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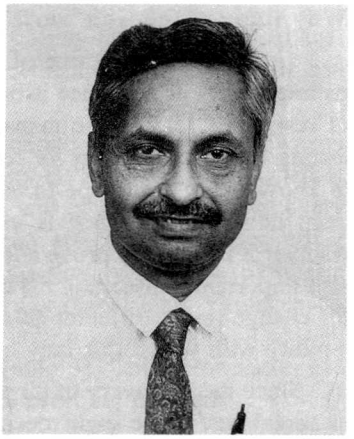

Pathogen diversity and plant disease mangement*

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I express my deep sense of gratitude and appreciation to the Executive Council of the Indian Phytopathological society (IPS) for nominating me to present this prestigious Jeersannidhi Award Lecture for 1998. By bestowing this honour, the IPS has put me in the select group of plant pathologists of India. I happen to be the 15th speaker of this important lecture of IPS. The topic for my presentation, "Pathogen diversity and plant disease management", is probably more relevant today than ever before.

Every plant species, wild or cultivated, has one or more diseases naturally associated with it. Plant diseases are normal part of every natural and semi-natural community, but their impact and role in these communities are usually unnoticed and unappreciated. In artificial cropping systems (particularly monocultures), plant diseases often cause death and destruction leading to yield loss. In natural ecosystems (biologically diverse), they are an unseen influence on both plant community and the evolution of individual species. In natural communities, plant diseases are in balance with their hosts; some host plants die, but enough survive to maintain the species. Plant pathogens, through the diseases they cause, influence the distribution of individual species, the genetic variation of that species with respect to disease resistance, the balance between sexual and asexual reproduction in the host plant, and interaction with other organisms.

According to a recent publication of the first *IUCN (International Unioin for the Conservation of Nature - Now called World Conservation Union)* Red List of the Threatened Plants (Walter and Gillet, 1998), some 34000 plant species (12.5% of the world flora) are facing extinction. In the natural ecosystem, each species has dependence upon it approximately 30 other species; that means for every plant species that becomes extinct 30 other species will go with it and many of these may be plant pathogens (Ingram, 1998).

Plant pathogens are key components of the biodiversity of all natural ecosystems. With the continuing rapid loss of habitats and ecosystem world-wide, the increased use of fungicides, pesticides and herbicides in agriculture, and the release of genetically modified organisms, the threats to pathogen diversity in the wild are immense.

There is equally serious concern about the pathogens of cultivated plants, especially those of major crop species. Such pathogens which are usually held *ex situ* either in official culture and DNA collections or, very frequently, in personal collections have a central role in revealing genetic diversity in crop cultivars.

Why preserve the biodiversity of plant pathogens?

Preservation of diversity in plant pathogens is important because:

they serve as a role in revealing genetic diversity in potential breeding material and provide vital screens for the development of new cultivars;

-they are the raw material for much of the basic scientific research on life cycles and genetics that generate an understanding of pathogen variation, evolution and population dynamics; and

-they constitute a potentially significant biotechnological resource of particular importance to the molecular geneticists.

Unless plant pathogens are conserved along with their hosts, the selection pressure that influences the host will be lost and gradually the genes that confer resistance in the host will fall to extremely low frequencies and will eventually be lost.

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Why study pathogenic variation?

The primary objective of studies on variation in pathogenecity or virulence is breeding and exploitation of resistance for disease management. And while selecting breeding material for resistance, it is important to know which pathotype to use in the screening process; how the resistance is expressed and inherited; whether it is likely to prove adequate and durable? It is also important to ascertain the capacity of the pathogen for change under different selection pressures and the rapidity with which this may occur.

Since the discovery of physiological specialization in cereal rust some eight decades ago (Stakman and Piemeisel, 1917) and later in powdery mildew fungi (Mains and Dietz, 1930), the race concept has become one of the tools for understanding the structure and dynamics of pathogen populations. Further, the classical "gene-for-gene" theory of Flor (1955) enabled a simultaneous analysis of host and pathogen genotypes. Earlier, the racial approach was limited to only biotrophic host-pathogen systems, such as rusts and mildews, but recently it has been extended to necrotrophic systems, such as *Pyricularia oryzae*-rice (Levy *et al.,* 1993); *Rhynchosporirrm secalis*-barley (Ali *et al.,* 1976); *Bipolaris maydis*-maize (Leonard, 1977); *Pyrenophora* spp.-elm (Schilders and Bergstrom, 1990) and others, for both theoretical and applied purposes.

Virulence diversity can be examined under three broad aspects:

- Mechanisms of evolution of virulence,
- Monitoring virulence diversity and
- Using the information in disease management.

Mechanisms of evolution of virulence

A fundamental question is how do pathogens evolve virulence towards their host? There are several mechanisms briefly discussed below.

