Characterization of Downy Mildew Resistance in Pearl Millet

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Submitted as ICRISAT Journal Article No.1871
Accepted for publication: 20 December 1996.

Abstract

Sixty-one pearl millet (*Pennisetum glaucum*) genotypes from diverse geographical origins identified as downy mildew (*Sclerospora graminicola*) resistant in several field tests at IAC and other locations in India and Africa were evaluated against five host-specific pathotypes and a field population of *S. graminicola*, referred to as pathotype 6. Pot-grown seedlings of all the pearl millet genotypes, were spray-inoculated with a sporangial suspension (1×10^5 sporangia ml^{-1}) of each pathotype in a replicated experiment in a glasshouse. Observations were recorded for latent period and disease incidence. Host resistance index (HRI) is suggested as a measure of resistance stability, and was calculated as $[1+(ab^{-1})]^{-1}$, where a = disease incidence (%), and b = latent period (day). There were highly significant ($P < 0.001$) effects of host genotype, pathotype, and their interaction on HRI. A number of genotypes were disease free to different pathotypes; IP 18292 was disease free from all six pathotypes. An additional 15 genotypes showed < 10% incidence to all six pathotypes. These genotypes with HRI values of 0.69 to 0.94 and are the best available sources of resistance. Eight genotypes (IP 5272-1, IP 18296, IP 18297, P 536-2, P 1564, P 2895-3, P 3281-1, and 700481-21-8) that showed differential reactions to six pathotypes can be utilized for characterizing *S. graminicola* isolates.

विभिन्न भौगोलिक मूल से एकत्रित अन्तर्राष्ट्रीय एशिया केन्द्र एवं भारत के दूसरे स्थानों पर अनेक क्षेत्र परीक्षणों में तुलासिता (स्के. ग्रेमिनिकोला) से रोगरोधी बाजरे के 61 जनपित्रैकों को पाँच पोषिता विशेष रोग प्ररूप व एक क्षेत्र जीव संख्या रोग प्ररूप 6 के विपरीत मूल्यांकित किया गया। गमलों में उगाये गये बाजरे के सब जनपित्रैकों को काँच घर में एक अभ्यावृत्ति प्रयोग में प्रत्येक रोग प्ररूप के बीजाणुधानी घोल (1×10^5 बीजाणुधानी मिली.$^{-1}$) से फुहारन संक्रमित किये गये। रोग आपात एवं गुप्तावधि के वर्णन अभिलिखित किये गये। पोषिता रोधिता द्यातांक रोधिता स्थायित्व के मापन के लिये काम में लिया गया जिसका कलन $[1+(ab^{-1})]^{-1}$, a = रोग आपात (%) व b = गुप्तावधि (दिन), द्वारा किया गया। पोषिता रोधिता द्यातांक पर पोषिता जनपित्रैक, रोग प्ररूप एवं उनकी मिथ क्रिया के उच्च सार्थक ($P< 0.001$) प्रभाव मिले। प्रत्येक जनपित्रैक विभिन्न रोग प्ररूपों से रोग मुक्त रहे। आई पी18292 ओं रोग प्ररूपों से रोग मुक्त रहा। एक अतिरिक्त जनपित्रैक 15 वे सब रोग प्ररूप पर से आपात 10% से अधिक दिखलाया। ये जनपित्रैक 0.69 - 0.94 एच आर आई के साथ रोधिता के सबसे अच्छे स्रोत रहे। आठ जनपित्रैक (आई.पी. 5272-1, आई पी 18296, आई पी 18297, पी 535-2, पी 1564, पी 2895-3 पी 3281-1 एवं 700481-21-8) ने सब रोग प्ररूपों से भिन्नक प्रतिक्रिया दर्शायी, स्के ग्रेमिनिकोला के अभिलक्षण के लिये काम में लिये जा सकते हैं।

Downy mildew of pearl millet [*Pennisetum glaucum* (L.) R. Br.], caused by *Sclerospora graminicola* (Sacc.) Schröt, is a highly destructive and widespread disease in Africa and Asia (Rachie

and Majmudar, 1980; Williams, 1983). Genetic resistance is the most effective and economic means of managing this disease. Therefore, breeding for downy mildew resistance continues to be a major component of pearl millet improvement programs. Genetically homogeneous F_1 hybrids, based on cytoplasmic male-sterile lines, are more susceptible to the disease than the heterogeneous open-pollinated varieties (Safeeulla, 1977). During the past 20 years, three commercial F_1 hybrids HB 3, BJ 104 and MBH 110, have succumbed to downy mildew within 5-7 years of wide-scale cultivation (AICPMIP, 1992).

