M-01AL-2Y-2M-OY-IAL-OY
2	7ARSN-68	IAS 58/4/KAL/BB//CJ "S"/3/ALD"S"/5/BOW"S" CM 81812-12Y-06PZ-3Y-7M-OY-2AL-OY
3	7ARSN-72	IAS 58/4/KAL/BB//CJ"S"/3/ALD"S"/5/BOW "S" CM 81812-12Y-06PZ-4Y-4M-OY-3AL-OY
4	7ARSN-85	CEP 7780/BOW "S" CM 81812-13Y-07PZ-3Y-10M-0Y-4AL-OY
5	ALDRM-64	CMH 79A,307/BN I146/BOW "S" CMH 83.2106-4B-1Y-1B-2Y-OB
6	C-2	BW-4015,KT/BAGE//FN/U/3/BZA/4/TRM/ALDAN"S"CM 47941
7	C-4	BW 5777, ALDAN "S"/IAS 58
8	C-7	BW 8535, S PB "S" CM 4340
9	C-9	BW 10284, PF 79765. LOTE 137-81
10	C-11	BW 11757, PF 74354/LD/ALD "S" OC 3551
11	C-12	BW 11770, China-7
12	C-17	BW 11775, Sanghai-5
13	C-19	BW 11763, Suzhoe-3
14	C-26	BW 12207, Au (ANHUI-11)//SUM3 NING-7840
15	C-27	BW 12208, Au (SUM 3/YMI)2 NING 8026
16	C-28	BW 12212, YMI 4/NING 7840. NING 8331
17	C-29	BW 12213, YMI 3/3/NGM No.3/SUM 1//SUM 3
18	C-31	BW 12226, 1683.8
19	C-32	BW 12238, MAYA "S"/BON
20	C-33	BW 12258, Whydah PF 7748
21	C-34	BW 12260, M2A*2/COC//ALD "S" CIT/86

Table 4 contd.....

22	C-36	BW 12584, MRNG/BUC"S"//BLO "S"/PSN"S"CM 69191
23	C-38	BW 12792, YMI-6
24	C-39	BW 12788, BR 1/3/KVZ/GV "S"/KA "S"/EMEK/32/ 4/Pat 70402/ALD "S".
25	C-43	BW 13826, Lira "S"/Ures
26	C-44	BW 14595, F.371/TRM//3383.20
27	C-49	BW 14622, ALD "S"/IAS 58,103A//NOB/3/ CEP 76184/4/GEN
28	C-50	BW 14626, Tob"S"/8156//YSOE/3*Kal/4/MRS//Kal/ BB/3/A2
29	DKW-104	Unknown
30	7DSN-2	SERI
31	7 DSN-53	CHIL "S" CM 66684-B-1M-6Y-1M-1Y-OM-68Y-OM
32	DWR-39	HDM 1508/S-308
33	DWR-151	Unknown
34	DWR-162	KVZ/BUHO//KAL/Bb
35	DWR-163	Gall/Aust II-61-157/Cno.67/3/Veery 'S'
36	18 EDYT-1*	GTA "S"/DUR 69 HRL-OY-6M-OY
37	18 EDYT-8*	CALI "S" CD 34934-I-3Y-1M-2Y-1M-1Y-2M-OY
38	18 EDYT-9*	STN "S" CD 38397-B-1M-4Y-4M-1Y-1M-1Y-OM
39	18 EDYT-21*	YAVAROS 79
40	E 506 P_3-P_1	Unknown
41	EIGSN-37	Unknown
42	EIGSN-89	Unknown
43	EIGSN-90	Unknown
44	EIGSN-118	Unknown

Table 4 contd.....

45	10 ESWYT-17	AGA/4*YR//4*HER
46	10 ESWYT-22	CAR 853/COC//VEE≠≠5"2"/3/URES
47	4 HCWSN-4	Unknown
48	4 HCWSN-73	Unknown
49	HD 2402	HD 2267 x HD 2236
50	HD 2501	HD 2189/HD 2160
51	HD 2546	VEE 'S'/PEW 'S'
52	HD 2580	TTR 'S'/JUN 'S'
53	HD 3456	Unknown
54	5 HTSN-1	GENARO
55	5 HTSN-11	FCT "S"/3/GOV/AZ//MUS "S"
56	5 HTSN-22	PARANA≠≠2//JUP/BJY"S"/3/VEE≠≠5"S"/JUN"S" CM 79694-B-1M-05Y-01M-12Y-OB
57	2HEWSN-11	ALDAN "S"/IAS 58 CM 53491-6Y-1Y-4M-1Y-1M-1Y-OM-01M'
58	2HEWSN-17	BAU"S" CM 59123-3M-1Y-1M-2Y-1M-OY
59	2HEWSN-35	SPB "S" CM 64340-4M-1Y-1M-4Y-3M-OY
60	2HEWSN-90	WRM//KAL/BB/3/BOW"S" CM70196-5Y-1M-1Y-1Y-03
61	2HEWSN-105	BOW "S"/URES CM74417-1M-06Y-06M-7Y-2B-OY
62	2HEWSN-134	F.1271/CDC//GEN CM 76689-23Y-05M-010Y-1B-2Y-0B
63	2HEWSN-175	IAS 58/4/KAL/BB//CJ"S"/3/ALD "S"/5/BOW "S" CM 81812-12Y-06PZ-3Y-1M-OY
64	2HEWSN-178	IAS 58/4/KAL/BB//CJ"S"/3/ALD "S"/5/BOW "S" CM 81812-12Y-06PZ-3Y-12M-OY
65	2HEWSN-193	VEE≠≠ 5"S"/3/BN "S"/CNT 8//ALDAN "S"/IAS 58 CM 82341-7Y-025H-OY-9M-OY
66	2HEWSN-195	TRF "S"/GHK "S"//BOW "S" CM 3042-06TCPM-18Y-01M-OY-3M-OY

Table 4 contd.....

67	2HEWSN-196	BOW "S"*2/PRL "S" CM 90319-B-4B-15Y-2B-OY
68	2HEWSN-205	CEP 7593/CEP 7887/3/PEL 72380/ATR71//BCW"S" 827812-E-OZ-OA-5A-8A-OY
69	2HEWSN-213	PF 74354//LD/ALD "S" OC 3551-4M-2Y-OZ-3Y-03AL-1Y-08-6A1-OY
70	2HEWSN-214	VEE "S"/ANA "S" CD 61785-2Y-9M-1Y-58-OY
71	HI 1077	GaIIo/Aust≠≠7/61-157//Cno/No.66/3/Kal/Bb
72	HS 295	CQT/AZ/IA S35/ALD 'S'/3/ALD'S'/NAEN/4/PIN'S'/ PEZSL 127
73	HS 321	DGA-BJY 'S'/GH 'S'
74	HUW 206	KVZ/Buho//KAL/Bb
75	HUW 284	(Pavon 76 x HUW 37) x HUW 202
76	HUW 315	HUW 206/HUW 202//CPAN 1962
77	HUW 376	CPAN 1962/HUW 206//HUW 202
78	HW 135	Unknown
79	HW 318	Unknown
80	HW 741	BB/CC//CNO/3/No/PI
81	HW 961	Unknown
82	HPW 90	Unknown
83	22 IBWSN-43	R 37/GHL 121//KAL/88/8/KLT "S" CM 64609-6Y-3M-1Y-OM-6M-OY
84	22 IBWSN-86	WRM/KAL/88/8/BOW "S" CM 70196-5Y-1M-1Y-2M-1Y-08-36M-OY
85	22 IBWSN-95	BOW "S"/BUC "S" CM 74005-8M-1Y-08M-5Y-2B-OY
86	22 IBWSN-116	KAL/88//OO "S"/8/ALD"S"/4/OPATA CM-76965-5Y-1M-06Y-1M-7Y-OB
87	22 IBWSN-148	CN087/MFD//MON "S"/3/6081 CM 81855-10Y-01M-OY-10M-OY

Table 4 contd.....

88	22 IBWSN-140	CN 087/MFD//MON "S"/3/6081 CM 81855-10Y-01M-0Y-10M-0Y
89	22 IBWSN-210	CNO 79*2/HE1 CM 90313-E-2B-3Y-2B-0Y
90	22 IBWSN-290	INIA/A.distichum//INIA/3/VEE"S"
91	24 IBWSN-49	BUC "S"/FCT "S" CM 84663-1M-0Y-0M-10Y-0M
92	20 IDSN-153*	HUI"S"//CIT 71/CII CD 64242-3M-1Y-7M-2Y-0M
93	22 IDSN-192*	DACK "S"/KIWI "S"/4/68111/RGB//WARD/3/ AMAL 72 CD 66493-B-1B-1Y-1M-BY-0B
94	ISEPTON-7	IAS 58/4/KAL/BB//CJ"S"/3/ALD"S"/5/BOW"S" CM 81812-12Y-06PZ-2Y-5M-0Y
95	ISEPTON-9	IAS 58/4/KAL/BB//CJ"S"/3/ALD"S"/5/BOW"S" CM 81812-12Y-06PZ-2Y-9M-0Y.
96	ISEPTON-17	IAS 58/4/KAL/BB//CJ "S"/3/ALD"S"/5/BOW"S" CM 81812-12Y-06PZ-4Y-17M-0Y
97	ISEPTON-25	IAS 58/4/KAL/BB//CJ"S"/3/ALD"S"/5/BOW"S" CM 81812-12Y-06PZ-4Y-9M-0Y
98	ISEPTON-30	IAS 58/4/KAL/ICJ"S"/3/ALD"S"/5/BOW"S" CM 81812-13Y-07PZ-2Y-0M-0Y
99	ISEPTON-37	IAS 8814/KAL/BB/CJ"S"/3/ALD"S"/5/BOW"S" CM 81812-8Y-08PZ-8Y-1M-0Y
100	ISWRN-193	Unknown
101	25 ISWYN-11	7C
102	IWWSN-1	3EZ ET 6224
103	K-8806	K 8002/HD 2204
104	5 KBSN-50	PARANA≠≠2//JUP/JBY"S"/3/VEE"S"/JUN"S" CM 79694-B1M-05Y-01M-11Y-08
105	5 KBSN-56	MRNG/BUC"S"//BLO"S"/PSN"S" CM 69191-A-5Y-1M-1Y-2M-2Y-2M-0Y
106	MACS 2495	Unknown
107	MACS 2496	SERI

Table 4 contd.....

108	N 9464	WH 147/HD 2189
109	PBW 154	HD 2160 x HD 2177
110	PBW 299	Bb/KAL//WL 711/PBW 65
111	PBW 316	BOW 'S'/Spt 'S'
112	PBW 320	Ures/BOW 'S'
113	RL-22	TL 68 x Shailaja
114	RL-76	Unknown
115	RL-88	Unknown
116	RL-116	TL 1210 x CPAN 1922
117	RL-117	TL 1217 x CPAN 1922
118	RL-118	TL 1217 x CPAN 1922
119	RL-122	TL 1217 x CPAN 1922
120	RL-124	TL 1217 x CPAN 1922
121	RL-126	TL 1217 x CPAN 1922
122	RL-127	TL 1217 x CPAN 1922
123	RL-132	TL 1217 x CPAN 1922
124	RL-134	TL 1217 x CPAN 1922
125	RNS-9	Unknown
126	RRM-61	AGA/4* ZA 75 CMH 84*3410-B-1B-2Y-1B-OY
127	RRM-62	AGA/5*ZA 75 CMH 79A 692-BB-1Y-1B-2Y-1B-OY
128	RRM-77	RL 6043/4*YR 70 CMH 82A.556-B-1B-6Y-1B-2Y-1B-2Y-OB
129	RRM-88	RL 6043/3*GEN 81 CMH 84.3427-A-1B-1Y-4B-OY
130	Shimla local	A local collection from Himachal Pradesh

Table 4 contd.....

131	VL-672	Unknown
132	VL-682	(Pika x HD 2160) CPAN 1557
133	VL-711	Hahn'S' *2/Pre 'S'
134	WOL(LRA)107	Sissonais/Depres//Cal/Hu/3/BUC'S'/Pun'S' T-79-154-18J-14BJ-1BJ-OBJ
135	WOL(MRA) 64	Unknown
136	WOM 35	Hahn'S'/4/Tob 66'S'/3/Cno 67/Jar 66//KVZ CM 72575-05AP-300AP-3AP-OAP
137	WOM 47	PH 93//Agatha/Ti 71*5 ICW 83-0197-0 6AP-300AP-300L-OAP
138	WON (HAA)12	Ns 984-1/NE 701136/Novosadska 2699 ICWH 82-0622-3AP-IAP-IAP-IAP-OAP
139	WON(HAA) 49	TTM'S'/Vee"S" CM 73906-05AP-300AP-4AP-300L-DAP
140	WH 569	Sepsuper/Moncha

*Triticum durum

Table 5. Avirulence/virulence formulae of the pathotypes of *Erysiphe graminis* f.sp. *tritici* used for postulation of resistance genes in 140 selected wheat stocks

Pathotype No.	Avirulence/virulence formula
1	Pm1, 2, 3b, 4, 6/ Pm3a, 3c, 5, 7, 8, Ma
2	Pm1, 2, 3a, 3b, 4, 5, 6, 8 / Pm3c, 7, Ma
3	Pm1, 2, 3c, 4, 6 / Pm3a, 3b, 5, 7, 8, Ma
4	Pm1, 2, 3a, 3b, 4, 6 / Pm3c, 5, 7, 8, Ma
5	Pm1, 2, 3b, 4, 5, 6 / Pm3a, 3c, 7, 8, Ma
6	Pm1, 2, 3a, 3b, 3c, 4, 8 / Pm5, 6, 7, Ma
7	Pm1, 2, 3a, 3c, 6, 8 / Pm3b, 4, 5, 7, Ma
8	Pm1, 2, 3b, 4, 5, 6, 8 / Pm3a, 3c, 7, Ma

field soil + FYM. In each pot, ten seeds of a cultivar were sown in a line along the diameter.

3.5.2 Inoculation of Seedlings: Eight day old potted seedlings were spread horizontally on a galvanized iron sheet with their abaxial surface upwards by placing light weights at their tips. The seedlings were inoculated by shaking the heavily mildewed seedlings of cv Agra local from a height 30 cm. Uniformity in inoculum distribution on seedling surface was examined by observing conidia trapped in vasaline coated slides placed along with these seedlings. These seedlings were incubated in the growth chambers for studying sequence of infection process at different time intervals.