Mutation: Mutation is the key source of all allelic variation in populations. How sexual and asexual populations of organisms in nature evolve with mutations has been a subject of considerable debate for many years. Most estimates of mutation rates in natural populations have been indirect; they are based on the frequencies of alleles whose precise origins are unknown and are dependent on simplifying assumptions (Caten, 1996). Natural mutation rates in pathogen population are difficult to measure accurately. Flor (1958) measured mutation rate at specific virulence loci between 10^{-5} and 10^{-6} in *Melampsora lini*, and Zimmer *et al.* (1963) between 10^{-3} and 10^{-4} in *Puccinia coronata*. Mutation to virulence in the pathogen population against monogenic resistance is a common phenomenon, and cultivars with such resistance are short lived. However, the cultivars possessing multiple resistance genes are more durable because of a low probability of simultaneous mutation for virulence in the pathogen at multiple loci corresponding to multiple resistance genes (Mundt, 1991).

Genetic drift: The random sampling of gametes between generations in a finite population results in random fluctuation in gene frequencies. It is considered to be generally weak force which acts to decrease genetic variation within populations through fixation of alleles. The effect is large in small population and small in large population.

Recombination: The means by which organisms reproduce and pass their genes on to the next generation have profound effects on the patterns of the genetic variations in populations (Milgroom, 1996). Many plant pathogens reproduce both sexually and asexually, but a large number of them reproduce either or entirely asexually. Asexual reproduction results in offspring that are genetically identical to each other and to their parents, except mutational changes. The result is clonal population structure. Asexual clonal population provides widespread occurrence of identical genotypes; absence of recombinant genotypes and good correlation between independent sets of genetic markers.

Sexual reproduction occurs through meiosis which results in independent assortment of chromosomes and recombination between chromosomes; unlinked genes segregate independently which provides offsprings with recombinant genotypes. Sexual populations provide relatively high levels of genotypic diversity and random association of alleles at different loci (McDonald *el al.,* 1989). Major evolutionary advantages of recombination are: (a) production of novel genotypes that allows organism to adapt quickly to changing environment; (b) advantageous gene combination can be formed more rapidly than in asexual population and (c) a way of purging deletarious mutations that would otherwise accumulate in asexual clones. In general, sexual populations tend to be more genetically diverse than asexual populations of the same species.

Gene flow: Gene flow is the movement of genes from one population to another including migration, the movement of gametes, the extinction of entire population or the movement of extranuclear segments of DNA, such as plasmids and viruses. Gene flow within a species determines the extent to which genetic changes within local population are independent of those in other population. It can be a creative force in evolution

through the spread of new genes or combination of genes.

Selection: This can be a strong force in establishing and maintaining local differences. Host population acts as a powerful selection force on pathogen population and vice-versa (Burden, 1987). Selection intensities often appear to differ in greenhouse and field studies of the same pathogen (Leonard, 1987). The difference is caused by more intense selection pressure in the greenhouse due to higher pathogen densities, or that the field estimates take into account selection during the survival phase between seasons.

Selection intensities on virulent and avirulent genotypes on susceptible hosts are likely to vary spatially and temporally within and between years because plant pathogen populations are notoriously sensitive to host genotype and environmental variations.

The principal environmental influence on obligately biotrophic plant pathogens is its host and it will evolve to exploit it full (Crute, 1981). If the host species is either continuously present or its presence at regular intervals is guaranteed in agriculture, this process of adaptation will continue at the expense of flexibility until individual is capable of parasitizing only a very restricted host range (Crute, 1981). In a host-parasite system, the level of genetic variance in resistance and virulence can strongly influence the population dynamics and equilibrium of the interacting species (Simms, 1996).

Monitoring virulence diversity

Pathogenicity is the genetic marker of most interest and has been studied intensively for a long time. Pathogenic variability has been studied through:

i) virulence survey - field survey and international virulence nurseries;

ii) reaction of host differential lines containing different resistance genes and

iii) use of DNA marker techniques.

Virulence survey: Our experience with survey of pearl millet crops for downy mildew has been very informative. Six years of survey in Maharashtra state has indicated the diversity of hybrids grown by farmers and the extent of downy mildew incidence (Table 1). Several private sector hybrids have shown very high susceptibility to downy mildew in farmers fields, although they remained resistant in the disease nurseries at research stations. In contrast to genetically uniform single-cross hybrids, ICTP 5203, a heterogeneous open-pollinated variety, has shown durable resistance to downy mildew. The information has been shared among the concerned people and results discussed at the annual crop workshops for proper action by the concerned authorities for a suitable replacement of the susceptible hybrids.

The collaborative International Pearl Millet Downy Mildew Virulence Nursery (IPMDMVN) conducted at 13-20 locations in India and countries in Africa every year since 1992 has been effective in understanding virulence pattern in the downy mildew pathogen (Thakur, 1995). The results reveal clear virulence diversity across locations within India (Table 2) as well as in other countries (Table 3). This information has been useful in resistance breeding program.