Major downy mildew resistance genes have been identified and incorporated in the commercial hybrid parents, and hybrids (ICMH 451 and Pusa 23) made from such parents are under cultivation in India (Singh *et al.*, 1993). However, the resistance durability of these hybrids may be short-lived because of genetic uniformity of F_1 hybrids and high level of genetic variability in the pathogen populations. A number of downy mildew resistant lines have been identified from germplasm and breeding materials at ICRISAT Asia Center (IAC) and other locations in India and Africa (Singh, 1990, 1995). Resistance in several of these lines has been variable at different locations indicating the existence of variable populations of the pathogen (ICRISAT, 1989).

In India, at least three distinct populations of the pathogen (referred to as pathotypes) specific to HB 3, MBH 110 and 852 B are known (R. P. Thakur, unpublished). Recently, at IAC Patancheru, we have established five pathotypes from a field population of *S. graminicola* by selection through asexual generations, on seedlings of five pearl millet hybrids and inbred lines (Thakur and Rao, 1993).

In the present study, 61 pearl millet genotypes that showed downy mildew resistance in multilocational tests in India and Africa (Singh, 1990), were evaluated against six pathotypes, including path 6 (a field isolate as a control) to determine the pathotype-specific/nonspecific resistance in these genotypes. A part of the results was presented earlier (Thakur *et al.*, 1995).

Materials and Methods

Downy mildew resistant pearl millet lines. Sixty-one pearl millet genotypes, identified as resistant to downy mildew (< 10% mean incidence) in several screenings at IAC and a number of locations in India and western Africa over several years, were either germplasm accessions or selections thereof from Botswana (1), Burkina Faso (4), Central African Republic (1), Cameroon (7), Gambia (1), India (4), Mali (4), Niger (11), Nigeria (11), Senegal (5), South Africa (1), Sudan (6), Togo (2), and Zimbabwe (1). A number of these lines also had resistance to rust in India (Singh, 1990). Original seed of these lines were obtained from the Genetic Resources Division, IAC.

The pathotype isolates. The five pathotypes of *S. graminicola*: Path 1, Path 2, Path 3, Path 4, and Path 5, selected on specific host genotype for increased virulence from field populations of the pathogen (ICRISAT, 1993), were maintained on seedlings of their respective selection host genotypes, NHB 3, BJ 104, MBH 110, 852B and 700651, respectively, in polyacralic isolation chambers in a glasshouse. Pot-grown seedlings of each host genotype were spray-inoculated with a sporangial suspension (1×10^5 sporangia ml^{-1}) obtained from previously inoculated seedlings. The sixth pathotype, Path 6, an isolate from the IAC field population of the pathogen from a mix of downy mildew susceptible pearl millet genotypes 7042S and NHB 3, was included as a control.

Experiment 1. The experiment with 62 host genotypes, including 7042S a downy mildew susceptible control, and six pathotypes was conducted in a completely randomized design (CRD) with three replications in a glasshouse. Potting mixture of alfisol, sand, and farmyard manure (2:1:1 by vol) was autoclaved at 121.6° C for 60 min for two cycles and filled in 15 cm dia. plastic pots. Seeds were surface sterilized with 2% NaOCl (diluted from 5.25% clorox bleach, Oakland, Ca 94623, USA) for 5 min, washed thoroughly in distilled water and dried at the laboratory temperature (22-24° C) on blotting paper sheets. Twenty-five to 30 seedlings were raised in each pot and watered everyday. Seedlings at 2-leaf stage were spray-in-

oculated with an aqueous sporangial suspension (1 x 10^5 sporangia ml^{-1}) using a hand sprayer. Sporangia for each pathotype were obtained from its respective host seedlings and suspended in sterilized ice-cold water (4-5° C) to prevent zoospore release prior to inoculation. Inoculation was done in an inoculation chamber and the seedlings were retained in this chamber at 20° C at high relative humidity (> 95%) for 24h (Singh and Gopinath, 1985). Seedlings were then moved to a glasshouse at 25± 2° C. The experiment was divided into six sets, each with one pathotype x 62 host genotypes x three replications x two pots per replication.