3.5.3 Leaf Clearing: Inoculated leaves were cut into about one cm bits, after specific period of inoculations (12, 24, 48, 72 and 96 hr) and decolorized in Cornoy's solution (ethanol : acitic acid : water, 3:1:1) for 72 hr.

3.5.4 Staining and Preparation of Slides: The decolourized leaf bits were stained in lactophenol-trypan blue solution (0.02%) for 24 hr. Permanent slides were prepared by mounting the leaf bits in lactophenol solution and ringing the cover slips with DPX mountant diluted with xylene.

3.5.5 Microscopic observations: Microscopic observations were carried on conidial germination, germ tube elongation appresorium production, elongated secondary hyphae, haustorial formation in about 90-100 conidia from three

replicate slides. Germination counts, germ tube length, haustorial size and elongated secondary hyphae length were made using ocular micrometer. Conidia were considered to have germinated when a germ tube equal in length to the diameter of spore was observed (Menzies and MacNeill, 1987). A hyphae which has grown from a mature appressorium and attained a length of approximately 30 micron or more was considered as elongated secondary hyphae (Hyde and Colhoun, 1975). Number of units which formed haustoria were counted and converted into percentage. Length of haustorium was recorded as the distance between ends of the digitate processes and breadth taken from the middle of oval haustorial body.

Following microscopic observations on the infection process, various components of rate reducing resistance viz. incubation period, latent period, colony size, number of colonies per unit area, sporulation index and sporulation capacity were also examined.

Incubation Period: The period between inoculation and appearance of symptoms on each cultivar was recorded by observing daily the appearance of first macroscopically visible colonies.

Latent Period: The period between the inoculations and development of sporulating colonies was recorded daily under microscope.

Colony Size: The size of individual colonies (π x ½ length x

½ breadth) was measured microscopically after 8 days of inoculation. Three replications of 20 colonies each were taken for each treatment (cultivar).

Number of colonies per unit area: The number of colonies was counted on each leaf segment under a microscope. The area of each segment was estimated as length x width, and the number of colonies/cm^2 calculated (Nashaat and Moore, 1991). Three replicates of ten segments each were taken for each cultivar.

Sporulation Index: Spore producing capacity of a colony was determined using the formula developed by Shaner (1973) where Sporulation index = (P1 x 0.07531) + (P2 x 0.22280) + (P3 x 1) where P1, P2 and P3 are percent colonies having conidial chain density classes 1,2 and 3, respectively. The colony size assigned to a density class irrespective of the area based on the density of the conidial chains is as follows:

Class	Description
0	Mycelium only, no conidia produced
1	Few conidial chains ($\angle$15)
2	Numerous conidial chains, but individual chains distinct from the base.
3	Abundant dense conidial chains, individual chains can not be followed to the base.

Twenty colonies from each of the three replicates for all cultivars were used for working out sporulation index.

Sporulation Capacity: The sporulation capacity of a genotype relative to that of the susceptible control cv Agra local is estimated using the formuale given by (Nashaat and Moore, 1991).

$$\text{Sporulation Capacity} = \frac{\text{Sporulation index} \times \text{No.of colonies/cm}^2 \text{ (host genotype)}}{\text{Sporulation index} \times \text{No.of colonies/cm}^2 \text{ (Agra Local)}} \times 100$$

Analysis of variations was carried out following completely randomized design (CRD).

EXPERIMENTAL RESULTS

4 EXPERIMENTAL RESULTS

4.1 Pathogenic Specialization in Erysiphe graminis f.sp. tritici (Egt)

4.1.1 Virulence Pattern of Isolates on Standard Differential Lines: Fifty six single colony isolates, 45 conidial and 11 ascosporic, were collected from various places in diverse agroclimatic regions of the state (Table 1). Based on their reaction on a standard set of differential lines having known powdery mildew resistant genes Pm1 through Pm8, Pm(Ma) (an unidentified gene from the cv Michigan Amber) and on genotypes with gene combination Pm2+4b, Pm4b+8 were tested. These isolates were grouped into 30 pathotypes (Table 6 and 7), out of which 24 were of conidial origin and 6 ascosporic origin. Among the conidial isolates pathotypes 7 and 12 identified among the isolates collected from Palampur (Mid hill Zone) and Paisa, Nehran Pukhar (Low hill Zone) were the most prevalent pathotypes. Pathotype 7 was the least virulent attacking only one gene Pm(Ma). Virulence of pathotype 1 collected from Dalang Maidan (Lahaul & Spiti) was restricted to the two genes i.e. Pm3c and Pm(Ma). Pathotype 16 identified from the isolates of Palampur and Jaisinghpur (Mid hill Zone) giving susceptible reaction on 8 genes i.e. Pm3a, Pm3b, Pm3c, pm5, Pm6 (Timgalin), Pm7, Pm8 and Pm(Ma) was the most virulent. Pathotypes 10, 12, 14 and 23 were virulent on 7 genes i.e. Pm3b Pm3c, pm5, Pm6(Timgalin), Pm7, Pm8, Pm(Ma); Pm3a, Pm3c, pm5, Pm6(Timgalin), Pm7, Pm8, Pm(Ma); Pm1, Pm3a, Pm3b, Pm3c, Pm5, Pm8, Pm(Ma) and Pm3c, Pm3b, Pm3c, pm5, Pm6(Timgalin), Pm7, Pm(Ma), respectively.

Table 6. Virulence pattern of conidial isolates of Erysiphe graminis f.sp. tritici on differential lines having known powdery mildew resistance genes.

Patho-type	Isolate No.	Pm1	Pm2	Pm3a	Pm3b	Pm3c	Pm4a	pm5	Pm6 (TP114)	Pm6 (Timgalin)	Pm7	Pm8	Pm2+4b	Pm4b+8	Pm (Ma)	Agra local
1	1	R	R	*	R	S	R	R	R	R	R	R	R	R	S	S
2	2	R	R	*	R	R	R	S	R	R	R	S	R	R	S	S
3	3,4	R	R	*	R	R	R	S	R	S	R	S	R	R	S	S
4	5	S	R	*	R	S	R	S	R	R	S	S	R	R	S	S
5	7,8	R	R	S	R	S	R	S	R	S	R	S	R	R	S	S
6	9	R	R	S	R	S	R	S	R	S	R	R	R	R	R	S
7	10,11,12,13,44	R	R	R	R	R	R	R	R	R	R	R	R	R	S	S
8	14	R	R	R	R	R	R	S	R	S	R	R	R	R	S	S
9	15,16	R	R	S	R	R	R	R	R	S	R	R	R	R	S	S
10	28	R	R	R	S	S	R	S	R	S	S	S	R	R	S	S
11	29,34,45	R	R	S	R	S	R	R	R	S	S	S	R	R	R	S
12	30,37,38,39,40	R	R	S	R	S	R	S	R	S	S	S	R	R	S	S
13	31	R	R	S	S	R	R	R	R	S	S	S	R	R	S	S
14	32	S	R	S	S	S	R	S	R	R	R	S	R	R	S	S
15	35,41	R	R	S	R	S	R	R	R	R	R	R	R	R	S	S
16	36,17	R	R	S	S	S	R	S	R	S	S	S	R	R	S	S
17	42,18,19	R	R	S	S	S	R	R	R	S	S	R	R	R	S	S
18	43	R	R	*	S	S	R	S	R	R	S	R	R	R	S	S
19	20,21	R	R	R	R	S	R	S	R	R	R	R	R	R	S	S
20	22,26	R	R	R	R	S	R	S	R	S	S	R	R	R	S	S
21	23	R	R	R	R	S	R	S	S	S	S	R	R	R	S	S
22	24,6	R	R	R	R	*	R	S	R	S	S	R	R	R	S	S
23	33	R	R	S	S	S	R	S	R	S	S	R	R	R	S	S
24	25,27	R	R	S	R	S	R	S	R	S	*	R	R	R	S	S

*Data not recorded

Table 7. Virulence pattern of ascosporic isolates of Erysiphe graminis f.sp. tritici on differential lines.

Patho-type	Isolate	Pm1	Pm2	Pm3a	Pm3b	Pm3c	Pm4a	pm5	Pm6 (TP114)	Pm6 (Timgalin)	Pm7	Pm8	Pm2+4b	Pm4b+8	Pm(Ma)	Agra local
25	A	R	R	S	R	S	R	S	R	S	S	R	R	R	S	S
26	B	*	R	R	R	S	R	S	R	R	R	R	R	R	S	S
27	C,D,E,F	R	R	R	R	R	R	S	R	S	S	R	R	R	S	S
28	G,H	S	R	R	R	*	R	S	R	S	S	R	R	R	S	S
29	I	R	R	R	R	S	R	S	R	S	S	R	R	R	S	S
30	J,K	R	R	S	R	R	R	S	R	S	S	R	R	R	S	S

* Data not recorded

Among the cleistothecial pathotypes, pathotype 26 with virulence on Pm3c, pm5 and Pm(Ma) was least virulent whereas pathotype 25 attacking genes Pm3a, Pm3c, pm5, Pm6(Timgalin), Pm7 and Pm(Ma)was the most virulent pathotype. Pathotype 20 and 29 identified among the conidial and ascosporic isolates, respectively were identical. Ulka x Cc^8 (Pm2), Khapli x Cc^8 (Pm4a), Sappo (Pm2+4b) and Kronjuwel (Pm4b+8) were the most effective genotypes as none of the conidial as well as ascosporic isolates could attack them. Virulences on genes Pm1 (Axminister x Cc^8) and Pm6 (TP 114) were less prevalent.

4.1.2 Virulence Structure:

4.1.2.1 Pathotypes Prevalence in Relation to Virulence: Results on relationship of virulence of pathotypes with frequency of their occurence are given in (Table 8). 33.93% isolates were virulent on six to eight powdery mildew resistance genes, while 10.72% isolates were virulent on one to two resistance genes. Majority of isolates (55%) were virulent on three to five powdery mildew resistance genes whereas only 3.57% isolates were virulent on eight genes and 8.93% on one gene. It is evident from this table that isolates with intermediate virulence are more frequent.

4.1.2.2 Changes in virulence frequency of E. graminis f.sp. tritici with advancement of the crop season: Virulence frequency of E. graminis f.sp. tritici population was monitored at Palampur by exposing the seedlings of wheat powdery mildew differential lines at 15 days interval, starting from

Table 8. Relationship of virulence with frequency of occurrence of different pathotypes of *E. graminis* f.sp. *tritici*

Pathotypes	No.of powdery mildew resistance genes attacked	No. of isolates	Frequency isolates (%)
7	1	5	8.93
1	2	1	1.79
2,8,9,15,19,26	3	9	16.07
3,6,22,27	4	9	16.07
11,18,20,24,28,29,30	5	13	23.21
4,5,13,17,21,25	6	9	16.07
10,12,14,23	7	8	14.29
16	8	2	3.57

January 20, 1993 till June 4, 1993 and the results are given in Table 9. During the period under study no virulence was trapped on genes Pm1 (Norka x Cc^8, Axminister x Cc^8), Pm2 (Ulka x Cc^8), Pm4a (Khapli x Cc^8), Pm6 (TP 114), Pm2+4b (Sappo) and Pm4b+8 (Kronjuwel). Virulences on genes Pm3a, Pm3c, pm5, Pm6 (Timgalin) and Pm(Ma) were present throughout the crop season. Virulences on genes Pm3b, Pm7 and Pm8 were trapped at the end of crop season.

4.1.2.3 Virulence Structure of Pathogen Populations at Different Locations: The results on virulence structure of E. graminis tritici as studied with the help of mobile nurseries or by inoculating seedlings of differential lines are given in the Table 10. No virulence was trapped on genes Pm2 (Ulka x Cc^8), Pm4a (Khapli x Cc^8), Pm2+4b (Sappo) and Pm4b+8 (Kronjuwel) at places under study while virulences on genes Pm1 (Axminister x Cc^8) and Pm6 (TP 114) were trapped at Kukumseri and Dhaulakuan respectively. Virulence on gene Pm3b (Chul x Cc^8) was trapped at Dehra (6.70%), Dhaulakuan (4.85%) and Jawalamukhi (11.20%). Virulences on genes Pm3a, Pm3c, pm5, Pm6 (Timgalin), Pm7, Pm8 and Pm(Ma) were most common at all the places, highest being at Kukumseri (32.40%), Jaisinghpur (30.76%), Jawalamukhi (17.90%), Dhaulakuan (17.40%), Nehran Pukhar (22.85%), Palampur (22.80%) and Kukumseri (28.75%), respectively.

4.1.2.4 Virulence Frequency of Pathotypes Attacking Pm Genes: Data on virulence frequency of pathotypes attacking specific powdery mildew resistance genes are given in Table 11. Gene

Table 9. Virulence frequency (%) of Erysiphe graminis tritici populations during different dates in 1993 at Palampur

Differentials	Gene(s)	January 20	February 4	February 19	March 6	March 21	April 5	April 20	May 5	May 20	June 4
Norka x Cc^8	Pm1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Axminister x Cc^8	Pm1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Ulka x Cc^8	Pm2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Asosan x Cc^8	Pm3a	11.90	10.53	10.58	10.50	15.15	12.15	12.15	8.00	16.27	8.90
Chul x Cc^8	Pm3b	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.66	0.95	0.66
Sonora x Cc^8	Pm3c	3.57	3.50	0.00	2.75	7.05	11.20	6.20	9.73	20.60	8.75
Khapli x Cc^8	Pm4a	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
CS/Hope	pm5	0.00	5.26	15.05	33.33	30.30	36.40	11.12	7.90	3.56	7.90
TP 114	Pm6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Timgalin	Pm6	21.43	43.86	44.05	27.00	25.15	22.15	15.15	16.62	12.59	17.64
Transec	Pm7	0.00	0.00	0.00	0.00	0.00	0.00	0.00	5.57	25.53	5.49
Kavkaz	Pm8	0.00	0.00	0.00	0.00	0.00	15.22	22.30	2.33	1.70	2.33
Aurora	Pm8	0.00	0.00	0.00	0.00	0.00	5.03	8.15	15.44	4.75	15.56
Veery	Pm8	0.00	0.00	0.00	0.00	0.00	7.00	10.10	16.62	3.68	17.64
Sappo	Pm2+4b	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Kronjuwel	Pm4b+8	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Michigan Amber x Cc^8	Pm(Ma)	63.10	35.04	35.20	41.66	19.20	14.00	24.00	17.12	8.91	17.14

Table 10. Virulence frequency (%) of *Erysiphe graminis* f.sp. *tritici* populations at different locations in Himachal Pradesh

Location	Differential lines with gene(s)													
	Pm1	Pm2	Pm3a	Pm3b	Pm3c	Pm4a	pm5	Pm6 (Tp 114)	Pm6 (Timgalin)	Pm7	Pm8	Pm2+4b	Pm4b+8	Pm(Ma)
Dehra	0.00	0.00	22.65	6.70	8.15	0.00	15.25	0.00	12.40	10.71	5.80	0.00	0.00	19.20
Dhaulakuan	0.50	0.00	29.40	4.85	6.70	0.00	10.90	3.00	17.40	8.55	7.26	0.00	0.00	10.65
Jawalamukhi	0.00	0.00	16.73	11.20	3.40	0.00	17.90	0.00	0.00	16.70	11.12	0.00	0.00	22.15
Jaisinghpur*	0.00	0.00	10.85	0.00	30.76	0.00	15.25	0.00	4.60	15.80	2.80	0.00	0.00	24.14
Kukumseri*	1.60	0.00	32.40	0.00	12.50	0.00	7.00	0.00	5.70	6.25	7.15	0.00	0.00	28.75
Nehran Pukhar	0.00	0.00	21.72	0.00	5.60	0.00	12.15	0.00	0.00	22.25	14.36	0.00	0.00	22.70
Palampur	0.00	0.00	12.75	0.00	6.80	0.00	11.72	0.00	15.50	5.57	22.80	0.00	0.00	24.90

*Data based on reaction of differential lines, with the mass inoculum brought from these places.