Use of host differential lines: In the host-pathogen systems where near isogenic lines with known resistance genes are available, use of host differentials has been very successful in monitoring and identifying new virulence or races of the pathogen, viz., flax-*M. lini*; wheat-*P. graminis*, potato-*Phytophthora infestans*, lettuce-*Bremia lactucea* and several others. In other cases where resistance genes are not fully identified and near-isogenic lines are not available, virulence monitoring has been less accurate and effective. In case of pearl millet-*Sclerospora graminicola* system, we have pearl millet lines that can reasonably discern the pathogen populations based on disease incidence, and these lines are used in the IPMDMVN and in greenhouse for further characterization of isolates from Indian locations. We are in the process of developing near-isogenic lines using molecular marker assisted back cross program for several QTLs.

There has been limitation in proper characterization of variability because of limitation of controlled environmental facilities at many locations and variation in data from field nurseries, both temporal and spatial (Thakur, 1995). However, characterization of isolates

Table 1. Field survey: Pearl millet cultivar diversity and downy mildew incidence (%)[1] on major cultivars in Maharashtra, 1993-98

Cultivar	1993 (3)[2]	1994 (3)	1995 (6)	1996 (14)	1997 (16)	1998 (21)
MLBH 104	53	20	90	80	70	75
MLBH 267	0	40	20	90	60	75
GK 1004	-[3]	-	80	20	75	95
Proagro 7701	-	-	40	1	75	55
Eknath 201	-	-	15	90	50	-
BK 560	90	70	-	-	50	-
ICTP 8203	0	0	2	0	1	2

[1]Maximum incidence recorded in any of the observed fields.
[2]Number of hybrid cultivars in different years.
[3]Cultivar not encountered.

Table 2. Differential disease reactions of five *Sclerospora graminicola* populations from India on six pearl millet lines

Path. Pop/ Location	IP 18292	IP 18293	P 310-17	MBH 110	7042R	852B	Putative pathotype
Patancheru	R	R	R	R	S	S	P1
Durgapura	S	S	S	S	S	S	P2
Mysore	R	R	S	R	S	S	P3
Aurangabad	R	R	R	R	R	S	P4
Jalna	R	R	R	S	S	S	P5

R ≤ 10% mean incidence, S > 10% mean incidence.

Table 3. Differential disease reactions at 20 locations in western and central Africa and India, 1992-97

Location/Pop.*	Differential line						
	852B	MBH 110	P310-17	IP 18292	IP 18293	P 7-4	700651
Bombay (4)**	R	R	R	R	R	R	R
Jamnagar (3)	R	R	R	R	R	R	R
Gwalior (2)	R	R	R	R	R	R	R
Patancheru (6)	R	R	R	R	R	R	S
Hisar (3)	R	R	S	R	R	R	R
Jalna (3)	R	S	R	R	R	R	S
Coimbatore (2)	R	S	R	R	R	S	R
Aurangabad (4)	S	R	R	R	R	R	S
Nioro (4)	S	R	R	S	R	R	R
Mysore (6)	S	R	R	R	R	S	S
Bengou (2)	S	S	R	R	R	S	S
Ludhiana (2)	S	S	R	R	R	S	S
Samanko (2)	S	S	S	R	S	R	R
Sadore (5)	S	S	S	S	R	S	R
Kamboinse (5)	S	S	S	S	S	S	R
Durgapura (6)	S	S	S	S	S	S	R
Jodhpur (3)	S	S	S	S	S	S	R
Mandor (3)	S	S	S	S	R	S	S
Bagauda (2)	S	S	S	S	S	S	S
Cinzana (3)	S	S	S	S	S	S	S

R ≤ 10% mean incidence, S > 10% mean incidence.

*Locations: Bambay and Nioro in Senegal; Bengou and Sadore in Niger; Samanko and Cinzana in Mali; Bagauda in Nigeria; Kamboinse in Burkina Faso; and Jamnagar, Gwalior, Patancheru, Hisar, Jalna, Coimbatore, Aurangabad, Mysore, Ludhiana, Durgapura, Jodhpur, Mandor in India.

**Number of years the nursery evaluated.

based solely on virulence data is not necessarily correlated with amount of genetic variation in the pathogen genome as a whole. Other traditional markers used were fungicide resistance, morphological markers, mating type and inter-sterility groups.

