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OPPORTUNITIES FOR RESEARCH ON DISEASES OF PULSE CROPS

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I am grateful to the Indian Phytopathological Society for inviting me to deliver the Jeersannidhi Award Lecture (1985), the third of the series.

Pulse crops or grain legumes are the major source of protein in the predominantly vegetarian diet of the people of India. We probably grow a greater variety of pulses than any other country. These pulses include : chickpea (*Cicer arietinum* L.) pigenopea (*Cajanus cajan* (L.) Millsp.), black gram (*Vigna mungo* (L.) Hepper), green gram (*Vigna radiata* (L.) Wilczek), lentil (*Lens culinaris* Medik), pea (*Pisum sativum* L.), moth bean (*Vigna aconitifolia* (Jacq.) Marechal), horse gram (*Macrotyloma uniflorum* (Lam.) Verdc.) khesari (grass pea) (*Lathyrus sativus* L.), and cowpea (*Vigna unguiculata* (L.) Walp.).

The area planted annually to pulses has varied in recent years between 20 to 24 million ha. The annual production is about 12 million tonnes, but our requirement is about 17 million tonnes, indicating a deficiency of over 30%. The overall yield has been fluctuating between 500 and to 700 kg/ha, but the potential yield from presently grown cultivars is over 3000 kg/ha. Among the pulses, chickpea and pigeonpea are the most important occupying about 60% of the area and contributing about 70% of the total production (Bolaria, 1982). In fact, India produces about 90% of the pigeonpea and 70% of the chickpea grown in the world. The deficit in pulse production should be overcome not through imports, but through increasing production in the country. Reference to increasing pulse production in the Prime Minister's 20-point economic program is, therefore, very meaningful.

Several explanations for the low production of pulses have been offered. Most of the pulse crops are grown by small farmers either on marginal lands and as intercrops or as catch crops on residual moisture (only 9% of the area under pulses is irrigated). The use of inputs, particularly improved seeds and chemicals is scanty. Pulse crops suffer losses due to diseases, insects pests, drought, waterlogging, salinity, and a variety of other stress factors. Support prices and marketing arrangements are inadequate. My own simple explanation is that farmers find pulses more risky than their preferred crops. Risks are two-fold; biotic and abiotic stress factors result in low production and fluctuating prices. I have no doubts that farmers will grow more pulses, if the risks are reduced. Diseases of pulse crops are serious yield reducers and contribute substantially

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to instability of production. We, the plant pathologists, can play a very vital role in increasing pulse production if we identify the pathological constraints properly and carry out problem-oriented research.

To do research we need resources, both human and physical. Almost every one agrees that facilities available to pathologists in India are inadequate. However, I find that we often forget our valuable resource of dedicated, well-trained persons. I have surveyed the literature on pulse pathology published from 1979 through 1985 and found that about 40 Indian pathologists have published approximately one paper on pulse pathology per year. Of these only about 20 can be considered as regular pulse pathologists, who spend the major part of their time on research on pulse crop diseases. The membership of Indian Phytopathological Society is about 1200 and thus we have less than 2% of the pathologists working on the pulse crop diseases. I consider, therefore, that our major resource constraint is human and we cannot hope to successfully contribute towards achieving our goal of increasing pulse production with such a small number of pathologists. My reasons for choosing the present topic for the Jeersannidhi Award Lecture should therefore be obvious. I hope my lecture will stimulate at least a few young scientists to dedicate themselves to pulse pathology and thus serve the nation in a meaningful way.

SYMPTOMATOLOGY

I am sure quite a few eyebrows will be raised if I say there are opportunities for research in symptomatology. However, let me remind you that until 1978 any prematurely dry plant of chickpea was considered affected by the so-called "wilt complex". In 1973 Dr. H. K. Jain, the then Director of the Indian Agricultural Research Institute, felt it necessary to organize a symposium at national level to discuss the mystery of the "wilt complex". As we know today, the key problem was the inability of pathologists to diagnose different diseases of chickpea that resulted in premature death. A critical study of differential symptomatology solved the 50-year mystery (Nene *et al.*, 1978). A similar situation exists in lentils today. Most of the seedling and post-flowering mortality is attributed to Fusarium wilt. There is no doubt in my mind that different pathogens are involved and someone needs to study the differential symptoms. A similar situation exists in pea because there are several causes of yellowing and drying, and diagnosis based on our present knowledge of symptomatology is often difficult.