Pm(Ma) succumbed to 28 pathotypes with virulence frequency of 92.86% followed by gene Pm6 (Timgalin) 73.24%. Virulence frequency on genes Pm3a, Pm3b, Pm3c, pm5, Pm7 and Pm8 ranged between 17.86 to 69.64% and the frequency on genes Pm1 (Axminister x Cc^8) and Pm6 (TP 114) was 7.27 and 1.79%, respectively. Genes Pm2 (Ulka x Cc^8), Pm4a (Khapli x Cc^8) Pm2+4b (Sappo), Pm4b+8 (Kronjuwel) were highly effective as no pathotype could attack these genes.

4.1.2.5 Association/Dissociation of Virulence: In order to determine whether virulence on certain combinations of powdery mildew resistance genes occurs in more or less than the expected frequency as calculated from virulence on single powdery mildew resistant genes, actual and theoretical frequencies of virulences on all possible gene combinations were calculated from 56 isolates and data are given in the Table 11. The diagonal entries shows virulence frequency percentage for powdery mildew resistant genes, individually. Thus 92.86% of isolates were virulent on Pm(Ma) and 7.27% on gene Pm1. Entries above the diagonal show actual frequency of virulence for specific Pm gene pairs. Thus 7.14% of the isolates were actually virulent on both genes Pm1 and Pm(Ma). Entries below the diagonal line shows the theoretical (expected percentage) frequency of virulence for each pair of genes, provided virulences are distributed randomly. Thus 92.86 x 7.27/100 = 6.75% of the isolates would, if randomly distributed be virulent on both Pm1 and Pm(Ma) genes.

Table 11. Actual and theoretical frequency of association of different genes for virulence in Erysiphe graminis tritici.

Theoretical	Actual	Percent isolates virulent on Pm genes											
	Pm1	2	3a	3b	3c	4a	5	6 (TP114)	6 (Timgalin)	7	8	2+4b	4b+8	Ma
Pm1	7.27	0.00	1.82	1.82	3.64	0.00	7.14	0.00	3.57	5.36	3.64	0.00	0.00	7.14
	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
a	4.07	0.00	56.00	14.54	42.59	0.00	33.33	0.00	46.30	34.61	26.92	0.00	0.00	48.00
b	1.30	0.00	10.00	17.86	16.07	0.00	10.71	0.00	16.07	16.07	8.93	0.00	0.00	17.86
c	4.75	0.00	36.61	11.68	65.38	0.00	48.07	1.79	48.09	39.29	26.79	0.00	0.00	57.69
a	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
	5.06	0.00	39.00	12.44	45.53	0.00	69.64	1.79	57.14	48.15	26.79	0.00	0.00	67.85
(TP 114)	0.13	0.00	1.00	0.32	1.17	0.00	1.25	1.79	1.79	1.79	1.79	0.00	0.00	1.79
(Timgalin)	5.32	0.00	41.01	13.08	47.88	0.00	51.00	1.31	73.24	57.40	28.57	0.00	0.00	66.07
	4.44	0.00	34.2	10.91	39.95	0.00	42.56	1.09	44.76	61.11	23.21	0.00	0.00	55.55
	2.47	0.00	19.00	6.06	22.18	0.00	23.63	0.61	24.85	20.73	33.93	0.00	0.00	28.57
+4	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
+8	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
	6.75	0.00	52.00	16.58	60.71	0.00	64.67	1.66	68.01	56.75	31.51	0.00	0.00	92.86

Hence, the frequency of isolates actually virulent on the genes Pm1 and Pm(Ma) is more than the theoretical figure (6.75%) indicating the associated virulence for these two genes. Similarly, the associated virulence for Pm1, with Pm3b pm5, Pm7 and Pm8 in which actual figures are greater than theoretical figures, was found to be associated. However, associated virulence of all available gene combinations with gene Pm2, Pm4a, Pm2+4b, Pm4b+8 and Pm1 with Pm6 (TP 114) Pm3a with Pm6 (TP 114), Pm3b with Pm6 (TP114) was found to be absent. Combined virulence for all other gene combinations was quite prevalent.

4.2 Postulation of Pm Resistance Genes

Wheat genotypes (140) comprising cultivated varieties and genotypes having field resistance were subjected to eight diverse pathotypes of Egt. Based on the differential response to these pathotypes all stocks were grouped into XII different categories (Table 12) and probable powdery mildew resistance genes were postulated in them (Table 13).

Group I: One genotype 7 ARSN-68 was susceptible to pathotype 3 and 5 and resistant to all other pathotypes. Data for pathotype 6 could not be recorded. Resistance of this genotype was postulated to be due to gene(s) Pm3a, Pm8 and some other gene(s), which could not be detected with the present pathotypes, individually or in combination.

Group II: Ten genotypes in this group were subdivided into three categories. Eight stocks in subgroup (a) were resistant to pathotypes 6 and 7 and susceptible to all other pathotypes,

Table 12. Reaction of 140 wheat cvs/genotypes to eight pathotypes of Erysiphe graminis f.sp. tritici and their groupings.

Group		Cultivar/Genotypes	Pathotype No.							
			1	2	3	4	5	6	7	8
I		7 ARSN-68	;	;	4	;	3	-	;	;
II	(a)	DWR-163, HUW 284,HW 135, MACS 2496,RRM-61,Shimla local, VL 672, WOM 49	4	4	-	4	4	;	;	-
	(b)	DWR-39	3	3	0	4	4	;	-	-
	(c)	HD 2501	4	3	-	4	4	2	0	4
III	(a)	18-EDYT-8	3	3	1	3	;	-	;	-
IV	(a)	10 ESWYT-17	4	-	-	3	4	;	;	2
	(b)	RL-116	4	;	-	4	4	;	;	-
V		C-32, C-34, 22IBWSN-116	3	;	3	4	;	4	3	;
VI	(a)	EIGSN-90, RNS-9	3	;	4	3	1	4	;	0
	(b)	5 HTSN-1	4	;	4	4	-	4	;	-
	(c)	5 HTSN-11, PBW 154	4	;	-	4	-	;	3	-
VII		C-36, C-44	3	;	3	4	;	;	1	-
VIII	(a)	7 ARSN-36, 7 ARSN-72, 2 HEWSN-35, 2 HEWSN-193	4	;	4	3	4	;	;	;
	(b)	4HCWSN-4, HS 321, RRM-77	4	;	3	4	4	2	;	;
	(c)	C-9, C-11, C-12, C-38, 7DSN-53, 2HEWSN-213, HW 318,19 ISEPTON-25,19 ISEPTON-30, ISWRN-193, K-8806, RL-117, RL-118, RL-122, RL-124, RL-126, RL-127, RL-132, RRM-88, VL 711, WON 12	3	;	4	4	4	;	;	-
	(d)	2HEWSN-134, RL-126	3	;	3	4	4	;	-	2
IX	(a)	C-39	3	;	1	4	3	;	;	-
	(b)	EIGSN-89	;	;	3	4	4	;	;	2

Table 12 contd.....

Group		Cultivar/Genotypes	Pathotype No.							
			1	2	3	4	5	6	7	8
X	(a)	ALDRM-64, DKW-104, EIGSN-37, HD 2546	3	4	4	4	3	;	;	3
	(b)	7ARSN-85, C-29, C-31	4	;	4	3	4	4	4	;
	(c)	C-2, C-17	4	3	4	;	;	4	4	4
	(d)	C-26,C-27,C-28	3	;	4	;	4	3	4	4
	(e)	DWR-162, EIGSN-118, HUW 315 HD 2402, 2HEWSN-214, 22 IBWSN-86, MACS 2495	4	3	4	4	4	4	;	4
	(f)	10 ESWYT-22, 2HEWSN-175	3	;	4	4	4	4	;	4
	(g)	E 506 P_1P_2, HD 2580, HS 295 2HEWSN-90, 2HEWSN-105, 5 HTSN-22, 20 IDSN-153, 5 KBSN-56, N 9464, VL-682	3	3	4	4	4	;	3	4
	(h)	2HEWSN-205, 22IBWSN-290, WOL 107	3	3	4	4	4	;	3	2
	(i)	2 HEWSN-196, 22 IBWSN-43, 22 IBWSN-148, ISEPTON-7, ISEPTON-9, ISEPTON-17, ISEPTON-37, PBW 316	4	;	4	4	4	4	3	3
	(j)	2HEWSN-178; RL-134	3	;	4	4	4	;	4	4
XI	(a)	RL-22	;	;	;	;	;	2	;	;
	(b)	WH 569	;	;	;	1	;	2	;	2
XII	(a)	C-4, C-7, C-49, C-50,DWR-151, 18 EDYT9, IWWSN-1,22 IDSN-192, PBW 299, PBW 320,RL-76, RRM-62, WOM 35, WOM 47	3	3	4	4	4	4	4	4
	(b)	C-19, C-33, IDSN-2, HUW 206 HUW 376, HW 961, HPW 90, 22 IBWSN-95, 24-IBWSN 49	4	4	4	4	4	4	4	4
	(c)	18 EDYT-1, HD 3456,4HCWSN-73, HW 741, 22 IBWSN-210	4	3	4	4	4	3	3	4
	(d)	18 EDYT-21, 2HEWSN-11, 2HEWSN-17, 2HEWSN-195,HI 1077, 25 ISWYT-11, 5KBSN-50	3	3	4	4	4	3	3	4
	(e)	22 IBWSN-149, RL-88, WO1 (MRA) 64	3	3	4	3	4	-	-	-

Table 13. Probable powdery mildew resistance genes in 140 genetic stocks classified into XII groups

Group	Cultivars/Genotypes	Probable gene(s)
I	7 ARSN-68	Pm3c, Pm8+
II	DWR-39, DWR-163, HD 2501, HUW 284, HW 135, MACS 2496, RRM-61, Shimla Local, VL 672, WOM 49.	Pm3c
III	18 EDYT-8	Pm3c+
IV	10 ESWYT-17, RL-116	Pm3c, Pm8
V	C-32, C-34, 22IBWSN-116	pm5
VI	EIGSN-90, 5 HTSN-1, 5 HTSN-11, PBW-154, RNS-9	pm5+
VII	C-36, C-34	pm5, Pm8+
VIII	7ARSN-36, 7ARSN-72, C-9, C-11, C-12, C-38, C-43, 7 DSN-53, 4 HCWSN-4, 2HEWSN-35, 2HEWSN-134, 2HEWSN-193, 2HEWSN-213, HS 321, HW 318, 19 ISEPTON-25, 19 ISEPTON-30, ISWRN-193, K-8806, RL-117, RL-118, RL-122, RL-124, RL-126, RL-127, RL-132, RRM-77, RRM-88, VL 711, WON(HAA) 12	Pm8
IX	C-39, EIGSN-89	Pm8+
X	ALDRM-64, 7ARSN-85, C-2, C-17, C-26, C-27, C-28, C-29, C-31, DKW-104, DWR-162, E 506, P_3P_1, EIGSN-37, EIGSN-118, 10ESWYT-22, HD 2402, HD 2546, 2HEWSN-90, 2HEWSN-105, 2HEWSN-175, 2HEWSN-178, 2HEWSN-196, 2HEWSN-205, 2HEWSN-214, HS 295, 5HTSN-22, HUW 315, 22 IBWSN-43, 22 IBWSN-86, 22 IBWSN-148, 22 IBWSN-290, 20 IDSN-153, 19 ISEPTON-7, 19ISEPTON-9, 19 ISEPTON-17, 19ISEPTON-37, 5 K BSN-56, MACS 2495, N 9464, PBW 316, RL-134, VL 682, WOL 107	New gene(s)
XI	RL-22, WR 569	?
XII	C-4, C-7, C-19, C-33, C-49, C-50, 7DSN2, DWR-151, 18 EDYT-1, 18 EDYT-9, 18EDYT-21, HD 3456, 4HCWSN-73, 2HEWSN-11, 2HEWSN-17, 2HEWSN-195, HI 1079, HPW 90, HW 741, HW 961, HUW 206, HUW 376, 22 IDSN-192, 22 IBWSN-95, 22 IBWSN-149, 22 IBWSN-210, 24 IBWSN-49, 25 ISWYT-11, IWWSN-15, 5KBSN 50, PBH 299, PBW 320, RL-76, RL-88, RRM-62, WOL(MRA) 64, WOM 35, WOM-47.	None

whereas stocks DWR-39 and HD 2501 in sub group b and c were resistant to pathotypes 3,6 and 6,7, respectively, and susceptible to rest of the pathotypes. Based on infection-type matching technique, gene Pm3c was found to be responsible for resistance in these genotypes.

Group III: One genotype, 18 EDYT-8 resistant to pathotypes 3,5 and 7 and susceptible to rest of the pathotypes was included in this group. The data for pathotypes 6 and 8 could not be recorded. The differential response to the pathotypes inferred the presence of gene Pm3c and some other unidentified gene(s) individually or in combination, in this genotype.

Group IV: On the basis of reaction to pathotypes, genotypes 10 ESWYT-17 and RL-116 in this group were subgrouped into two categories. Genotype 10 ESWYT-17 and RL-116 showed resistance to cultures 6,7 and 8 and 2,6 and 7, respectively and susceptibility to rest of the pathotypes. Data for pathotypes 2 and 3 and 3,8 could not be recorded for genotypes 10 ESWYT-17 and RL-116, respectively. Subsequently, genes Pm3c and Pm8 individually or in combination were found to be responsible for the resistance of these genotypes.