Experiment 2. Fourteen genotypes that showed either clear differential reactions (incidence), or uniformly low or high incidence to the six pathotypes in all three replicates of experiment 1, were re-evaluated to confirm the results. Experimental details were similar to those described above, except that all treatments and replications were conducted at one time.

Disease assessment. Disease assessment was done for latent period (time in days from inoculation to appearance of downy mildew symptom with sporulation) and disease incidence (percentage of seedlings showing symptoms). Seedlings were examined daily, from the 4th to 15th day after inoculation for latent period. Downy mildew incidence was recorded 15 days after inoculation and per cent disease incidence was determined.

Host resistance index (HRI). In a systemic disease, like downy mildew of pearl millet, where large variation exists for disease incidence and latent period, incidence alone may not provide a representative measure of resistance in a host genotype. We suggest that 'host resistance', in such pathosystems, could be better described by HRI, which as a function of incidence and latent period is defined by the equation:

$$HRI = [1 + ab^{-1})]^{-1} \quad ...(1)$$

where a = incidence (%) and b = latent period (days). In equation (1), HRI = 1, if a = 0 which corresponds to a completely resistant line, and HRI <1, if a > 0, which corresponds to a less than completely resistant line. The degree of resistance will be directly proportional to the value of HRI.

Statistical analysis. Data on downy mildew incidence, latent period and HRI were subjected to analysis of variance (ANOVA) using the GENSTAT ANOVA procedure (GENSTAT, 1986) for a two-factorial (host genotype and pathotype) CRD for experiment 1 and for three factorial (experiment, host genotype and pathotype) CRD for experiment 2. The error mean squares for the data sets from the two experiments for 14 genotypes were examined for homogeneity using F-test of significance before pooling them for analysis. A correlation analysis was run to determine the relationships among incidence, latent period, and HRI. Host genotypes were grouped, based on centroid clustering analysis (SAS, 1985) of HRI values.

Results

Downy mildew incidence. The mean and range of downy mildew incidence of 61 genotypes for individual pathotypes varied from 9 to 20% and from 0 to 86%, respectively (Table 1). The number of genotypes in each of seven incidence classes was quite variable for individual pathotypes. The number of genotypes with 0 incidence varied from 3 for Path 6 to 17 for Path 5, and there were 13 genotypes each for Path 1 and Path 4, 16 for Path 3, and only 5 for Path 2. However, with the > 50% incidence class, the number of genotypes varied from 1 to 5 for the six pathotypes.

Of the 61 genotypes, IP 18292 showed 0 incidence and 7042S between 72-97% to all six pathotypes (Table 2). There were 18 other genotypes that had between 0 and 10% mean incidence to any one of the six pathotypes. Highly significant ($P < 0.001$) effects of host genotypes, pathotypes, and their interaction on incidence were observed (Table 3).

Latent period. The latent period was quite variable for different host genotype x pathotype combinations (Table 4). The mean latent period across six pathotypes varied from 5.6 d for a highly susceptible genotype 7042S to 11.5 d for a highly resistant genotype IP 18293. Genotype P 24-1 with a mean

Table 1. Number of pearl millet genotypes in different downy mildew incidence (DMI) classes based on reaction to six pathotypes of *Sclerospora graminicola*

Pathotype	DMI (%) class							DMI (%)	
	0	1–5	6–10	11–20	21–30	31–50	> 50	Mean[a]	Range
Path 1	13	14	15	4	7	6	2	13	0–82
Path 2	5	23	7	14	5	5	2	13	0–77
Path 3	16	18	5	11	5	1	5	11	0–60
Path 4	13	24	7	11	0	3	3	11	0–86
Path 5	17	17	8	13	1	4	1	9	0–80
Path 6	3	11	12	11	5	16	3	20	0–79

a. Rounded to whole number.