I have heard some virologists saying that one can't identify mosaics caused by different viruses in a crop on the basis of symptoms. I have often observed virologists rushing to the laboratory to diagnose the diseases. I have no quarrel with them on that approach. However, based on my own experience, maintain that one can make at least a tentative diagnosis in the field itself if one learns (and this is research) to distinguish finer differences in symptoms. One needs a critical eye and of course a lot of patience.

LOSS ASSESSMENT

In the past I have had interesting discussions with administrators on this topic. Some administrators question spending resources on research on crop disease losses as they feel "farmers can tell us" which disease causes losses and which does not. While there is some truth in this statement, I often find that many of these administrators

happily use any published loss data when they want to tell us that we are not properly focussing our attention on disease problems. As pathologists we know that assessment of losses of various kinds is essential to any serious discussion on disease management. Management of plant diseases presupposes that accurate and reliable estimates of losses are available, to farm managers, extension workers, and other agricultural decision makers. Our recent study (Kannaiyan *et al.*, 1984) that wilt in pigeonpea causes an annual loss of Rs. 37 crores (US $ 37 million at 1981 price level) and that sterility mosaic causes a loss of Rs. 75 crores in India has helped us at ICRISAT to reorient our pigeonpea breeding strategies for India.

A research area which has received virtually no attention in India is the development quantitative models for disease assessment. To carry out research in this area, one needs a good background of mathematics and a liking for the use of computers. It is high time that our young scientists recognize the need and make a special effort to familiarize themselves with the use of computers during their student life. There are no limits to research opportunities in this area. We have virtually no idea of the quantitative losses caused by some of the diseases such as, to name a few, Ascochyta blight and Botrytis grey mold of chickpea, Phytophthora blight of pigeonpea, leaf curl (tomato spotted wilt virus) of green gram and black gram, powdery mildew of green gram and black gram in south India, wilt and rust of lentil, and powdery mildew and rust of pea.

Before I leave this topic I would like to touch upon the subject of disease surveys and surveillance. These activities are definitely research activities and not sightseeing tours, as some non-pathologists seem to think. Loss estimation studies cannot be carried out without surveys. However, I feel so sad when I find that many of the pulse pathologists have no idea what diseases are prevalent in their own regions and states at a given time. This is not because these pathologists do not want to know the status of disease incidence, but often it is because the transport facility is denied. The only answer to this is to exert appropriate pressures at all levels and make research administrators appreciate our needs.

PATHOGEN TAXONOMY

Whenever I see rusty brown pustules on chickpeas, I just assume that the fungus that is responsible for these pustules is *Uromyces ciceris-arietini*. In 1983 and 1984 chickpea rust was observed in Bangalore, but not in 1985. To my complete surprise, I found rust in a chickpea agronomy plot at ICRISAT Centre in 1983 where vegetative growth was excessive, but not in any of the normal plots. I have not observed the rust at ICRISAT Centre since then. Rust is observed almost every year in the post-flowering stage of chickpea in northern India. It is reported that the fungus can survive in the Himalayan mountains on *Trigonella polycerata*, a common weed (Saksena and Prasada, 1956). It is not known whether this weed exists in the hills in southern India. Thus, we do not know the primary source of rust inoculum on chickpeas in Bangalore and Hyderabad. On the other hand, is it possible that the rust observed at these two locations is not *U. ciceris-arietini*. A recent report indicates that chickpea can be infected by a race of another rust species, *U. striatus* (Goulter, 1984), that is normally found on lucerne in Queensland in Australia. We need to investigate whether the rust

species occurring in Bangalore is in fact *U. ciceris-arietini*. Also, we can ask ourselves if we need to reexamine our identification of other important, well-accepted pathogens.

The Phytophthora blight of pigeonpea is caused by *P. drechsleri* f.sp. *cajani* Kannaiyan *et al.* There are reports of other *Phytophthora* spp. attacking pigeonpeas in other countries (Nene *et al.*, 1985). Do we have only one species in India or more?