Group V: Genotypes C-32, C-34, 22 IBWSN 116 showing resistance to pathotypes 2,5 and 8 and susceptibility to the remaining pathotypes were included in this group. Analysis of data following infection-type matching technique, inferred the presence of gene pm5 for resistance.

Group VI: Five genotypes in this group were further sub grouped into 3 categories. Genotypes EIGSN-90, RNS-9 showing resistance to pathotypes 2,5,7 and 8; genotype 5 HTSN-1 to 2 and 7 and genotypes 5 HTSN-11, PBW 154 to 2 and 6, respectively and susceptibility to rest of the pathotypes were subgrouped into a, b and c. However, the data was not recorded for pathotypes 5,8 and 3,5,8 respectively, for the genotypes in subgroup b and c. Based on the infection-type matching technique, gene(s) pm5 and some other unidentified gene(s) were found to be conferring resistance to these cultivars.

Group VII: Two genotypes C-36 and C-44 showed resistance to pathotypes 2,5,6 and 7 and susceptibility to pathotypes 1,3 and 4. Resistance of these cvs was found to be due to gene pm5, Pm8 and other gene(s) individually and in combination.

Group VIII: Based on the differential reaction with the pathotypes, 30 genotypes of this group were further subgrouped into four categories. Genotypes 7 ARSN-36, 7 ARSN-72, 2 HEWSN-35 and 2 HEWSN-193 showed resistance to pathotypes 2,6,7 and 8 and were susceptible to pathotypes 1,3,4 and 5 and were included in subgroup 'a'. Genotypes 4HCWSN-4, HS 321 RRM-77 developed infection types ";" with pathotypes 2,7 and 8, infection type 2 with pathotype 6 and IT = 3-4 with rest of the pathotypes. Twenty stocks in subgroup 'b' developed infection type ";" with pathotypes 2,6 and 7 and infection types 3-4 to rest of the pathotypes except pathotype 8 for which data could not be recorded. Genotypes 2 HEWSN-134, RL-126 showed resistance (1T = ;) to pathotypes 2,6, and 8 and

susceptibility (IT = 3-4) to rest of pathotypes, however, data for pathotype 7 could not be recorded. On the basis of infection-type matching technique resistance in these genotypes was postulated to be due to gene Pm8.

Group IX: Two stocks C-39 and EIGSN-89 in this group were subgrouped into 2 subcategories. Genotype C-39 and EIGSN-89 showed resistance to pathotypes 2,3,6,7 and 1,2,6,7,8 respectively and susceptibility to rest of the pthotypes. However, data for pathotype 8 could not be recorded in C-39. Hence, resistance of these genotypes was found to be due to gene(s), Pm8 and some other gene(s), which could not be identified with the pathotypes used.

Group X: This group comprised of 44 genotypes, subgrouped into 10 categories. Genotypes ALDRM-64, DKW-104, EIGSN-37, HD 2546 in subgroup 'a' showed resistance (IT = ;) to pathotypes 6 and 7 and susceptibility (IT = 3-4) to all other pathotypes. Genotypes 7 ARSN-85, C-29, C-31 showed resistance (IT = ;) to pathotypes 2 and 8 and susceptibility (IT = 3-4) to rest of the pathotypes, were included in subgroup 'b'. Genotypes C-2 and C-17 showing resistance to pathotypes 4 and 5 and susceptibility reaction to the remaining pathotypes were subgrouped in group 'c'. In subgroup 'd', three genotypes C-26, C-27, C-28 gave resistance reaction (IT = ;) to pathotypes 2 and 4 and susceptible (IT = 3-4) to rest of the pathotypes. Genotypes DWR-162, EIGSN-118, HD 2402, 2HEWSN-214, HUW 315, 22 IEWSN-86, MACS 2495 showing resistant reaction (IT = ;) to pathotype 7 only were included in subgroup 'e'. In subgroup 'f', 2 genotypes 10 ESWYT-22 and 2HEWSN-175 gave resistant (IT = ;) reaction for

pathotype 2 and 7 and susceptible (IT = 3-4) to rest of pathotypes. Eleven genotypes in subgroup 'g' showed resistant reaction (IT = ;) to pathotype 6 and susceptible to rest of the pathotypes. Three genotypes 2 HEWSN-205, 22-IBWSN-290 and WOL 107 resistant (IT = ;-1) to pathotypes 6 and 8 and susceptible to the remaining pathotypes were included in subgroup 'h'. Eight genotypes in subgroup 'i' were found to be resistant (IT = ;) to pathotype 2 and susceptible to all other pathotypes. Two cvs 2HEWSN-178, RL-134 in subgroup 'j' were found to be resistant (IT = ;) to pathotype 2 and 6 and susceptible to rest of the pathotypes. It is clear from the reaction pattern that genotypes in group x behaved differently to one pathotype or the other, however, using infection-type matching tehcnique, resistance in these genotypes could not be attributed to any of the known gene(s).

Group XI: Two genotypes namely RL-22 and WH 569 in this group showed resistance (IT = ; -2) to eight pathotypes. Due to non differential response of these genotypes to the pathotypes used, the resistance gene(s) in these two cultivars could not be specified.

Group-XII: The cultivars in this group were found susceptible (IT = 3-4) to all the pathotypes. So none of the resistance gene(s) found in this group.

4.3 Components of Resistance
Infection Process of Erysiphe graminis f.sp. tritici pathotype 4 on Wheat Genotypes:

A. Compatible Host-Parasite Combination: In compatible host parasite combination involving universal susceptible cv Agra

PLATE I

Infection process of E. graminis f.sp. tritici pathotype 4 on cv Agra Local

Fig 1-6

1 Mature appressoria (app), septa (se) and conidia (C), (12 hr after inoculation) x 200.

2 Papilla (pa), primary haustoria (Pha) and Primary germ tube (Pgt), (24 hr after inoculation) x 400.

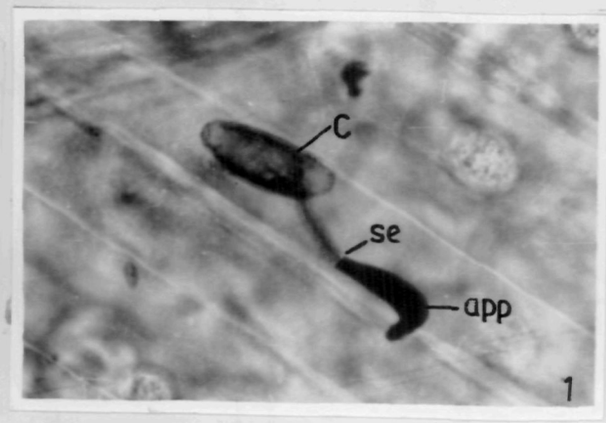
3 Digitate processes (DP), elongated secondary hyphae (ESH) and conidiophore (cp), (48 hr after inoculation) x 400.

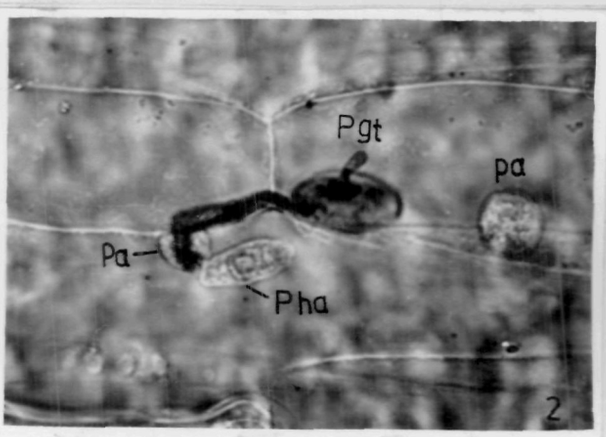
4 Secondary haustoria (Sha), (72 hr after inoculation) x 400.

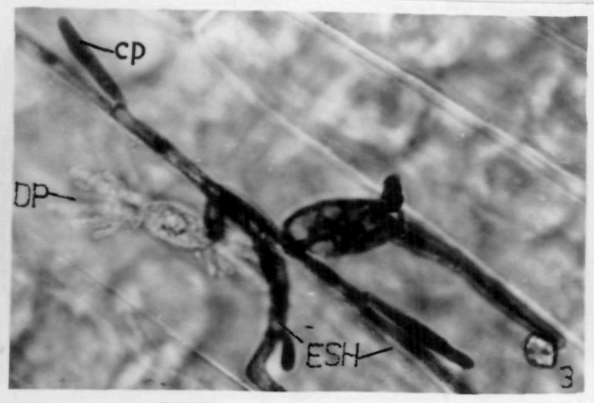
5a Secondary penetration sites with blue haloes, indicated by arrow, (6 day after inoculation) x 400.

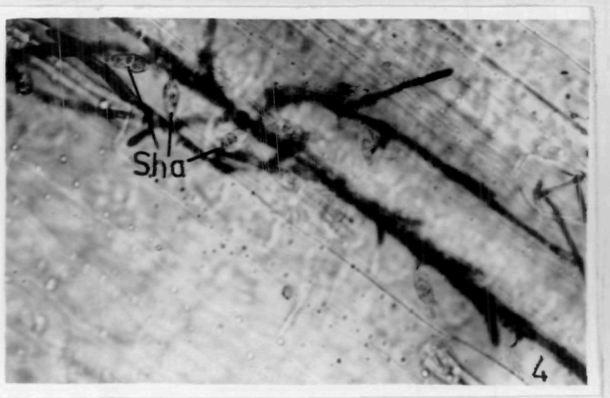
5b Smooth penetration hole in the haloes, indicated by arrow, (6 day after inoculation) x 400.

6 Conidiophores (cp) and Conidial Chains (CC), (6 day after inoculation) x 200.


C
se
app
1


Pgt
pa
Pa
Pha
2


cp
DP
ESH
3


Sha
4


5a


5b


CC
cp
6

PLATE II

Infection Process of *E. graminis* f.sp. *tritici* pathotype 4 on genotypes Sappo (Fig 7-9) and Kronjuwel (Fig 10-13).

Fig 7-13

7 Highly reduced germ tube growth, indicated by arrow, (12 hr after inoculation) x 200.

8 Mature appressoria (app) along with ungerminated conidia (UC), (24 hr after inoculation) x 200.

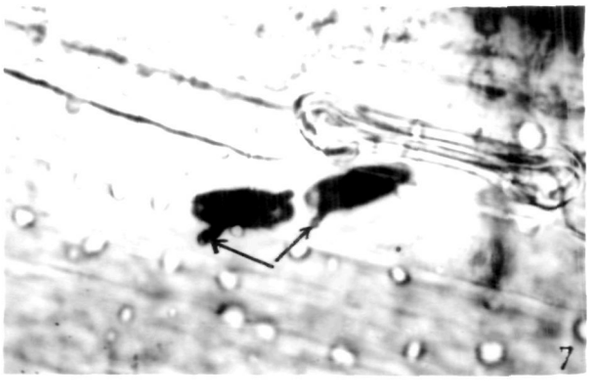
9 Shrivelled and distorted growth of the appressorial germ tube, indicated by arrow, (48 hr after inoculation) x 200.

10 Mature appressoria (app) and Primary germ tube (Pgt), (12 hr after inoculation) x 200.

11 Papilla (pa), (24 hr after inoculation) x 400.

12 Shrivelled and distorted growth of the pathogen, indicated by arrow (48 hr after inoculation) x 200.

13 Loose and disorganised mesophyll cells, (48 hr after inoculation) x 400.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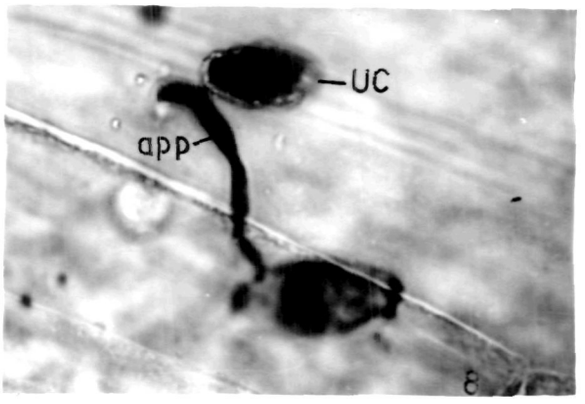

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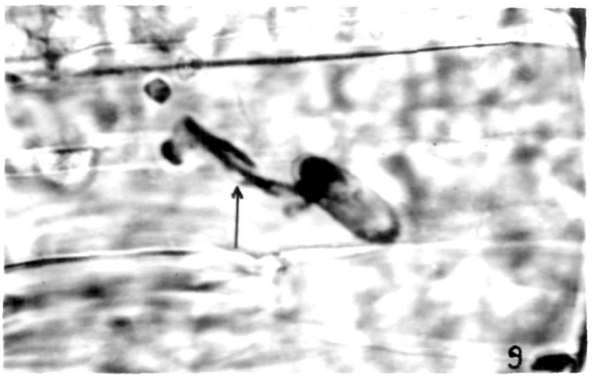
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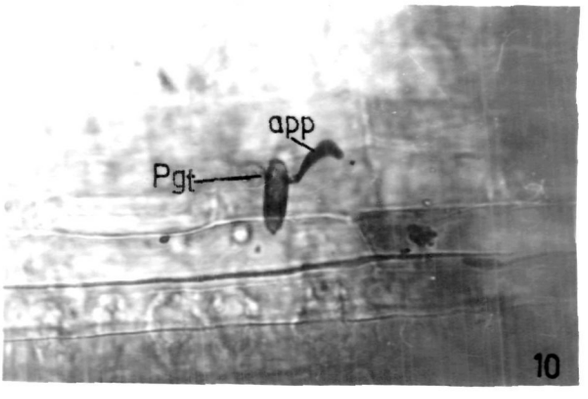

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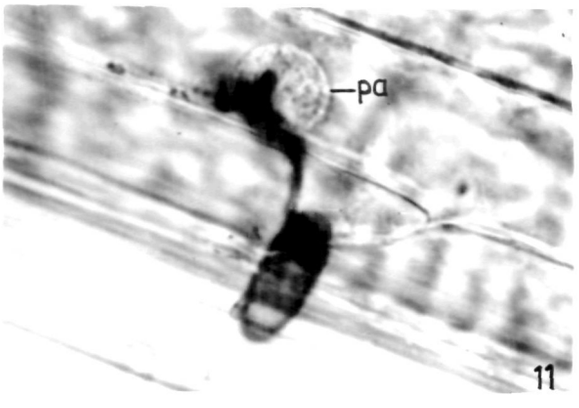

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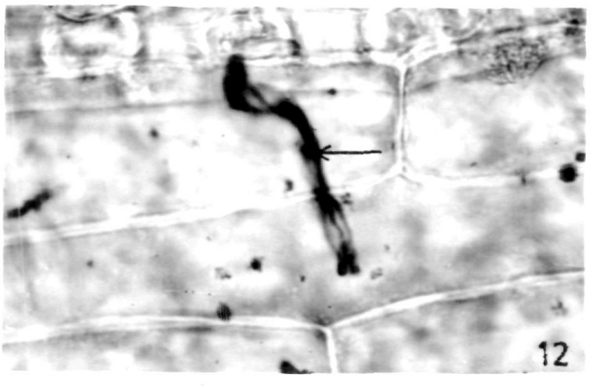
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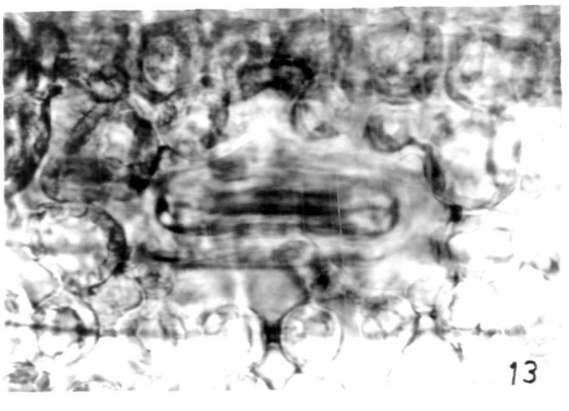

13

PLATE III

Infection Process of *E. graminis* f.sp. *tritici* pathotype 4 on genotype TP 114.