Table 2. Downy mildew incidence and host resistance index (HRI) of 61 pearl millet genotypes caused by six pathotypes of *Sclerospora graminicola* in a glasshouse experiment

Pearl millet genotype	Origin	Downy mildew incidence (%)[a]							HRI[b]
		Path 1	Path 2	Path 3	Path 4	Path 5	Path 6	Mean	
D 332/1/2-2	Niger	0	1	1	1	1	3	1	0.91
DIC-P4-P2	–[c]	1	1	2	0	39	41	7	0.68
IP 70-L-1	India	0	0	0	2	1	1	1	0.94
IP 94/1/2-1	Niger	10	14	2	0	0	15	7	0.71
IP 537B	Mali	16	24	24	1	14	31	18	0.40
IP 1481-L-2	India	3	2	18	6	4	35	11	0.61
IP 2084-1	South Africa	1	4	4	0	8	7	4	0.74
IP 5272-1	Niger	4	3	0	87	1	56	25	0.60
IP 5749-1	Nigeria	23	32	54	19	80	79	48	0.16
IP 6138-3	Cameroon	39	44	54	16	9	20	30	0.27
IP 6140-1	Cameroon	7	11	0	2	0	6	4	0.75
IP 6147-4	Cameroon	48	37	60	9	9	33	33	0.28
IP 6240-2	Cameroon	7	11	16	4	0	4	7	0.63
IP 6249-4	Cameroon	5	3	6	14	15	16	10	0.52
IP 8695-1	Sudan	30	25	4	17	32	34	24	0.28
IP 8695-4	Sudan	10	15	28	2	15	22	15	0.41
IP 8703-1	Sudan	23	12	12	2	1	27	13	0.62
IP 8710-1	Sudan	0	1	2	1	0	10	2	0.91
IP 8714-1	Sudan	10	7	11	10	2	13	9	0.51
IP 8715-4	Sudan	32	37	4	3	10	43	22	0.43
IP 8830-2	Zimbabwe	0	1	0	16	0	17	6	0.81
IP 8876-2	Burkina Faso	82	58	16	17	4	25	34	0.37
IP 8877-3	Burkina Faso	7	12	14	8	17	13	12	0.45
IP 8998-1	Nigeria	17	9	3	11	3	7	8	0.59
IP 11541-1	Burkina Faso	26	28	13	19	35	39	27	0.24
IP 18292	ICRISAT[d]	0	0	0	0	0	0	0	1.00

(Continued)

Table 2 Continued

Pearl millet genotype	Origin	Downy mildew incidence (%)[a]							HRI[b]
		Path 1	Path 2	Path 3	Path 4	Path 5	Path 6	Mean	
IP 18293	ICRISAT[d]	0	0	0	1	0	1		0.98
IP 18294	Mali	0	4	8	0	1	2	3	0.89
IP 18295	Cameroon	0	3	1	2	0	2	1	0.91
IP 18296	Nigeria	29	18	0	0	14	2	11	0.67
IP 18297	Mali	0	0	0	39	0	0	7	0.90
IP 18298	Burkina Faso	1	3	0	1	0	0	1	0.94
P 7–4	Nigeria	5	5	5	5	0	21	7	0.66
P 10–1	Nigeria	8	3	2	3	16	39	12	0.59
P 24–1	Cameroon	6	4	0	1	1	10	4	0.77
P 120–1	Cameroon	3	2	3	1	3	7	3	0.78
P 181–2	C A R	0	3	16	7	4	15	8	0.74
P 536–2	Mali	13	19	1	0	2	38	12	0.59
P 1449–2	Senegal	8	20	8	2	6	9	9	0.60
P 1564	Senegal	31	11	0	86	0	35	27	0.52
P 1577	Senegal	71	77	16	85	34	41	54	0.15
P 1591	Senegal	19	16	21	19	12	34	20	0.30
P 1596	Senegal	7	3	21	8	6	35	13	0.47
P 2819–1	Niger	2	4	0	1	0	4	2	0.87
P 2880	Niger	8	3	5	5	2	8	5	0.69
P 2895–3	Niger	0	1	12	50	0	15	13	0.65
P 2910–2	Niger	0	0	0	0	0	2		0.98
P 2914–3	Niger	5	7	1	0	13	10	6	0.65
P 2925–1	Niger	4	5	1	0	8	8	4	0.72
P 2947–2	Niger	10	8	6	5	2	21	9	0.63
P 2950	Niger	10	9	54	31	16	64	31	0.26
P 3281–1	Togo	0	1	0	0	0	4	1	0.93
P 3346–1	Togo	6	17	9	0	7	12	9	0.61
P 8749–1	Botswana	2	4	3	10	0	9	5	0.73
RC-011–2	Gambia	4	6	1	2	3	2	3	0.80
SDN 503	Nigeria	31	25	34	20	11	34	26	0.25
700251	Nigeria	3	6	0	4	2	7	4	0.74
700481-21–8	Nigeria	39	49	59	0	25	43	36	0.30
700481-22–8	Nigeria	7	13	13	4	12	18	11	0.49
700481-23–2	Nigeria	28	20	0	14	11	18	15	0.44
700512-3–2	Nigeria	27	29	26	3	12	42	23	0.38
7042S control	India	83	82	76	72	97	90	83	0.06