Use of molecular markers: Isozymes and DNA markers are being used widely these days. These methods have advantages of precise scoring, inexpensive on a large sale and simultaneous measurement of variability at several loci including those with co-dominant alleles. Some of the techniques used are AFLP (amplified fragment length polymorphism), ALP (amplicon length polymorphism), AP-PCR (arbitrary primed-PCR), DAF (DNA amplification fingerprinting), RAPD (randomly amplified polymorphic DNA), RFLP (restriction fragment length polymorphism), SSCP (single-strand conformation polymorphism), SSLP (simple sequence length polymorphism), SSR (simple sequence/repeats), etc. (Mohan *et al.*, 1997). DNA fingerprinting has been used to detect genetic diversity among six populations of *S. graminicola* from several pearl millet cultivars (Sastry *et al.*, 1995).

Recent advances in DNA marker technology and the concept of marker-assisted selection (MAS) provide a new solution for selecting and maintaining desirable genotypes. Once molecular markers closely linked to desirable traits are identified, MAS can be performed in early segregating generation and at early stage of plant growth. MAS can be used to pyramid major genes including resistance genes. MAS is quite effective and fast, and can be done under greenhouse conditions on pot-grown seedlings. However, the effectiveness of MAS

is reduced when the distance between the marker and the gene of interest increases to the extent that allows frequent cross-over between the marker and the gene. This produces a high percentage of false-positives/negatives in the screening process.

Using information in disease management

Pathogen variability is also one of the main causes of failure of the fungicidal control of plant diseases. Studies over the past two decades have shown that several classes of single-site fungicides are prone to the development of resistance in their target pathogen population. This has, in some cases, led to decrease or loss in activity of such products under field conditions.

For effective disease management, cultivars with durable resistance are required. Durable resistance is the resistance that remains effective while a cultivar possessing it is widely grown for many years (Johnson, 1981). For effective use of host plant resistance (hpr), full understanding of the basis of durable resistance requires knowledge of the genetics of resistance, the population genetics and evolutionary biology of the pathogen, and the interaction of crop management practices with hpr. Thus, developing crop management systems that promote durability will require multidisciplinary efforts.

The use of multiple resistance genes to produce durable resistance can be effective, because there is low probability of a pathogen mutating to virulence simultaneously at multiple loci (Mundt, 1991). Major gene resistance may probably be exploited more efficiently if it is developed either to prevent the pathogen population from easily adapting or to minimize the selection pressure exerted on the pathogen population. Molecular approaches assist in following two alternative strategies: (a) pyramiding of two or more R genes in a system by MAS breeding and (b) genetic modification of resistance genes.

CASE STUDIES

Genetic change in pathogen population : Migration and recombination

Phytophthora infestans - late blight of potato

The devastating effect of potato late blight epidemic in Europe and the famine in Ireland in 1845 is well known. The causal organism was identified as *Phytophthora infestans* (Mont.) de Bary in 1861 (de Bary, 1876). The rapid spread of this pathogen over Europe in 1945 demonstrated the potential of *P. infestans* to migrate and cause disease. This extraordinary late blight epidemic marked the beginning of plant pathology as a science. The estimated global loss due to late blight of potato is US$3 billion annually. A Global Initiative on Late Blight (GILB) has recently been organized involving 19 countries and CIP (Centro Internacional de Papa) to tackle this problem through a network.

The disease was not a serious problem in the US and Canada during 1970s and 1980s; it re-emerged as a serious problem during the early 1990s (Fry and Goodwin, 1997). Disease control failures occurred due to development of a strain resistant to metalaxyl and this was recorded during 1990 (Deahl *et al.*, 1993). By 1993, late blight epidemics were reported throughout the US and western Canada, and it continued up to 1995 (Goodwin *et al.*, 1996). The economic loss to farmers in US was estimated $230 million in 2 years (Johnson *et al.*, 1997).

The cause of and the hypothesis about the epidemic was suggested to be the major genetic change in the pathogen populations in US and western Canada. The hypothesis was experimentally tested. Allozymes and DNA fingerprinting analyses of large number of isolates revealed two new genotype US-7 and US-8 introduced into US from north-western Mexico during 1991-92 (Goodwin, 1997). These were A2 mating types resistant to metalaxyl and highly pathogenic which did not exist in US and Canada before (Goodwin *et al.*, 1996; Koto *et al.*, 1997). During 1993, US-7 was predominant (55% of all isolates) both numerically and geographically and was highly pathogenic to both potato and tomato (Goodwin *et al.*, 1995).

During 1993, US-8 was limited to only one county in Maine (Goodwin *et al.*, 1998). Rapid spread of US-7 A2 mating type and persistence of US-1 Al mating type meant that both mating types were widely distributed during 1993 (Chycoski and minja, 1996). Field survey samplings revealed limited number of both mating type isolates and possibility of limited sexual reproduction. Devastating late blight epidemics were recorded during 1994-96 (Goodwin *et al.*, 1998). These were caused by the genotype US-8; US-7 was not detected during 1994-96 but a new genotype US-17 appeared (Goodwin *et al.*, 1998). By 1994, US-1 clonal lineage was completely replaced by new genotypes US-7 and US-8 from Mexico within 3 years (Goodwin *et al.*, 1998). Genotype US-1 was no more a major component of the population. Similar rapid replacement of US-1 was also reported in Europe (Day and Shattock, 1997; Drenth *et al.*, 1994), Asia (Koh *et al.*, 1994) and

Southern America (Forbes *et al.*, 1997), but by different genotypes. These results strongly suggested the potential migration of new genotypes across countries.