Fig 14-18

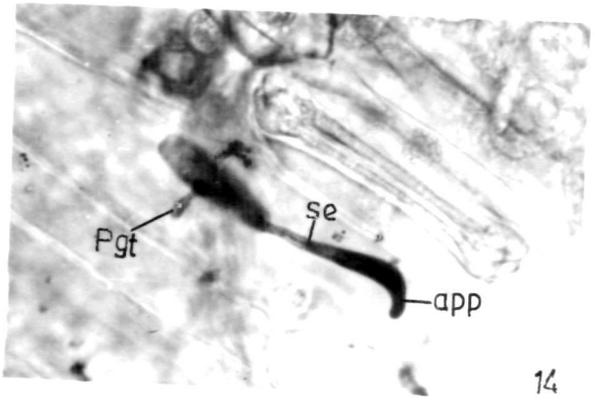
14 Mature appressoria (app), septa (se), Primary ger tube (Pgt) and conidia (C), (12 hr after inoculation) x 200.

15 Primary haustoria (Pha), (24 hr after inoculation) x 400.

16 Digitate processes (DP), elongated secondary hyphae (ESH), (48 hr after inoculation) x 400.

17 Secondary haustoria (Sha), (72 hr after inoculation) x 400

18 Weak mycellium and secondary penetration sites (SPS), (96 hr after inoculation) x 400.



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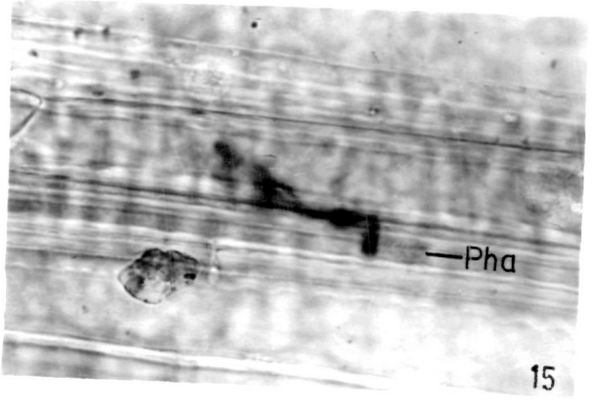

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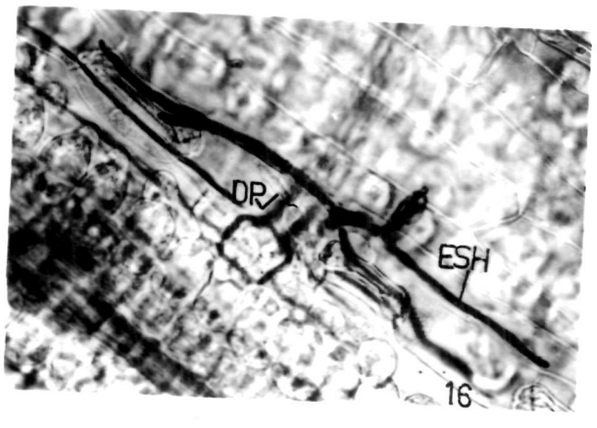

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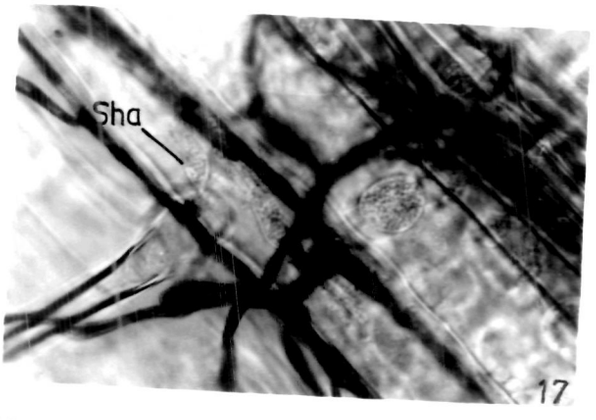

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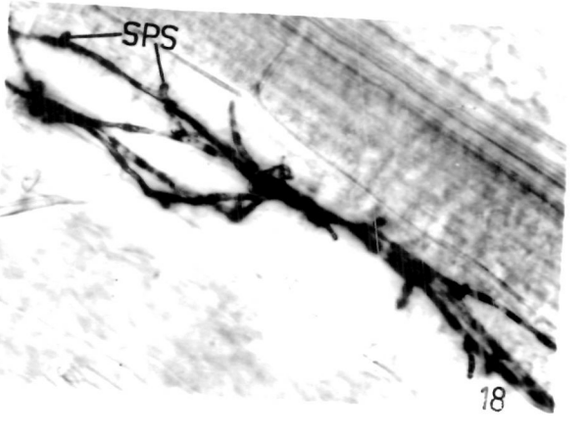


18

local - Egt pathotype 4, germinated conidia produced appressoria terminally on germ tubes within 12 hr of inoculation. Mature appressorium was subtended by the septum formed across the germ tube. Appressoria were more or less foot shaped, closely appressed to the host surface and produced primarily on periclinal host cell wall (Fig.1). Primary penetration of host surface was direct and infection peg formed on undersurface of the appressorium penetrated through host cuticle and outer epidermal cell wall. Mostly penetration was effected within 24 hr after inoculation through the penetration holes with smooth margins (Fig.2). Occasionally epidermal cell wall thickened and formed a collar like structure (papilla) around the infection peg.(Fig.2). An oval shaped primary haustorial body developed within the epidermal cell at the tip of penetration hypha (Fig.2). Between 24 and 72 hr after inoculation the haustorium increased in size and digitate, elongated finger like appendages developed from both ends. Concomitant with haustorial development, formation of elongated secondary hyphae from appressorium on the host surface was also observed within 48 hr after inoculation (Fig.3). A mycelial weft developed on leaf surface with continued growth and branching of elongated secondary hyphae. Growth of mycelial weft was accompanied by secondary penetrations of the leaf surface and subsequent production of secondary haustoria within 48-72 hr after inoculation (Fig.4). In stained material blue "haloes" were often observed 6 days after inoculation along the sites of subsequent penetrations. Secondary penetrations were

effected through the penetration holes produced within "haloes" (Fig.5a,b).Conidiophore development sites were deeply stained, terminally swollen appendages on the mycelial weft within 48-72 hr after inoculation (Fig.3). These had swollen bases and produced conidial chains with terminally mature conidia in acropetal succession within 6 days after inoculation (Fig.6).

A similar sequence of events was observed in the infection of other susceptible host genotypes viz CS/Hope, Transec, Kavkaz Amigo, HS 240 and HS 295 when inoculated with Egt pathotype 4. However, development of sporulating colonies in genotypes Amigo, HS 240 and HS 295 was observed after seven days of inoculation as compared to six days after inoculation in genotypes Hope, Transec and Kavkaz as well as Agra local. However, marked differences were observed in latent period, colony size and sporulation index, in the susceptible genotypes as compared to Agra local, (Described, under the quantitative aspects of resistance components).

B. Incompatible Host-Parasite Combination: Various levels of resistance mechanisms were observed in resistant genotypes Norka x Cc^8, TP 114, Sappo and Kronjuwel as compared to susceptible genotype Agra local and other susceptible genotypes, when inoculated with Egt pathotype 4. Conidial germination was poor in resistant genotypes, particularly in Norka x Cc^8 and Sappo, as compared to Agra local. Normal germ-tube growth was observed on all resistant genotypes except Sappo, showing meagre germ-tube growth (Fig.7). In genotypes Norka x Cc^8, and

Sappo delayed development of mature appressorium was recorded as compared to susceptible genotypes. In resistant genotypes, mature appressorium appeared 24 hr after inoculation (Fig.8) as compared to 12 hr in resistant genotypes TP 114 and Kronjuwel (Fig.14,10) and susceptible genotypes. In Kronjuwel and Sappo, germ tubes appeared weak, shrivelled and distorted after 24-72 hr of inoculation (Fig.11,12 and 8,9, respectively). Appressoria appeared reduced in size, flattened and deformed. In genotype Kronjuwel, mycelial growth ceased after papilla formation (Fig.11). In genotypes Norka x Cc^8, Sappo and Kronjuwel, mesophyll cells underlying the infection court assumed loose disorganised appearance within 48 hr after inoculation (Fig.13). In genotypes Norka x Cc^8 and Sappo, further development of pathogenic unit was not observed after appressorium formation. Penetration of host cell wall, primary haustorial development, elongation of secondary hyphae, secondary penetrations, development of less secondary haustoria and restricted growth of mycelial weft were observed in genotype TP 114 (Fig. 15, Fig.16, Fig. 17). No primary or secondary haustoria were observed in this genotype after 96 hr of inoculation (Fig.18). Although formation of conidiophores initials was observed in this genotype yet no conidiophores or conidia developed.

The response of pathogenic units during penetration and development on test genotypes along with universal susceptible cv Agra local, in terms of conidial germination, germ tube length, appressorial production, elongated secondary hyphae, haustorial formation and size were recorded and the results are

given in Table 14.

Conidial germination: Conidial germination recorded 12 hr after inoculation was at par in susceptible cv. and HS 240 and significantly higher in genotypes CS/Hope, Transec, Kavkaz and Amigo as compared to susceptible cv. Agra local. In resistant genotypes Norka x Cc^8, TP 114, Sappo and Kronjuwel, conidial germination was significantly less as compared to susceptible cv Agra local, and it was markedly less in genotype Norka x Cc^8 (13%) as compared to other resistant genotypes.

Germ-tube length: Germ-tube elongation recorded 24 hr after inoculation was significantly less in all susceptible genotypes except in Kavkaz and Amigo. In Kavkaz, germ-tube elongation was significantly more and in genotype Amigo, it was at par with susceptible check Agra local. In resistant genotypes Norka x Cc^8, TP 114 and Kronjuwel it was significantly less, while in genotype Sappo it was at par with the susceptible check.

Appressorial production: All the susceptible genotypes except var HS 240 showed significantly higher appressorial formation after 24 hr of inoculation. No significant differences were observed between HS 240 and Agra local. In all resistant genotypes, appressorial production was significantly less as compared to susceptible check Agra Local, least being in Norka x Cc^8, followed by Sappo and Kronjuwel.

Elongated secondary hypha (ESH/App,%): In susceptible genotypes all mature appressoria formed elongated secondary hyphae. However, compared to susceptible genotypes a restriction in

Table 14. Conidial germination, germ tube elongation, appressorial formation, elongated secondary hypha production and haustorial development by Erysiphe graminis f.sp. tritici pathotype 4 on wheat genotypes.

Host genotype	Gene(s) and (Reaction)	% Conidial germination 12 hr*	Germ tube length (μm) 24 hr*	% Appressorial production 24 hr*	ESH/ App. (%) 48 hr*	% Primary haustorial production 24 hr*	% Primary haustorial production 48hr*	Mean primary haustorial size Lengthx Breadth (μm) 72 hr*	Mean secondary haustoria/ Unit 72 hr*
Agra local	None (S)	86 (68.03)	52.2	85 (67.21)	100	80	85	107.3x13.5	5.1
Norka x Cc[8]	Pm1 (R)	13 (21.13)	36.8	13 (21.13)	-	-	-	-	-
CS/Hope	pm5 (S)	93 (74.66)	38.9	90 (71.57)	100	70	85	107.3x13.5	5.4
TP 114	Pm6 (R)	62 (51.94)	39.9	60 (50.77)	80	25	15	68.8x13.5	0.6
Transec	Pm7 (S)	92 (73.57)	39.4	90 (71.57)	100	75	82	118.4x13.5	5.9
Kavkaz	Pm8 (S)	96 (78.46)	57.3	96 (78.46)	100	78	80	99.9x13.5	5.1
Amigo	Pm17 (S)	92 (73.57)	50.4	90 (71.57)	100	85	90	108x13.5	5.8
Sappo	Pm2+4b (R)	50 (45.00)	50.0	34 (35.67)	-	-	-	-	-
Kronjuwel	Pm4b+8 (R)	48 (43.85)	34.5	48 (43.85)	-	-	-	-	-
HS 240	? (S)	89 (70.63)	46.7	88 (69.33)	100	80	80	118.4x13.5	6.0
HS 295	? (S)	91 (72.54)	45.6	90 (71.57)	100	75	85	107.3x13.5	6.1
S.E.		±2.3	± 1.8	±1.3					
CD (5%)		4.8	3.7	2.7					

Angular transformed values in parentheses
* after inoculation; R = Resistant; S = Susceptible

ESH formation occured in resistant genotype TP 114 while on other resistant genotypes Norka x Cc^8, Sappo and Kronjuwel ESH formation was absent.