LSD ($P < 0.05$) for DM incidence for pathotype x host genotype means=13.00 and for HRI for pathotype x host genotype means=0.26

a. Mean of 150-180 seedlings in each of three replications.
b. HRI based on the formula $[1+(axb^{-1})]^{-1}$ where a=downy mildew incidence %; b=latent period (days).
c. Not known. CAR = Central African Republic.
d. Derived from crosses involving lines from Sudan, India and USA.

Table 3. Analysis of variance for downy mildew incidence (DMI), latent period (LP), and host-resistance index (HRI)

		MS			
Source of variation	df	DMI	df	LP	HRI
Replication	2	874.26	2	63.81	0.20
Pathotype (P)	5	2671.72***	5	208.32***	1.79***
Host genotype (G)	61	4004.45***	60/61[a]	21.07***	1.02***
P x G	305	470.61***	239/305[a]	5.67***	0.11***
Error	742	66.02	454/742[a]	2.45	0.03

*** Significant at 0.1% a. For HRI

Table 4. Latent period[a] (in days) on 61 pearl millet genotypes induced by six pathotypes of *Sclerospora graminicola* in a glasshouse experiment

Pearl millet genotype	Path1	Path2	Path3	Path4	Path5	Path6
D 332/1/2–2	–[b]	6.5	12.0	14.0	11.0	6.5
DIC–P4–P2	11.0	6.0	8.0	–	7.2	7.0
IP 70–L–1	–	–	–	11.5	8.0	7.0
IP 94/1/2–1	8.3	7.5	10.0	–	–	7.5
IP 537B	7.7	6.5	8.3	14.0	7.3	6.2
IP 1481–L–2	6.0	9.5	9.0	11.0	7.8	6.0
IP 2084–1	9.0	11.7	9.0	–	8.8	7.3
IP 5272–1	7.7	10.8	–	7.3	11.0	6.0
IP 5749–1	7.3	5.7	7.0	7.3	5.7	5.7
IP 6138–3	7.7	6.0	7.3	9.2	7.8	6.2
IP 6140–1	7.7	6.7	–	12.0	–	6.0
IP 6147–4	7.0	6.3	8.0	10.0	9.7	6.2
IP 6240–2	10.3	7.0	8.3	9.0	–	7.3
IP 6249–4	7.8	6.5	8.0	8.8	6.8	6.3
IP 8695–1	5.8	6.5	7.0	7.2	5.8	5.7
IP 8695–4	7.3	7.2	7.2	12.5	6.8	7.2
IP 8703–1	9.0	6.5	9.3	12.0	8.0	6.5
IP 8710–1	–	8.5	14.5	14.0	–	6.5
IP 8714–1	7.2	6.5	6.8	9.8	7.5	6.3
IP 8715–4	7.5	6.2	11.3	9.5	8.3	6.3
IP 8830–2	–	9.0	–	9.0	–	7.0
IP 8876–2	5.8	5.7	8.8	9.5	13.0	6.8
IP 8877–3	10.2	6.7	9.3	9.8	8.0	7.3
IP 8998–1	7.8	6.0	9.0	8.5	7.8	7.3
IP 11541–2	6.5	6.8	7.7	8.5	6.7	6.2
IP 18292	–	–	–	–	–	–
IP 18293	–	–	–	14.0	–	9.0
IP 18294	–	9.3	12.0	–	11.0	11.0
IP 18295	–	7.5	8.0	11.5	–	6.5
IP 18296	7.5	9.0	–	–	8.7	7.0

(Continued)