Sexual reproduction was limited during 1994-96 because the two mating types were rarely detected in the same field (Goodwin *et al.*, 1995). This happened due to metalaxyl sensitivity of US-1 isolate. Genotype US-7 did not have advantage despite it infected potato and tomato, indicating that host species diversity may not necessarily be an advantage to the pathogen isolate. Some rare genotypes, such as US-15 originated by sexual recombination, but were not widespread: US-8 spread through seeds in US and Canada, but not US-1 because of its metalaxyl sensitivity. US-8 also has higher fitness and infects both tuber and foliage and also *Solanum sarrachoides* (Vartanian and Endo, 1985). Also that US-8 has been difficult to control even by increased dosages of fungicides.

Virulence change in pathogen population: Host selection

Sclerospora graminicola - Downy mildew of pearl millet

The disease was first reported from India in 1907 (Butler, 1907). The first epidemic of downy mildew was recorded during 1970-71 on a pearl millet F_1 hybrid HB 3 (Safeeulla, 1977), which led to a significant decline in total production of pearl millet. A new hybrid NNB 3 that replaced HB 3 succumbed to downy mildew during 1977-78, BJ 104 during 1983-85, MBH 110 during 1987-89 in Maharashtra, MLBH 104 during 1993-96, again in Maharashtra and several other private sector hybrids during 1995-98 (Thakur and Rao, 1996, 1997; Thakur *et al.*, 1998b). The overall yield loss caused by downy mildew in pearl millet is about 20% which is estimated to be about Rs. 480 crores (US$ 137 million) annually in India (Thakur, 1997).

Physiological specialization in the pathogen was reported in 1981 (Shetty and Ahmed, 1981) and heterothallism was demonstrated in 1982 (Michelmore *et al.*, 1982). Pathogenic variability and sexual compatibility between isolates from Africa and India were reported (Ball, 1983; Idris and Ball, 1984; Werder and Ball, 1992).

A greenhouse experiment clearly demonstrated evolution of host-specific virulence when field population of the pathogen was serially passed on to seedlings of some pearl millet hybrids and lines for several asexual generations (Fig. 1) (Thakur *et al.*, 1992; Thakur and Rao, 1997). An international collaborative virulence nursery (IPMDMVN) was constituted in 1992 to monitor virulence across the major pearl millet growing

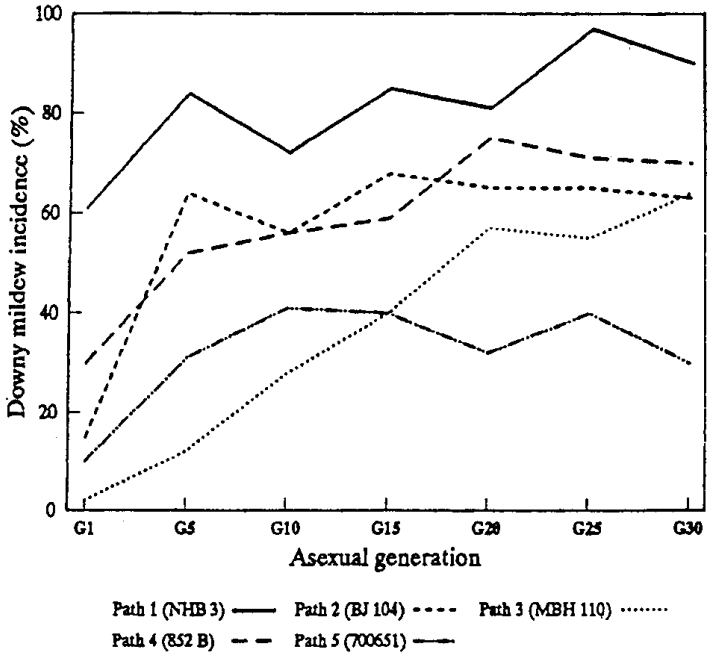

Fig. 1. Disease incidence (%) induced by pathotypes of *Sclerospora graminicola* on their selection host cultivars of pearl millet at different asexual generation in a greenhouse experiment

regions of the world. Pathogenic variations have been reported among single-oospore (Thakur and Shetty, 1993) and single-zoospore isolates (Thakur *et al.*, 1998a). DNA fingerprinting was used to demonstrate genetic variation in host-specific-pathotypes (Fig. 2), and also among single-oospore and single-zoospore