Primary haustorial production and its size: Maximum production of primary haustoria in susceptible genotypes observed 48 hr after inoculation was 80-90%. Among susceptible genotypes maximum haustorial production was observed in Amigo, followed by HS 295 and CS/Hope. In genotype TP 114 primary haustorial production reduced with time, maximum being 25%, 15% and nil at 24,48 and 96 hr, respectively after inoculation.

Size of primary haustorium was recorded after 72 hr of inoculation. Length was more in genotypes Transec and HS 240, while least in resistant genotype TP 114. Interestingly, breadth was same in all observed cases.

Secondary pathogenic unit: All susceptible genotypes developed more or less same mumber of secondary haustoria, 72 hr after inoculation, while these were less in resistant genotype TP 114. No secondary haustorial production was observed in genotypes Norka x Cc^8, Sappo and Kronjuwel.

In resistant genotypes, viz. Norka x Cc^8, Sappo and Kronjuwel development of haustorium and elongated secondary hyphae was not observed. Although the resistant genotype TP 114 showed production of haustoria and ESH, yet development of observable colonies was not seen in this genotype. However, development of well developed colonies were observed in susceptible genotypes. In these stocks further observation

Table 15. Incubation period, latent period, colony number/cm^2, colony size, sporulation index and sporulation capacity of *E. graminis* f.sp. *tritici* pathotype 4 on primary leaves of susceptible and resistant wheat genotypes.

Genotype/cv	Incubation period (days)	Latent period (days)	Colonies/ cm^2	Colony size (mm^2)	Sporulation index	Sporulation capacity (%)
Agra local	4	6	32.4	0.8	87.6	100.0
Hope	4	6	11.9	0.4	24.9	10.4
Transec	4	6	16.6	0.7	58.0	33.9
Kavkaz	4	6	22.1	0.3	33.6	26.2
Amigo	5	7	20.3	0.4	26.4	18.9
HS 240	5	7	18.9	0.6	73.3	48.8
HS 295	5	7	20.2	0.5	44.4	31.6
S.E.	-	-	±3.1	±0.1	±7.8	-
C D (5%)	-	-	6.6	0.1	16.7	-

were recorded on components of rate reducing resistance i.e. incubation period, latent period, colony number, colony size, sporulation index and sporulation capacity and the results are given below and in Table 15.

Incubation period: First macroscopically visible symptoms appeared four days after inoculation in genotypes Kavkaz, Transec and CS/Hope and susceptible check Agra local, whereas in genotypes Amigo, HS 240 and HS 295, these symptoms were observed on 5th day after inoculation.

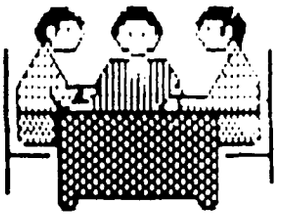
Latent period: A trend similar to incubation period was observed for latent period on different genotypes.

Colony number/cm^2 of leaf: All test genotypes developed significantly less number of colonies as compared to susceptible check Agra local. Genotype CS/Hope exhibited least number of colonies followed by Transec and HS 240.

Colony size: Colony development was significantly reduced in all test genotypes except Transec as compared to susceptible check Agra local. In Transec colony size was at par with Agra local.

Sporulation index: Conidial production in all genotypes, except HS 240, was significantly less as compared to susceptible check Agra local. Minimum sporulation was observed in CS/Hope followed by Amigo.

Sporulation capacity: Test genotypes showed low sporulation capacity than susceptible check Agra Local. Least sporulation capacity was observed in genotype CS/Hope followed by Amigo, Kavkaz, Transec, HS 240 and HS 295.



DISCUSSION

5 DISCUSSION

Powdery mildew of wheat caused by Erysiphe graminis f.sp. tritici can be effectively managed with the cultivation of resistant varieties. However, life cycle of the pathogen is such that evolution of new virulences is enevitable, rendering the resistant varieties susceptible after a short period of their commercial exploitation. Therefore, it is necessary to continuously monitor the virulence spectrum of pathogen populations in an epidemiologically important area. Thus, breeding for powdery mildew resistance needs to be a continuous process in accordance with the prevailing virulences. It requires identification of diverse sources of resistance and their characterization in terms of diversity and postulation of resistance gene(s). It is helpful in efficient and effective utilization of such resistance sources in the breeding programme and their deployment for effective management of the disease (Moseman et al.,1984; Heun and Fischbeck,1987a,b;Leath and Heun, 1990). The knowledge about components of race-specific resistance is helpful in properly understanding the stage at which resistance mechanism become operative and sequence of potential barrier to infection (Nashaat and Moore, 1991). The results obtained on these aspects are being discussed under the following headings:

1. Physiologic specialization
2. Postulation of powdery mildew genes in wheat genotypes
3. Components of resistance.

5.1 Physiological specialization in E. graminis f.sp. tritici

The work on pathogenic variation in E. graminis tritici started with the identification of distinct races from Australian and American powdery mildew populations (Waterhouse, 1930 and Mains, 1933). Subsequently, physiological specialization was studied in various European, American and Australian countries (Moseman, 1966; Wolfe and Schwarzbach, 1978) and in India (Arya, 1962; Prabhu and Prasada, 1963). However, all the studies were based on differential cultivars selected on emperical basis, without knowing their genetic constitution. Moreover, differential sets used were not uniform, so results from various countries were not comparable. In 1969, Briggle developed a set of near isogenic lines, in the background of cv Chancellor, having single genes for resistance viz. Pm1, Pm2, Pm3a, Pm3b, Pm3c, Pm4a, and Pm(Ma). Subsequently, genes pm5, Pm6, Pm7 and Pm8 were identified (McIntosh, 1988). Thereafter, pathogenic variation in European (Szunics and Szunics, 1987; Kwiatkowski, 1986), Asian (Wu et al., 1983; Li and Huang, 1991; Oku et al., 1987) and American (Menzies and McNeil, 1986; Royer et al.,1984) countries was studied, using these differential cultivars, supplemented with resistant cultivars of local importance. In India, Singh and Sood (1977) worked out physiological specialization of single conidial isolates collected from Kangra valley based on their reaction on powdery mildew resistant genes Pm1 through Pm4a and Pm(Ma). Subsequently, virulence spectrum of conidial and ascosporic isolates, collected from Himachal Pradesh and Punjab, was studied using differential set having genes Pm1 to

Pm8 and Pm(Ma) (Sharma et al., 1990; Sharma and Singh, 1990c Kanwar, 1993). The present studies were carried out using the same differential set, sulpplemented with lines having gene(s) Pm6 (Timgalin), Pm2+4b (Sappo) and Pm4b+8 (Kronjuwel).

Fifty six single colony conidial and ascosporic isolates collected from different agroclimatic zones of Himachal Pradesh, were grouped into 30 distinct pathotypes. Twenty four pathotypes were identified from the conidial and six from the ascosporic isolates. One ascosporic and conidial isolate was identical. Among the conidial isolates pathotype 7 and 12 were the most prevalent pathotypes. Pathotype 7 attacking only one gene Pm(Ma), was the least virulent. Isolates with virulence on gene Pm(Ma) were widely prevalent during the years 1990-91, 1991-92 in Himachal Pradesh and in 1976-78 and 1980 in Pennysylvania, U.S.A. (Royer et al., 1984).

In the present studies about 60 percent virulences were identical with the virulences already identified in the state (Sharma and Singh, 1990c and Kanwar, 1993). Pathotype 1 with virulence on gene Pm3c and Pm(Ma) is identical to race 15 (Sharma and Singh, 1990c). Pathotypes 5,9, 10, 12, 16, 19,20 have the virulence spectrum identical to the pathotypes 6, 18, 37, 22, 38, 27 and 36 respectively (Kanwar, 1993) and pathotypes 1,14, 24, 27, are similar to the races 15, 13, 15, and 9 (Sharma and Singh, 1990c). Pathotypes 10, 12, 14 and 23 were virulent on 7 genes each. Pathotype 16 giving susceptible reaction on lines with genes Pm3a, Pm3b, pm5, Pm6 (Timgalin), Pm7, Pm8 and Pm(Ma) was the most virulent pa4hotype. It was detected among isolates

collected from Palampur and Jaisinghpur (Mid hill Zone) and is identical with pathotype 38 identified among the ascosporic isilates collected from Kukumseri (Kanwar, 1993). Sharma et al. (1991) detected a similar pathotype virulent on all these genes except Pm7 from Punjab. Among the cleistothecial pathotypes, pathotype 26 with virulence on genes Pm3c, pm5 and Pm(Ma) was the least virulent and it is identical to pathotype 27 identified among the cleistothecial isolates collected from Kukumseri (Kanwar, 1993) and isolates with similar virulence were reported from Pennysylvania, U.S.A. (Royer et al.,1984). Pathotype 25, attacking genes Pm3a, Pm3c, pm5, Pm6 (Timgalin), Pm7, Pm8 and Pm(Ma) was the most virulent pathotype and it is identical to race 9 (Sharma and Singh, 1990c). In the present studies generally, ascosporic isolates, attacking on an average 4.5 genes, are more virulent as compared to the conidial isolates attacking 2.5 genes.

In the present studies, no correlation could be established between the virulence of the isolates and their occurence in different agroclimatic zones of the State. Pathotype with extreme virulence were rare, as among the 56 isolates only 8.93% isolates were virulent on one gene and 3.57% on 8 genes. The isolates with virulence on 3-6 genes were more prevalent. The present observations with intermediate virulence are supported by earlier studies (Stojanovic and Ponos, 1990; Sharma and Singh, 1990c; Kanwar, 1993). However, in Sichuan province of China isolates with virulence on 7 and 8 genes were more frequent (Li and Huang, 1992) which may be explained on the basis

of fact that, in that area virulence on genes Pm1, Pm2, Pm6 was widely prevalent in addition to other genes, whereas, in the present studies no virulence was detected on genes Pm2, Pm4a and it was rare on genes Pm1 and Pm6 (TP 114). In Himachal Pradesh, most of the cultivated wheat varieties viz. CPAN 1796, VL 421, VL 616, HD 2380 and Sonalika are susceptible to the disease. However, virulences have been detected against most of the resistant genes indicating that virulences can arise and maintain themselves even in the absence of resistance genes in the host populations. This is well evidenced by the prevalence of virulences on alleles of gene Pm3 locus, Pm6 (Timgalin) and Pm7. Gene Pm7 has been transferred from chromosome 2R of Rosen rye to chromosome 4A of wheat (Driscoll and Jensen, 1965) and it had been effective in many areas of the world (Driscoll and Jensen, 1975; Leath and Murphy, 1985; Bennett, 1984). Gene Lr 25 for leaf rust resistance is always linked with gene Pm7 (McIntosh, 1988). Interestingly, no leaf rust isolate virulent on gene Lr 25 has been detected in India, whereas gene Pm7 is highly vulnerable to the prevailing virulencesin the country (Sharma et al., 1990; Sharma and Singh, 1990c;Kanwar, 1993).

An experiment was conducted at Palampur to determine the changes in virulence frequencies of E. graminis tritici population during different times in the season. Virulences on genes Pm1, Pm2, Pm4a, Pm6(TP 114) and on varieties with combination of genes Pm2+4b and Pm4b+8 was not detected throughout the season. Virulence on genes Pm3a, Pm6 (Timgalin), Pm3c and Pm(Ma) was detected throughout the season, but changes in

virulence frequency did not follow a set pattern. Virulences on genes Pm3b, Pm7 and Pm8 appeared late in the season.

In the previous studies conducted during the month of March and April 1986-87, 1990-91 and 1991-92 (Sharma and Singh, 1990d; Kanwar, 1993) no virulence was trapped on genes Pm1, Pm4a and Pm6 and it was rare on genes Pm2 and Pm3b (Kanwar, 1993). Sharma and Singh (1990a) and Kanwar (1993) reported that on cvs CPAN 1922, HS 240 and HPW 42 powdery mildew appeared late in the season. These cvs involve Orlando, Veery and Aurora, possessing gene Pm8 in their pedigree. Presence of gene Pm8 for powdery mildew resistance was further evidenced by postulation of gene Lr 26 for leaf rust resistance in these varieties (Plaha et al., 1993; Anon., 1992). Hence,the late appearance of disease on these varieties may be due to the late appearance of the virulences attacking gene Pm8. Virulence pattern in races of Puccinia graminis tritici originating near the barbery bushes were different as compared to the races collected later in the season (Roane et al., 1960). Similar results were reported in case of Puccinia coronata (Simon et al., 1979). However, in both the studies, the differences were observed in virulences of sexual and asexual populations of the pathogen. However, in the present case only asexual populations were studied at different periods of time, so the results are not comparable. In dry temperate zone of Himachal Pradesh, cleistothecia mature into ascospore which form the primary source of inoculum (Kapoor, 1988)
and the subsequent spread of disease is through conidia. The

change in virulence frequency with changes in time can be fruitfully worked out under these conditions.