Table 4 Continued

Pearl millet genotype	Path1	Path2	Path3	Path4	Path5	Path6
IP 18297	–	–	–	9.0	–	–
IP 18298	9.0	9.3	–	11.0	–	–
P 7–4	7.8	9.0	8.5	10.2	–	6.3
P 10–1	9.5	10.0	14.0	11.5	6.8	6.0
P 24–1	7.8	6.7	–	6.0	12.5	7.8
P 120–1	9.5	9.0	8.5	9.0	9.7	7.5
P 181–2	–	10.0	11.3	8.8	13.0	7.0
P 536–2	7.8	6.5	9.0	–	8.0	6.2
P 1449–2	10.0	7.3	9.0	7.5	7.5	8.3
P 1564	8.8	10.7	–	7.2	–	6.8
P 1577	7.3	7.2	8.2	6.7	6.0	6.5
P 1591	7.3	6.8	7.8	8.0	6.5	5.8
P 1595	8.5	6.7	8.0	9.0	7.7	6.2
P 2819–1	9.5	7.5	–	13.0	–	9.2
P 2880	8.7	9.7	11.8	10.0	8.3	8.3
P 2895–3	–	11.0	9.7	7.2	–	7.2
P 2910–2	–	–	–	–	–	8.0
P 2914–3	8.3	6.8	13.0	–	7.2	7.3
P 2925–1	9.0	7.2	11.0	–	6.3	6.5
P 2947–2	8.3	7.7	9.0	11.0	10.0	6.8
P 2950	8.2	6.8	7.0	7.3	6.8	5.7
P 3281–1	–	7.0	–	–	–	8.2
P 3346–1	9.8	6.5	10.0	–	8.3	7.5
P 8749–1	8.0	9.0	9.0	10.3	–	7.0
RC-011–2	9.0	9.0	6.0	12.0	10.3	6.5
SDN 503	6.7	6.7	7.5	8.7	8.0	5.8
700251	10.0	6.3	–	11.8	8.0	6.3
700481–21–8	7.8	6.3	8.2	–	7.5	6.3
700481–22–8	9.3	7.0	10.5	7.5	8.3	7.0
700481–23–2	8.2	6.7	–	9.0	8.0	6.2
700512–3–2	11.0	7.5	7.5	13.5	9.0	6.8
7042S Control	5.3	5.3	6.3	5.7	5.7	5.6

LSD ($P < 0.05$) for pathotype x host genotype means = 2.51

a. Mean of 3 replications. b. Data not available because of no infection.

disease incidence of 4% had a latent period of 6 d with Path 4 and 12.5 d with Path 5; and IP 8710-1 a highly resistant genotype (2% mean incidence) had a latent period of 6.5 d with Path 6 and 14.5 d with Path 3. There were significant effects of host genotypes, pathotypes and their interaction on latent period (Table 3). A significant negative correlation ($r = -0.45$ at $P < 0.01$, df=756) was found between latent period and downy mildew incidence across host genotypes and pathotypes.

Differential reactions. A number of host genotypes exhibited clear and strong differential incidence to specific pathotypes. For example, IP 18297 from Mali had 39% incidence with path 4 and 0 with other pathotypes (Table 2). The differential reactions of 8 of the 12 host genotypes (IP 5272-1, IP 18296, IP 18297, P 536-2, P 1564, P 2895-3, P 3281-1 and 700481-21-8) were confirmed in experiment 2 (Table 5). Genotype P 1564 showed 0% incidence with Path 3, 2% with Path 5, 11% with

Path 2, 26% with Path 1, 34% with Path 6 and 77% with Path 4; IP 18297 showed 0% incidence with each Path 1, Path 2, Path 3, and Path 5, < 1% with Path 6, and 33% with Path 4. These genotypes can serve as good host differentials for pathogenic variability studies. ANOVA indicated strong and significant ($P < 0.001$) effects of host genotype, pathotype and their interaction on downy mildew incidence, latent period and HRI (Table 6).

Table 5. Mean per cent downy mildew incidence (DMI) and host resistance index (HRI)[a] in 14 pearl millet genotypes caused by six pathotypes of *Sclerospora graminicola*