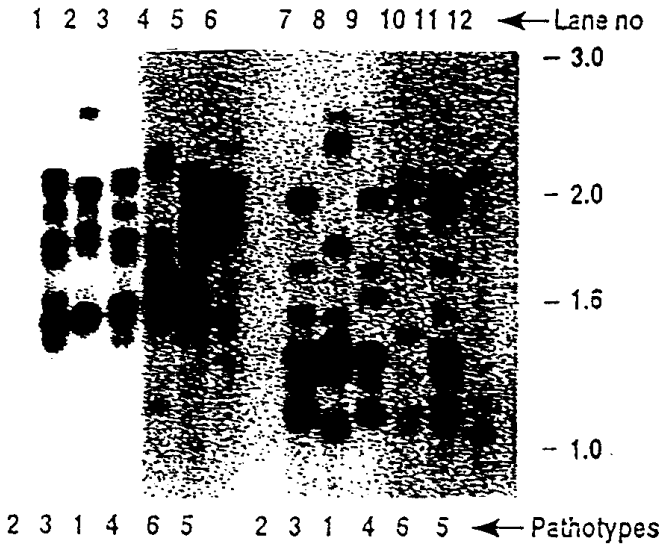

Fig. 2. Autoradiogram of the DNA fingerprints of six host cultivar-specific pathotypes of *Sclerospora graminicola:* fungal DNA digested with *Msp*I (lanes 1-6) and *Taq*I (lanes 7-12) and hybridized with (GATA) 4. Lanes 1&7 Path-2 (BJ 104); 2&8 Path-3 (MBH 110); 3&9 Path-1 (NHB 3); 4&10 Path-4 (852B); 5&11 Path-6 (7042S); 6&112 Path-5 (700651). Molecular size of the markers in kb are indicated in the right margin

isolates of *S. graminicola* (Sastry *et al.*, 1995).

The first DNA markers for QTLs against different isolates of the pathogen were identified during 1993-94 (Jones *et al.*, 1995) and since then over 20 QTLs have been identified on different linkage groups of pearl millet (C.T. Hash, personal communication). QTLs for DMR indicated genetic variation in the pathogen population. These QTLs, dispersed on several linkage groups in pearl millet genome, account for 20-70% variability in resistance against specific pathogen populations from India and Africa.

Epilogue

Understanding and protecting biodiversity of plant pathogens is crucial to the management of genetic diversity for host plant resistance. A legislation similar to that of plant species and other life forms is needed to improve the current situation.

Mycologists, bacteriologists, virologists, nematologists and plant pathologists have played important roles in collecting, characterizing and preserving the type cultures of some of the pathogens, but the list is far from being adequate. Conservation of plant pathogens both *ex-situ* and *in-situ* are important. As we know, every pathogen species has numerous biotypes, races, or pathotypes with specific genes for virulence that correspond to the corresponding genes in the respective host plants. It means that losing a particular race or pathotype will eliminate that resistance gene. Greater efforts on the part of plant pathologists are needed to convince the funding agencies to strengthen the gene bank of plant pathogens.

Proper understanding of the spectrum of diversity in the pathogen population is highly crucial in the process of resistance identification and breeding for resistance against a particular disease. Regular monitoring for the evolution of new pathotypes is of great help to develop strategic resistance breeding program and resistance deployment for the genetic management of a disease.

REFERENCES

All, S.M., Mayfield, A.H. and Clare, B.C. (1976). Pathogenicity of 203 isolates of *Rhynchosporium secalis* on 21 barley cultivars. *Physiol. Plant Path.* **9**: 135-143.

Ball, S.L. (1983). Pathogenic variability of downy mildew (*Sclerospora graminicola*) on pearl millet, I. Host cultivar reactions to infection by different pathogen isolates. *Ann. appl. Biol.* **102**: 257-264.

Burden, J.J. (1987). Diseases and Plant Population Biology. Cambridge Univ. Press. 208 pp.

Butler, E.J. (1907). Some diseases of cereals caused by *Sclerospora graminicola*. Memoirs of the Department of Agriculture in India. *Botanical Series* **2**: 1-24.

Caten, E.C. (1996). The mutable and treacherous tribe revisited. *Plant Pathology* **45**: 1-12.

Chycoski, C.I. and Punja, Z.K. (1996). Characteristics of populations of *Phytophthora infestans* from potato in British Columbia and other regions of Canada during 1993 to 1995. *Plant. Dis.* **80**: 579-589.

Crute, I.R. (1981). The host specificity of Peronospraceous fungi and the genetic relationship between host and parasite. In *The Downy Mildews* (Spencer, D.M. Ed.). Academic Press, London, NY. pp 237-253.