Virulence structure of E. graminis f.sp. tritici populations in different areas of Himachal Pradesh was determined with the help of mobile nurseries (Wolfe and Schwarzbach, 1975). Virulence was frequent on genes Pm3a, Pm3c, pm5, Pm6 (Timgalin), Pm7, Pm8 and Pm(Ma). No virulence was trapped on genes Pm2, Pm4a and on cvs with combination of genes Pm2+4b, Pm4b+8 and it was rare on genes Pm1, Pm6 (TP 114). Genes Pm1, Pm2, Pm4a and Pm6 were found to be highly effective in Punjab and Himachal Pradesh (Saharan et al., 1981; Sharma and Singh, 1990b; Kanwar, 1993) and in many other countries (Negulexu et al., 1978; Li and Huang, 1992; Lesvoi et al., 1980; Linhares, 1986). Heun (1987) also reported that Sappo (Pm2+4b) and Kronjuwel (Pm4b+8) were effective against pathogen populations in parts of Federal Republic of German. Kronjuwel (Pm4b+8) have been found to be effective against virulences in United Kingdom, Netherland, Italy and in parts of Federal Republic of Germany (Limpert et al., 1987) and cvs with gene combination Pm2+4b in China (Lu, 1986; Li and Huang, 1992). However, matching virulences against genes Pm1, Pm2, Pm4a, Pm6, Pm2+4b, Pm4b+8 have been reported in several countries (Wolfe and Schwarzbach, 1978; Bennett, 1984; Menzies and MacNeill, 1986; Royar et al., 1984; Menzies et al., 1989; Heun, 1987; Streckeisen and Fried, 1985; Limpert et al., 1987). Cultivar Kronjuwel was cultivated in 10-15 percent area in United Kingdom which might have exerted selection pressure on the pathogen to evolve virulences matching this gene. For powdery

mildew of wheat and barley, Europe has been considered as one epidemiological unit (Wolfe and Schwarzbach, 1978; Limpert et al., 1987), hence, dispersal of conidia of strains virulent on Kronjuwel in other European countries must have led to detection of isolates virulent on these varieties. In contrast to present studies, wheat powdery mildew populations virulent on genes Pm2, Pm4b and Pm6 are quite frequent in European and American countries. In these countries cvs having resistance genes Pm2, Pm4b, and Pm6 are widely cultivated and used in the wheat breeding programme,aimed at developing powedery mildew resistant wheat varieties, whereas, these resistant genes havenot been exploited in India. In the present investigations,virulence on genes Pm3 locus, pm5, Pm7, Pm8 and Pm(Ma) are widely prevalent. Varieties like Sonalika, VL 421, CPAN 1796 and Kalyansona involving Hope and Siete Cerros in their pedigree, are widely grown in Himachal Pradesh. Hope and Siete Cerros possess gene pm5 for powdery mildew resistance (Zeven, 1976; Hovmoller, 1989). High frequency of virulence on gene pm5 may be due to selection pressure on the pathogen to evolve virulences against this gene. Sharma and Singh (1990c) reported that during the year 1986-87, only 4 percent siolates were virulent on gene Pm8. Subsequently, its frequency increased and 50 percent during the years 1990-91 and 1991-92 (Kanwar, 1993). During the last few years a number of varieties viz. HPW 42, HS 240, HS 277 resistant to leaf and yellow rust have been released for cultivation in the State. These varieties involve Veery, Aurora or Kavkaz having genes Lr 26 and Yr 9 for leaf rust and yellow rust resistance, respectively(Anonymous, 1992). Genes Lr 26 and Yr 9 have been

transferred from rye as a result of 1B/1R reciprocal translocation and are closely linked with powdery mildew resistance gene Pm8 (McIntosh, 1988). Cultivation of these varieties might have exerted selection pressure on the pathogen to evolve virulences for gene Pm8. Moreover, the isolate have also been collected from the research stations of University viz. Dhaulakuan, Kukumseri etc. where a lot of breeding material having genes Lr 26 and Yr 9 resistant to rusts is being grown. All this might have increased virulence on gene Pm8. Virulence structure of E. graminis tritici is greatly influenced by host genes (Wolfe and Schwarzbach, 1978; Limpert et al., 1987; Heun, 1987).

In the present studies genotypes with genes Pm2, Pm4a, Pm6(TP 114), Pm2+4b, Pm4b+8 were found to be effective against the virulences prevailing in the State. However, matching virulences against all known powdery mildew resistance genes Pm1 through Pm8 and Pm(Ma) have been detected in various parts of the world (Wolfe and Schwarzbach, 1978; Bennett, 1984) and on Pm17 at Palampur (Personal observation). However, cvs with combination of different genes are expected to impart stable and durable resistance against the populations of the pathogen. The comparison of actual and theoretical virulence frequencies on different combinations of host genes revealed that combination of genes Pm2, Pm4a, Pm6(TP 114), Pm2+4b and Pm4b+8 with all the genes studied; Pm1 with Pm3a, Pm3b with Pm6(TP 114) and Pm6(TP 114) with Pm8 would be effective against the prevalent virulences. The present results are in confirmatity with the previous studies suggesting effectiveness of gene combinations

Pm1 with Pm2, Pm4a with Pm6, Pm2 with Pm4a, Pm2 with Pm6, Pm2 with Pm8, Pm4a with Pm8, Pm6 with Pm8 and combinations of genes Pm1 with Pm2, Pm1 with Pm6, Pm2 with Pm4a, Pm2 with Pm8, Pm4 with Pm6, Pm4 with Pm8, Pm6 with Pm8, Pm7 with Pm8 against the virulences studied (Sharma and Singh, 1990c; Kanwar, 1993). However, the results are not consistent over the years as some of the gene combinations expected to be effective in the previous studies were found ineffective. Hence, it is necessary to analyse a large number of isolates differing temporally and spatially to suggest suitable gene combinations, with durable resistance. Moreover, matching virulences on various genotypes having combination of genes viz. Pm2+4b, Pm2+4b+6, Pm4b+8 and Pm2+6 have been reported from Europe (Streckeisen and Fried, 1985; Heun, 1987; Hovmoller, 1987; Limpert et al., 1987). However, combinations of genes whose resistance has been matched by the virulent strains have been reported to impart rate reducing resistance (Nass et al., 1981; Royer et al., 1984; Sharma et al., 1991), resulting in less disease development.

5.2 Characterization of Resistance Genes

The principle underlying the genetic control of plant diseases as explained by the work of Flor (1942) with Flax-rust was applied in the present study. Usefulness of this technique to identify powdery mildew resistance genes in wheat cvs has already been demonstrated (Sebastian et al., 1983; Moseman et al., 1984; Gerechter et al., 1984; Heun and Fischbeck, 1987; Leath and Heun, 1990; Lutz et al., 1992; Sharma et al., 1993 and Kanwar, 1993). In the present investigations race-specific powdery mildew resistanc

genes were identified in 140 wheat genotypes based on their differential reaction to 8 pathotypes of Egt with defined virulence spectrum. The results are being discussed in the light of pedigree relationships.

Resistance of genotype 7 ARSN-68, the only genotype in group I, was attributed to the genes Pm3a, Pm8 and some other gene(s) which could not be postulated with the present pathotypes. pedigree of 7ARSN-68 involved Aurora and Alondra, which are known to possess gene Pm8 (McIntosh, 1988). However, origin of gene Pm3a and the other unidentified genes could not be ascertained.

Resistance of genotypes DWR 39, DWR 168, HD 2501, HUW 284, HW 135, MACS 2496, RRM-61, Shimla local, VL 672 and WOM 49 was attributed to gene Pm3c. The pedigree of genotypes DWR 162, MACS 2496 and WOM 49 has Veery having gene Pm8 (Bennett, 1984). However, gene Pm8 could not be postulated using the pathotypes available. The phenomenon of presence of the short arm of rye chromosome 1R and non-expression of the presumed resistance gene has already been described by Friebe et al. (1989).

The resistance in genotype 18 EDYT-8, in group III, was attributed to gene Pm3c and some other genes which could not be identified with the available pathotypes. 18 EDYT-8 is a T.durum genotype and presence of gene Pm3c in it could not be explained.

Resistance in two genotypes 10 ESWYT-17 and RL-116 was attributed to the genes Pm3c and Pm8. Genotype RL-116 is a stable wheat-like triticale-wheat derivative obtained from the cross CPAN 1922 x TL 1210 . CPAN 1922 involves orlando in its

pedigree having gene Yr 9 for yellow rust resistance (McIntosh, 1988) and TL 1210 is a triticale line involving rye genome. Gene Pm8 has been transferred from rye by 1B/1R translocation. Hence, origin of gene Pm8 in RL-116 may be from Orlando or TL 1210. Leaf rust resistance in RL-116 was attributed to gene Lr 26 and some other unidentified gene(s) (Plaha et al., 1993) and gene Lr 26 is closely linked with gene Pm8 (McIntosh, 1988). However, presence of gene Pm3c, could not be ascertained. Pedigree relations of 10 ESWYT-17 do not indicate the postulation of gene Pm8.

Resistance of genotypes C-32, C-34 and 22 IBWSN-116 was postulated to be due to gene pm5. Pedigree records show the involvement of Hope and Siete Cerros in these stocks, both of which are known to possess gene pm5 (Hovmoller, 1988; Lutz et al., 1992). Additionally, pedigree of genotype C-32 and 22 IBWSN-116 involve Alondra and Aurora having gene Pm8. (Bennett, 1984; McIntosh, 1988). Resistance in genotypes EIGSN-90, 5HTSN-1, 5HTSN-11, PBW 154 and RNS-9 (Group VI) was attributed to gene pm5 and some other genes which could not be postulated with the available pathotypes. Pedigree relationships of 5 HTSN-1 and 5 HTSN-11 shows the involvement of Siete Cerros, Hope, Kavkaz, and Veery, thereby indicating the presence of gene pm5 and Pm8. However, these genotypes showed, heterogeneity of reaction to pathotypes virulent/avirulent on gene Pm8 which may be due to the missing translocations, as has been confirmed with some other IBL. 1RS carrying wheat cvs using cytological techniques (Lukaszewski, 1992) or due to non-expression of gene Pm8 even the presence of short arm of rye chromosome 1B.1R

(Friebe et al., 1989).

Resistance genes pm5, Pm8 and some other unidentified gene(s) were postulated in two genotypes C-36 and C-44 in group VII. In genotype C-36 and C-44, pedigree analysis of both the varieties reveal the presence of Hope and Siete Cerros through the involvement of Kalyansona and Hope in them. However, involvement of parent having gene Pm8 could not be ascertained, hence the presence of gene Pm8 could not be explained.

Resistance in thirty genotypes in group VIII, was attributed to the gene Pm8. Pedigree relations of genotypes 7ARSN-36, 7ARSN-72, C-11, C-12, C-38, C-43, 7DSN-53, 2HEWSN-35, 2HEWSN-134, 2HEWSN-193, 2HEWSN-213, 19ISEPTON-25, 19ISEPTON-30, K 8806, RL-117, RL-118, RL-122, RL-124, RL-126, RL-127, RL-132, RRM-88, VL 711 and WOL 12, reveal the involvement of parents Alondra, Aurora, Kavkaz, Veery, Ures and/or Orlando possessing powdery mildew resistance gene Pm8 (Bennett, 1984; McIntosh, 1988). Hence, pedigree relations of these genotypes confirm the postulation of gene Pm8. The pedigree of genotypes 4HCWSN-4, ISWRN-93 is not known. Genotypes RL-117, RL-118, RL-122, RL-124, RL-126, RL-127 and RL-132 are hexaploid triticale x bread wheat derivatives having identifiable rye traits. These genotypes showed adult plant resistance to leaf rust, yellow rust and powdery mildew and seedling resistance to leaf rust which was found to be due to gene Lr 26 (Plaha et al., 1993), confirming the presence of gene Pm8, as leaf rust resistance gene Lr 26 and powdery mildew resistance gene Pm8 are closely linked (Bennett, 1984).

Resistance in genotypes C-39 and EIGSN-89 was postulated to be due to gene Pm8 and some other gene(s) which could not be

identified with the available pathotypes. Pedigree relations of genotype C-39 shows the involvement of Kavkaz possessing gene Pm8. However, records of pedigree of EIGSN-99 are not available.

Forty four genotypes in group X showed resistant reaction to one or two pathotypes. However, using infection-type matching technique resistance in these genotypes could not be attributed to any of the known powdery mildew resistance gene(s). Pedigree records of genotypes AlDRM-64, 7 ARSN-85, C-2, C-17, C-26, C-27, C-28, C-29, C-31, DWR-162, 10 ESWYT-22, HD 2546, 2 HEWSN-105, 2HEWSN-175, 2HEWSN-178, 2HEWSN-196, 2HEWSN-214, HS 295, 5HTSN-22, HUW 315, 22 IBWSN-86, 22 IBWSN-290, 19ISEPTON-7, 19 ISEPTON-9, 19ISEPTON-17, 19ISEPTON-37, PBW 316 and RL-134 show the involvement of Alondra, Kavkaz, Veery, Ures, Orlando carrying gene Pm8. However, gene Pm8 could not be depicted with the present pathotypes. This may be explained on the basis of phenomenon of the presence of the short arm of chromosome IR and non-expression of the presumed resistance gene Pm8 on 1RS as has been reported in varieties Olymp, Heinrich and Florid (Friebe, 1989) or it may be due to the missing translocations as have been confirmed cytotologically in some 1BL.1RS carrying cultivars (Lukaszawski, 1992).

Two genotypes RL-22 and WH 569 in group XI showed low infection types to all the eight pathotypes of E. graminis f.sp. tritici. Line RL-22 has been derieved from intergeneric crosses of wheat with Triticale, and genes Pm7 and Pm8 have been derived from rye (Moseman et al., 1984). However, RL-22 gave low

infection types with the cultures virulent on genes Pm7 and Pm8 indicating the presence of additional gene(s). RL-22 has shown consistent resistance to powdery mildew in field and laboratory conditions (Plaha, 1986; Kanwar, 1993). However, it showed differential reaction to leaf rust races avirulent/virulent on gene Lr 26 (Kanwar, 1993), indicating that gene Lr 26 is responsible for leaf rust resistance. Genes Lr 26 and Pm8, present on short arm of chromosome 1R as result of 1B/1R translocation, are closely linked (McIntosh, 1988; Bennett, 1984). Hence, detection of gene Lr 26 indicates the presence of gene Pm8. However, resistance of RL-22 to pathotypes virulent on gene Pm8 infers the involvement of some additional gene(s). Plaha (1986) reported on the basis of chromosome morphology, morphological markers and C-banding, substitution of chromosome 6R of rye in genotype RL-22 and some unidentified powdery mildew resistant genes are reported to be located on this chromosome (Miller, 1984).

Thirty eight genotypes in group XII, were susceptible to all the available cultures of powdery mildew. Therefore, presence of genes conferring resistance, could not be postulated with the available pathotypes. Pedigree of genotypes C-4, C-7, C-19, C-33, C-49, C-50, 7DSN-2, 2HEWSN-195, HUW 206, 22 IBWSN-195, 22 IBWSN-149, 5 KBSN-50, PBW 320 and WOM 35 revealed the involvement of lines Aurora, Veery and/or Kavkaz, which are known to have Pm8, but it could not be detected due to non differential behaviour of these cultivars against available pathotypes.

3.3 Components of Resistance:

In the present investigations, components of resistance to Erysiphe graminis f.sp. tritici (Egt) pathotype 4 were worked out in eleven genotypes, comprising of near-isogenic lines having powdery mildew resistance genes Pm1, pm5 and cvs with genes Pm6, Pm7, Pm8, Pm17, gene combinations Pm2+4b and Pm4b+8 and varieties HS 240 and HS 295,to understand mechanisms of resistance and sequence of barriers to fungal penetration and development. Resistance mechanism in E. graminis-wheat and barley systems have already been worked out (Ghemawat, 1969; Hyde and Colhoun, 1975; Wu et al., 1985; Iliev, 1989; Wright and Heale, 1988; Donchev and Iliev, 1987; Nashaat and Moore, 1991). However, mechanisms of resistance in genotypes with powdery mildew resistance genes Pm7, Pm8, Pm17, Pm2+4b and Pm4b+8 and variety HS 295 have been worked out for the first time in the present investigations.