Pearl millet genotype	Path1		Path2		Path3		Path4		Path5		Path6	
	DMI	HRI	DMI	HRI	DMI	HRI	DMI	HRI	DMI	HRI	DMI	HRI
IP 5272–1	4	0.68	4	0.69	0	1.00	78	0.08	1	0.93	42	0.14
IP 5749–1	21	0.26	25	0.21	50	0.12	17	0.30	72	0.08	72	0.08
IP 8715–4	29	0.21	34	0.17	4	0.71	4	0.72	19	0.29	3	0.15
IP 8876–2	66	0.09	53	0.11	17	0.31	17	0.33	7	0.57	26	0.22
IP 18296	28	0.21	19	0.35	0	1.00	1	0.83	13	0.41	2	0.84
IP 18297	0	1.00	0	1.00	0	1.00	33	0.21	0	1.00	< 1	0.96
P 536–2	9	0.48	21	0.25	4	0.63	0	1.00	7	0.51	34	0.17
P 1564	26	0.22	11	0.47	0	1.00	77	0.08	2	0.79	34	0.17
P 1577	67	0.11	67	0.10	15	0.35	78	0.0	5	0.11	4	0.16
P 2895–3	0	1.00	< 1	0.97	30	0.21	46	0.13	< 1	0.97	24	0.24
P 3281–1	0	1.00	2	0.81	0	1.00	0	1.00	< 1	0.97	7	0.55
700481–21–S	35	0.17	44	0.14	52	0.13	0	1.00	34	0.18	48	0.12
Controls												
IP 18292	0	1.00	0	1.00	0	1.00	0	1.00	0	1.00	0	1.00
7042S	81	0.07	78	0.07	84	0.07	71	0.08	89	0.06	93	0.06

LSD ($P < 0.05$) for DMI for P x G means = 11.37
HRI for P x G means = 0.10

a. Mean of 2 experimental runs.

Table 6. Analysis of variance for downy mildew incidence (DMI), latent period (LP) and host-resistance index (HRI)

Source of variation	MS				
	df	DMI	df	LP	HRI
Replication	2	247.0	2	31.82	0.042
Experiment (E)	1	334.1	1	13.73	0.094
Error	2	1388.8	2	19.39	0.075
Host genotype(G)	13	18300.4***	12	40.48***	1.267***
E x G	13	159.0	12	8.46***	0.053***
Pathotype (P)	5	2476.0***	5	28.84***	0.666***
E x P	5	383.2	5	27.88***	0.093***
P x G	65	2135.7***	47	9.18***	0.218***
E x G x P	65	155.3	41	2.43*	0.015**
	332	100.9	207	1.57	0.008

*, ** and *** Significant at 5, 1 and 0.1%, respectively.