Day, J.P. and Shatteck, R.C. (1997). Aggressiveness and other factors relating to displacement of populations of *Phytophthora infestans* in England and Wales. *Eur. J. Plant Pathol.* **103**:379-391.

Deahl, K.L., Inglis, D.A. and DeMuth, S.P. (1993). Testing for resistance to metalaxyl of *Phytophthora infestans* isolates from northwestern Washington. *Amer. Potato J.* **70**:779-795.

de Bary, A. (1876). Researches into the nature of the potato fungus *Phytophtora infestans. J. Royal Agric. Soc. England* **12**:239-269.

Drenth, A., Tas, I.C.Q. and Govers, F. (1994). DNA fingerprinting uncovers a new sexually reproducing population of *Phytophtora* in the Netherlands. *Eur. J. Plant Pathol.* **100**: 97-107.

Flor, H.H. (1955). Host-parasite interaction in flax rust- its genetics and other implications. *Phytopathology* **45**: 650-685.

Flor, H.H. (1958). Mutation to wider virulence in *Melampsora lini. Phytopathology* **48**: 297- 301.

Forbes, G.A., Escobar, X.C., Ayala, C.C., Revelo, J., Ordonez, M.E., Fry, B.A., Doucett, K. and Fry, W.E. (1997). Population genetic structure in *Phytophthora infestans* in Ecuador. *Phytopathology* **87**: 375-380.

Fry, W.E. and Goodwin, S.B. (1997). Resurgence of the Irish potato famine fungus. *Bio Science* **47**: 363-371.

Goodwin, S.B. (1997). The population genetics of *Phytophthora. Phytopathology* **87**: 462-473.

Goodwin, S.B., Smart, C.D., Sandrock, R.W., Deahl, K.L., Punja, Z.K. and Fry, W.E. (1998). Genetic change within populations of *Phytophthora infestans* in the United States and Canada during 1994 to 1996: Role of migration and recombination. *Phytopathology* **88**: 939-949.

Goodwin, S.B., Sujokowski, L.S., Dyer, A.T., Fry, B.A. and Fry, W.E. (1995). Direct detection of gene flow and probable sexual reproduction in *Phytophthora infestans* in the northern North America. *Phytopahology* **85**: 473-479.

Goodwin, S.B., Sujokowski, L.S. and Fry, W.E. (1996). Widespread distribution and probable origin of resistance to metalaxyl in clonal genotypes of *Phytophthora infestans* in the United States and western Canada. *Phytopathology* **86**: 793-800.

Idris, M.O. and Ball, S.L. (1984). Inter- and intra-continental sexual compatibility in *Sclerospora graminicola*. *Plant Pathology* **33**: 219-223.

Ingram, D.S. (1998). Plant pathogens, diversity and conservation. *Int. Plant Path. Cong.* 3.5PL (abstr.).

Johnson, D.A., Cummings, T.F., Hamm, P.B., Rowe, R.C., Miller, J.S., Thornton, R.E., Pelter, G.Q. and Sorensen, E.J. (1997). Potato late blight in the Columbia Basin: An economic analysis of the 1995 epidemic. *Plant Dis.* **81**: 103-106.

Johnson, R. (1981). Durable resistance: Definition of genetic control and attainment in plant breeding. *Phytopathology* **71**: 567-568.

Jones, E.S., Liu, C.J., Gale, M.D., Hash, C.T. and Witcombe, J.R. (1995). Mapping quantitative trait loci for downy mildew resistance in pearl millet. *Theor. Appl. Gen.* **91**: 448-456.

Kato, M., Mizubuti, E.S., Goodwin, S.B. and Fry, W.E. (1997). Sensitivity to protectant fungicides and pathogenic fitness of clonal lineage of *Phytophthora infestans* in the United States. *Phytopathology* **87**: 973-978.

Koh, Y.J., Goodwin, S.B., Dyer, A.T., Cohen, B.A., Ogoshi, A., Sato, N. and Fry, W.E. (1994). Migration and displacements of *Phytopththora infestans* populations in East Asian countries. *Phytopathology* **84**: 922-927.

Leonard, K.J. (1977). Races of *Bipolaris maydis* in the southeastern US from 1974-1976. *Plant Dis. Reptr.* **60**: 245-247.

Leonard, K.J. (1987). The host population as a selective factor. In: *Population of Plant Pathogens: Their Dynamics and Genetics* (Wolfe, M.S. and Caten, E.C., Eds.). Blackwell Scientific Publications, Oxford, pp. 163-179.

Levy, M., Correa-Victoria, F.J., Zeigler, R.S., Xu, S. and Hammer, J.E. (1993). Genetic diversity of the rice blast fungus in a disease nursery in Colombia. *Phytopathology* **83**: 1427-1433.

Mains, E.B. and Dietz, S.M. (1930). Physiological forms of barley mildew *Erysiphe graminis hordei* Marchal. *Phytopathology* **20**: 229-239.