Conidial germination was significantly low in the resistant genotypes Norka x Cc^{8}, Sappo, Kronjuwel and TP 114 as compared to the susceptible check Agra local. The present findings are in agreement with the results of Donchev and Iliev (1987), who reported the highest and lowest spore germination in Sadovska Ranozreinka 4 and Kenya page, the least and the most resistant varieties, respectively.

All the germinated conidia produced appressoria in resistant as well as in susceptible genotypes. However, in resistant genotypes, germ tubes were weak, shrivelled and distorted, and appressoria were reduced in size, flattened and deformed. The germ tube length was significantly less in all the resistant genotypes

as compared to susceptible check Agra local, however, in cv Sappo germ tube length was statistically at par with the susceptible check. This may be due to cutical thickness or production of some biochemical substance in resistant genotypes inhibiting to the conidial germination and appressorial formation. Conidial germination in resistant genotypes is supported by the results of Donchev and Iliev (1987), attributing the resistance in genotypes 5517AS-5-IR-3 and Sadovska to late and slow spore germination. All the appressoria produced elongated secondary hyphae (ESH) in susceptible cvs indicating the establishment of functional relationship of Egt pathotype 4 in all these genotypes. In genotype TP 114, 80% appressoria produced ESH, however, mycelial weft showed restricted growth. In resistant genotypes Norka x Cc^8, Sappo and Kronjuwel, no ESH formation was observed and mesophyll cells underlying the infection court assumed loose and disorganised appearance indicating that in these genotypes, resistance mechanism became operative at the cuticular level, before the establishment of functional relationship with the host. Such type of resistance expressed by necrosis of mesophyll cells underlying infection court has also been reported in P2/Pm2 (Ulka x Cc^8) combination (Hyde and Colhoun, 1975). There was no formation of primary or secondary haustoria in these genotypes even after 96 hr after inoculation. In genotype Kronjuwel, papilla formation was observed underneath the appressorial lobe which might have resulted into cessasion of fungal growth as has been reported by Nashaat and Moore (1991) who found papilla mediated powdery

mildew resistance in accessions of T. araraticum and T. timopheevii. Resistance in line T. araraticum was attributed to epidermal hypersensitivity (Nashaat and Moore, 1991). In genotype TP 114 primary and secondary haustoria, and ESH were formed. However, size of primary haustoria was markedly small, and the number of secondary haustoria/pathogen unit were very less. The haustoria became disorganised and aborted with passage of time and no haustoria was observed after 96 hr of inoculation. The present results are in agreement with the results of Hyde and Colhoun, (1975) attributing the resistance in lines having genes Pm3a, Pm3b, Pm3c, Pm4a and Mle to the necrosis of epidermal and mesophyll cells and formation of low number of elongated secondary hyphae. Iliev (1989) attributed adult plant resistance in some wheat varieties to the production of low number of elongated secondary hyphae.

In susceptible genotypes, viz. CS/Hope, Transec, Kavkaz, Amigo, HS 240, HS 295 and Agra local, 82-90 percent pathogenic units formed 100-118 μm long primary haustoria, 72 hr after inoculation, whereas in resistant genotype TP 114, only 15 percent pathogenic units formed only 68.8 μm long primary haustoria. Moreover, in contrast to 0.3 secondary haustoria/pathogenic unit in TP 114, 5-6 secondary haustoria/ pathogenic unit were observed in the susceptible genotypes and no haustoria was observed in the former 96 hr after inoculation. The present results are in agreement to Wu et al. (1985) who attributed resistance in 16 wheat varieties to delay

in haustorial development, reduction in number and size of haustoria and abortion of haustoria and similarly, Haywood and Ellingboe (1979) reported that in compatible host-parasite Px/Pmx combinations 57% parasitic units formed 35-55 μm long haustoria within 30 hr after inoculation, whereas in incompatible host-parasite combinations P1a/Pm1a, P2a/Pm2a, P3a/Pm3a and P4a/Pm4a only 15, 16, 18 and 3 percent haustoria, respectively attained same size in 30 hrs.

Genotypes Norka x Cc^8, Sappo, Kronjuwel and TP 114 did not develop visible colonies, implying that resistance mechanism operative in these genotypes is capable of inactivating the initial inoculum. It shows that resistance is governed by major gene(s). According to Flor,s (1942) gene for-gene hypothesis single major resistance genes are either qualitatively highly effective or totally ineffective. Evolution of new virulences overcome the effective genes and such defeated genes are replaced with the new race-specific major genes, which are generally readily and easily available in wheat rusts and powdery mildew system and defeated genes are sent in obilition. According to Nelson (1975) and Nelson (1978,1979) race-specific major and non race-specific minor genes for resistance are the expression of different actions of the same gene(s) in different genetic backgrounds or when confronted by different pathogen genotypes. According to Clifford (1975) polygenes are archaic major genes which have lost their large effect through the evolution of virulences in pathogen, but have a residual or ghost effect. The residual effect of defeated genes can be determined by studying components of rate

reducing resistance (Nass et al., 1981; Sharma et al.,1991a). In the present investigations, Egt pathotype 4 was virulent on lines/genotypes having powdery mildew resistance genes pm5, Pm7, Pm8, Pm17 and HS 240 involving Veery (possessing gene Pm8) and variety HS 295 having field resistance. Hence, it was considered worthwhile to work out components of rate reducing resistance viz, incubation period, latent period, colony number/area, colony size, sporulation index and sporulation capacity in these genotypes.

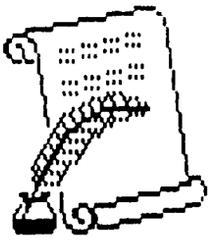
Genotypes Hope, Transec and HS 240 exhibited longer incubation and latent periods as compared to susceptible check Agra local. Similarly, longer latent period in Chul x Cc^8 having gene Pm3b against a virulent Egt culture (Sharma et al., 1991a) and longer incubation and latent periods in HS 240 (Kanwar, 1993) were responsible for rate reducing resistance. In leaf rust and powdery mildew-wheat systems rate reducing resistance, in many cvs has been attributed to longer incubation and latent periods (Sharma and Gupta, 1986; Sharma and Basandrai, 1991; Kanwar, 1993). All genotypes developed significantly less colonies/area, compared with susceptible check Agra local. The results are in confirmity with the results of Nass et al. (1981) and Sharma et al. (1991a), who attributed residual rate reducing resistance in isogenic-lines with genes Pm3a, Pm3b, Pm3c, Pm4 and Pm8 and a gene known as Michigan Amber (Pm(MA) to the development of less number of colonies/area, as compared to susceptible cvs Chancellor or Agra local. Similarly, rate reducing resistance to E. graminis f.sp.tritici in wheat genotypes was reported to be due to development of

less number of colonies/area (Sharma and Basandrai, 1992; Kanwar, 1993; Shaner, 1973). In all the genotypes except Transec and HS 240, colony size and sporulation was significantly low as compared to susceptible cv Agra local. In Transec and HS 240, colony size and sporulation, respectively, was at par with susceptible check Agra local. Similarly, rate reducing resistance in isogenic-lines having genes Pm3c, Pm4, Pm(Ma) was attributed to low sporulation and lines with genes Pm3a, Pm3b, Pm3c and Pm8 produced smaller colonies and less number of spores/colony as compared to susceptible cv Agra local (Sharma et al., 1991).

According to Vanderplank (1963, 1968), race-specific vertical resistance genes function against epidemic development by reducing the effective initial inoculum (x_o), available for disease onset and have no influence on disease increase by races having matching virulence genes. Such type of resistance was shown in genotypes Norka x Cc^8, TP 114, Sappo and Kronjuwel. In other genotypes, viz. CS/Hope, Transec, Kavkaz, Amigo, HS 240, HS 295, colony formation was as good as in universal susceptible Agra local. All these lines have known major genes for resistance which have been matched by Egt pathotype 4. However, residual effect of these defeated genes was exhibited by reduced disease development as is evident from the longer incubation, latent period, smaller colonies, less number of colonies/area and/or reduced sporulation. Genotypes CS/Hope, Transec and Kavkaz show race-specific resistance against pathotypes 11, 1 and 5 respectively (Table 6) and race specific resistance in Amigo has been reported by Lowry et al.(1984). It shows that race-

specific resistance may also reduce the apparent infection rate (r), when confronted with virulent races, a trait generally attributed to gene for horizontal resistance (Vanderplank, 1963, 1968). From the data it is clear that the genotypes carrying genes pm5, Pm7, Pm8, Pm17 and varieties HS 240 and HS 295 possess rate reducing resistance against Egt pathotype 4.

The genotypes with defeated powdery mildew resistance genes can be utilized more effectively than the susceptible components in the development of multiline varieties. Multiline variety develop less disease because of buffering effect of multilines. Supposing a component line, carrying a specific resistance gene in multiline variety was rendered susceptible by evolution of matching virulences, then the final disease severity will be less than expected disease severity as buffering effect of the multiline will be supplemented with rate reducing resistance of defeated gene.



SUMMARY

6 SUMMARY

Present studies were conducted on powdery mildew of wheat caused by Erysiphe graminis f.sp. tritici, to work out pathogenic specialization in the pathogen and to postulate race-specific resistance genes in Indian and exotic wheats using infection-type matching technique. In addition, attempts were made to understand the mechanisms of resistance and sequence of barriers to fungal penetration by studying components of resistance in some lines with known gene(s) for powdery mildew resistance and in commonly grown cultivars.

Fifty six isolates of the pathogen were collected from different agroclimatic zones of Himachal Pradesh. On the basis of their virulence pattern on seedlings of differential lines having genes Pm1 to Pm8 and Pm(Ma) and combination of genes Pm2+4b and Pm4b+8, the isolates were grouped into 29 distinct pathotypes. Twenty three pathotypes were of conidial and five of ascosporic origin, and one conidial and ascosporic pathotype was identical. Pathotype 16, attacking genes Pm3a, Pm3b, Pm3c, pm5, Pm6(Timgalin), Pm7, Pm8 and Pm(Ma) was the most virulent, whereas pathotype 7, attacking gene Pm(Ma) only, was least virulent. Pathotype 7 and 12 were the most prevalent pathotypes. Among ascosporic isolates, pathotype 25 attacking genes Pm3a, Pm3c, pm5, Pm6(Timgalin), Pm7 and Pm(Ma) was the most virulent, whereas, pathotype 26 with susceptible reaction on genes Pm3c, pm5, and Pm(Ma) was the least virulent pathotype. Pathotype 20 and 29 identified

among the conidial and ascosporic isolates, respectively, were identical.

Virulence structure of the pathogen populations was also studied using mobile nursery of the differential lines, at seven places located in different agroclimatic zones of Himachal Pradesh. Genes Pm2, Pm4a, Pm2+4b and Pm4b+8 were resistant to all the isolates and no virulence could be trapped on these genes in mobile nurseries. Virulence on genes Pm1 and Pm6(TP 114) was very rare. The changes in virulence frequency of powdery mildew populations were determined by exposing the seedlings of the differential lines at fortnightly interval starting from January, 1993 to June, 1993 at Palampur. No virulence was trapped on genes Pm1, Pm2, Pm4a, Pm6(TP 114), Pm2+4b and Pm4b+8 and virulence on genes Pm3b, Pm7, and Pm8 appeared late in the crop season. Virulence on genes Pm3a, Pm3c, pm5, Pm(Ma) was trapped throughout the season, but changes in virulence frequency did not follow a defined pattern. The relationship of virulence of isolates with frequency of their occurence, revealed that isolates with intermediate virulence were more prevalent.

The comparison of actual and theoretical virulence frequencies of all possible gene combinations was calculated from 56 isolates. Associated virulence for the combination of genes Pm2, Pm4a, Pm2+4b, Pm4b+8 with all the known genes and Pm1 with Pm6(TP 114), Pm3a with Pm6(TP 114), Pm1 with Pm3a, Pm1 with Pm3c, Pm1 with Pm6 (Timgalin) was found to be absent.

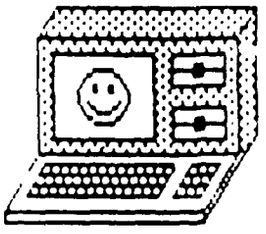
The combined virulence for all other gene combinations was quite prevalent.

Resistance genes were postulated in a set of 140 wheat genotypes. These were subjected to eight pathotypes of Egt with specific and diverse virulence. Based on infection-type matching technique, these genotypes were categorised into 12 groups, out of which resistance in first 9 groups was governed by genes Pm3c, pm5, Pm8, and some unidentified gene(s) individually or in combination. In majority of genotypes, resistance was attributed to the gene Pm8 individually or in combination with other genes. Forty three genotypes in group X, behaved differentially to the pathotypes, however, resistance in these genotypes could not be attributed to any known genes. Two genotypes RL-22 and WH 569 in group XI showed resistance to all the pathotypes, hence, resistance genes could not be postulated. Genotypes, in group XII, showed susceptible reaction to all the pathotypes indicating absence of resistance genes in these genotypes.

Components of resistance against E. graminis f.sp. tritici pathotype 4 were studied in 11 genotypes viz. Norka x Cc^8 (Pm1), Hope (pm5), TP 114 (Pm6), Transec (Pm7), Kavkaz (Pm8), Amigo (Pm17), Sappo (Pm2+4b), Kronjuwel (Pm4b+8), HS 240, HS 295 and Agra local. In genotypes Norka x Cc^8, Sappo and Kronjuwel, the resistance was manifested in the form of less and abnormal conidial germination and appressorial formation, and necrosis in the mesophyll tissues. Additionally,papilla formation beneath the infection court might have resulted into cessassion of fungal penetration in cv Kronjuwel. The growth of the

pathogen was completely inhibited after appressorial formation, as there was no production of haustoria and elongated secondary mycelium, indicating that resistance mechanism became operative at earlier stage. In genotype TP 114, conidial germination and appressorial formation was comparatively more and restricted ESH and haustorial formation was recorded. However, haustoria aborted with the passage of time.

Rest of the genotypes, developed visible colonies comparable with susceptible check Agra local. These genotypes showed rate reducing resistance in the form of longer incubation and latent period, production of less number of colonies/area, smaller colony size, reduction in sporulation and less sporulation capacity. Incubation period and latent period was more in Amigo, HS 240 and HS 295 as compared with the susceptible check Agra local. All genotypes exhibited less number of colonies/area of smaller size and reduced sporulation as compared with the susceptible check. However, in genotype Transec and variety HS 240, colony size and sporulation index, respectively were at par with Agra local. Sporulation capacity was less in all the genotypes as compared to susceptible check, least being in CS/Hope.



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