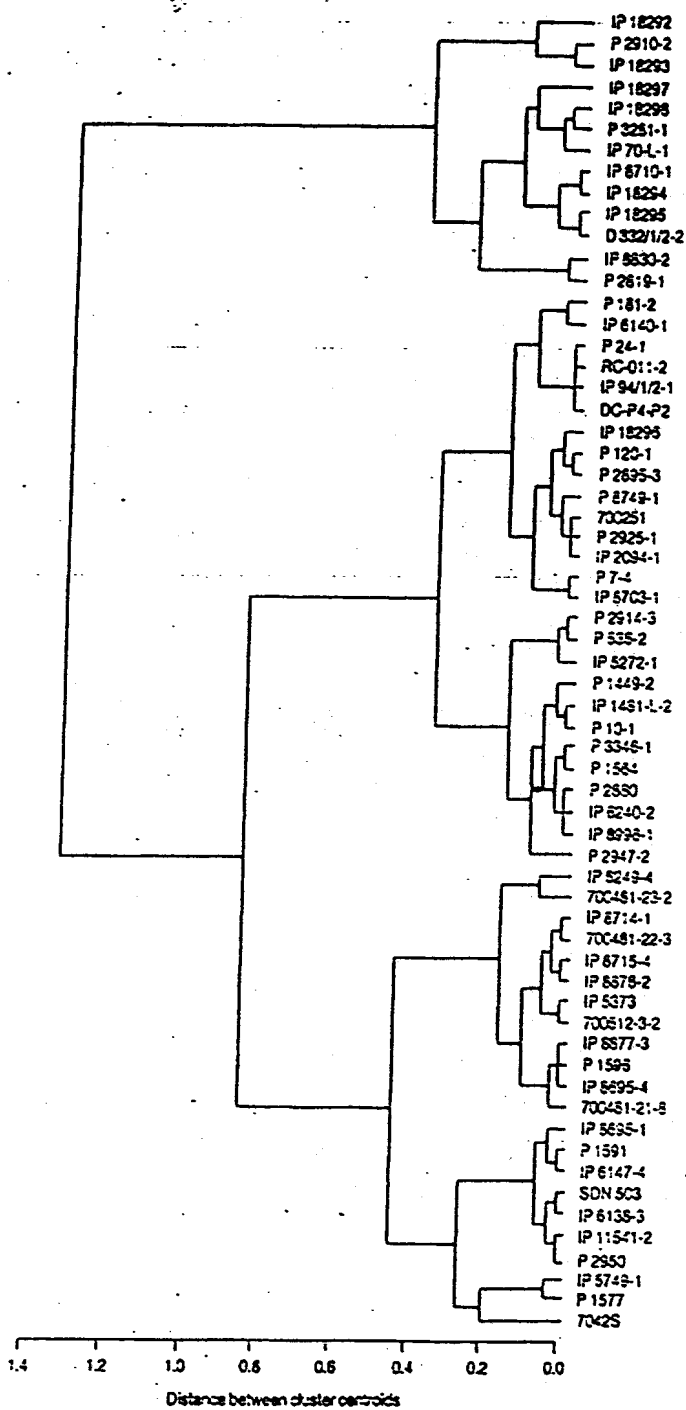

Figure 1. Classification of 62 pearl millet genotypes into distinct groups based on cluster analysis of HRI values across six pathotypes of *Sclerospora graminicola*. The cluster indicates five groups from highly resistant to highly susceptible genotypes.

Host resistance index (HRI). Large and significant variations were observed for HRI among host genotype x pathotype combinations. We considered the mean HRI value across all pathotypes to indicate the likely resistance stability of a line. A highly resistant genotype IP 18292 with no infection had HRI value 1.0 and a highly susceptible 7042S with 83% mean incidence had HRI of 0.06 (Table 1). Highly significant ($P < 0.001$) effects of host genotype, pathotype and their interaction were observed on HRI (Table 3). The cluster analysis provided useful groupings of pearl millet genotypes from a highly resistant to highly susceptible level and indicated the possible evolution of susceptibility/resistance in the gene pool (Fig. 1).

Discussion

We have introduced a new concept of 'HRI' for measuring resistance in a systemic disease, such as downy mildew of pearl millet where both the host and the pathogen are highly variable. The two components of HRI -- disease incidence and latent period, measured in this study, are considered important for characterizing resistance of a host genotype resistance to different populations of a pathogen. Of the 61 known resistant genotypes, only 19 were uniformly resistant (< 10% incidence) to all the six pathotypes, and the remaining 42 had variable reactions. We consider that host genotypes that show higher HRI values across a number of pathotypes or locations (environments) over time would probably be more stable than those with lower HRI values. Sixteen of the 61 host genotypes that had < 10% downy mildew incidence with all six pathotypes, also had HRI values > 0.69 and these are likely to be more stable than others. It is interesting to note that host genotypes that have shown high HRI values have their origin in western Africa - the primary center of origin of pearl millet, conforming the hypothesis of existence of greatest diversity in the center of origin. The results provide ample evidence that resistance in the other 45 genotypes may not be useful in different parts of country and might be easily overcome by virulence factors in the pathogen populations, if utilized as resistance source. It is pertinent to mention here that pathogen isolates tested in this study represent only a fraction of the total likely variability in *S. graminicola* populations in India, and therefore a wider testing involving more isolates will be required to account for the entire range of variability. Nevertheless, this study provided a vital indication of the likely variability in the pathogen population to serve as a guideline for further investigations.

Highly significant negative correlations between incidence and latent period provide a reasonable basis of understanding resistance stability in the source lines. However, the lower disease incidence alone cannot be taken as a true measure of resistance in a highly variable pearl millet-downy mildew pathosystem. A cultivar having fewer infected plants with shorter latent period could contribute more towards disease spread in a crop than those having a greater number of infected plants with longer latent period. Our earlier studies (Thakur *et al.*, 1992) indicated a negative relationship between latent period and sporulation intensity. The results also provide useful information on differential reactions of some genotypes to the six pathotypes. However, these genotypes need to be made genetically homozygous for downy mildew reaction before they can be used as true differentials. These results and evidence (Gill *et al.*, 1978; Jones *et al.*, 1995) that resistance to downy mildew in pearl millet is governed by major dominant genes, form a good basis of future research in developing genotypes with different resistance genes and their strategic utilization in breeding programs to develop cultivars with stable resistance to downy mildew. Pearl millet genotypes, such as IP 18292 and IP 18293 are the best source of resistance available which could provide stable resistance to downy mildew in India.

Acknowledgements

We wish to thank D.R. Butler, K.N. Rai, S. Chandra, B.S. Talukdar and J.M. Lenné for their useful comments on the earlier draft of the manuscript.

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