McDonald, B.A., McDermott, J.M., Goodwin, S.B. and Allard, R.W. (1989). The population biology of host-pathogen interactions. *Annu. Rev. Phytopathol.* **27**: 77-94.

Michelmore, R.W., Pawar, M.N. and Williams, R.J. (1982). Heterothallism in *Sclerospora graminicola*. *Phytopathology* **72**: 1368-1372.

Milgroom, M.G. (1996). Recombination and multilocus structure of fungal populations. *Annu. Rev. Phytopathol.* **34**: 457-477.

Mohan, M., Nair, S., Bhagwat, A., Krishna, T.G. and Yano, M. (1997). Genome mapping, molecular markers and marker-assisted selection in crop plants. *Molecular Breeding* **3**: 87-103.

Mundt, C.C. (1991). Probability of mutation to multiple virulence and durability of resistance gene pyramids: Further comments. *Phytopathology* **81**:240-242.

Safeeulla, K.M. (1977). Genetic vulnerability: The basis of recent epidemics in India. *Annals N.Y. Acad. Sci.* **287**:72-85.

Sastry, J.G., Ramakrishna, W., Sivaramkrishnan, S., Thakur, R.P., Gupta, V.S. and Ranjekar, P.K. (1995). DNA fingerprinting detects genetic variability in the pearl millet downy mildew pathogen (*Sclerospora graminicola*). *Theor. Appl. Genet.* **91**:856-861.

Schilders, A.M.C. and Bergstrom, G.C. (1990). Variation in virulence within the population of *Pyrenophora tritici* in New York. *Phytopathology* **80**: 84-90.

Shetty, H.S. and Ahmad, R. (1981). Physiologic specialization in *Sclerospora graminicola*. *Indian Phytopath.* **34**: 307-309.

Simms, E.L. (1996). The evolutionary genetics of plant pathogen systems. *Bioscience* **46**: 136-145.

Stakman, E.C. and Piemeisel, F.S. (1917). A new strain of *Puccinia graminis* (abstract). *Phytopathology* **7**: 73.

Thakur, R.P. (1995). Status of international sorghum anthracnose and pearl millet downy mildew virulence nurseries. In: *Disease Analysis Through Genetics and Biotechnology-Interdisciplinary Bridges to Improved Sorghum and Pearl Millet Crops* (Leslie, J.F. and Frederiksen, R.A., Eds.). Iowa State Univ. Press. Ames, USA, pp. 75-92.

Thakur, R.P. (1997). Host plant resistance in integrated disease management of pearl millet. *Int. Conf. Integrated Plant Disease Management for Sustainable Agriculture.* P. 22 (abstr.). Indian Phytopathological Society, New Delhi, India.

Thakur, R.P. and Rao, V.P. (1996). Evidence for a new virulent pathotype in *Sclerospora graminicola* on pearl millet. *Indian J. Mycol. Plant Pathol.* **26**: 133 (abstr.).

Thakur, R.P. and Rao, V.P. (1997). Variation in virulence and aggresiveness among pathotypes of *Sclerospora graminicola* on pearl miliet. *Indian J. Mycol. Plant Pathol.* **50**: 41-47.

Thakur, R.P. and Shetty, K.G. (1993). Variation in pathogenicity among single-oospore isolates of *Sclerospora graminicola*, the causal organism of downy mildew in pearl millet. *Plant Pathology* **42**: 715-721.

Thakur, R.P., Pushpavathi, B. and Rao, V.P. (1998a). Virulence characterization of single-zoospore isolates of *Sclerospora graminicola* from pearl millet. *Plant Dis.* **82**: 747-751.

Thakur, R.P., Shetty, K.G., and King, S.B. (1992). Selection for host-specific virulence in asexual populations of *Sclerospora graminicola*. *Plant Pathology* **41**: 626-632.

Thakur, R.P., Rao, V.P. and Hash, C.T. (1998b). Emergence of a highly virulent pathotype of *Sclerospora graminicola* at Jodhpur, Rajasthan, India. *Int. Sorghum Pearl Millet Newslet.* (accepted).

Vartanian, V.G. and Endo, R.M. (1985). Overwintering hosts, compatibility types and races of *Phytophthora infestans* on tomato in southern California. *Plant Dis.* **69**: 516-519.

Walter, K.S. and Gillet, H.J. (1998). Red List of Threatened Plants. IUCN publication, Cambridge, UK.

Werder, J. and Ball, S.L. (1992). Variability of pearl millet downy mildew (*Sclerospora graminicola*) and its implications for breeding for stable and durable resistance. *Tropical Pest Management* **38**:43-47.

Zimmer, D.E., Schafer, J.F. and Patterson, F.L. (1963). Mutations for virulence in *Puccinia coronata*. *Phytopathology* **53**: 171-176.