I always feel uncomfortable in identifying bacterial species attacking pulse crops. When I scanned through the list of 20 active pulse pathologists in India, I could locate perhaps one bacteriologist. No wonder that most of us look outside India for help in identifying bacterial species. Here is a wide open opportunity for young plant bacteriologists to make a career and build upon a reasonably good base that already exists. Opportunities exist not only in the field of bacterial taxonomy, but in all aspects of bacterial phytopathology.

Compared to bacteriology, we have done well in nematode taxonomy. However, we need young pathologists who will make a career in nematode diseases of pulse crops. I shall speak later on the subject of virus identification.

PATHOGEN VARIABILITY

Workers have provided some evidence for the existence of races of a few pathogens such as *Ascochyta rabiei* (ICRISAT, 1985; Satya Vir and Grewal, 1974), *Fusarium oxysporum* f. sp. *ciceri* (Haware and Nene, 1982), *F oxysporum* f. sp. *lentis* (Kannaiyan and Nene, 1978), *F. oxysporum* f. sp. *pisi* (Ram Phal and Choudhury, 1983), *Uromyces viciae-fabae* (Singh and Sokhi, 1980), *Botrytis cinerea* (personal communication-Dr. J. S. Grewal), and Xanthomonads of green gram and cowpea (Jindal *et al.*, 1981). However, there is so much more that needs to be done in a systematic fashion, not only in the case of pathogens listed above, but also for other pathogens. No disease resistance breeding programme can be successful unless we have a good idea of the pathogenic variability that exists in different regions of the country. I fully appreciate that the work on establishing variability in pathogens is not easy. At ICRISAT, while we have succeeded in establishing a system of identifying chickpea Fusarium wilt pathogen races, we are still struggling with *Fusarium udum*, the pigeonpea wilt pathogen. Our difficulty has been mainly the availability of seed that is homozygous for susceptibility, because of the open-pollinated nature of pigeonpea and the possibility of several minor genes contributing towards the overall defence of the host cultivar.

Pathogens that require attention, as far as the variability is concerned, are : *Ascochyta rabiei, Botrytis cinerea* and *Fusarium oxysporum* f.sp. *ciceri*, on chickpea; *Fusarium udum*, sterility mosaic virus, and *Phytophthora drechsleri* f.sp. *cajani* on pigeonpea; *Cercospora canescens* on green gram; *Fusarium oxysporum* f.sp. *lentis on lentil; powdery mildews on pea, green gram, and black gram; rusts on pea, chickpea, and lentil; and Xanthomonas campestris* pv. *vignicola* on cowpea.

EPIDEMIOLOGY

This is an area of research which is very wide in its scope and offers us excellent opportunities for a very meaningful research. So often do I find that certain aspects of epidemiology are accepted as facts but the evidence is just not there. One example will

be adequate to explain the point I am making. Workers have accepted that the primary inoculum of *Ascochyta rabiei*, that causes a severe blight of chickpea, is the infected seed. Evidence elsewhere suggests that the infected plant debris does not serve as a primary source of inoculum, but no acceptable confirmation has ever been made under the north Indian situations. Also only *Cicer* spp. are infected by this fungus under natural conditions. It is often stated that once the primary infection occurs in a field, rapid spread in the field and in the area occurs during rains with winds. This latter part is again an assumption that has come to be regarded as a fact. No one has demonstrated how far the spores can be carried from the infection source during the rains of various intensities or with winds of differing velocities. When seeds produced at Hyderabad, where Ascochyta blight does not occur, were grown in Lahaul valley, where chickpeas are not grown, blight incidence was observed. Where did that infection come from? If rains with winds are responsible for the spread, why was it that in Jabalpur the disease remained restricted around the primary source of infection (the seed was from north India, and so was possibly infected) in spite of several successive spells of rain storms from the last week of December 1983 through January 15, 1984? Again, if rains with wind are the major contributing factor towards the spread, why is it that the disease appears suddenly and synchronously often within 1 to 2 weeks in the states of Punjab, Haryana, and even northern Rajasthan. Is it possible that the inoculum is lifted by winds into the low clouds and that we get "showers of spores"? When we look for answers to these questions, we find that we have none that are based on solid experimental evidence.

Recent work at ICRISAT has established that *Phytophthora drechsleri* f.sp. *cajani* of pigeonpea survives from one season to another on infected stubble (personal communication-Dr. V. S. Bisht). We do not know of any other host of this fungus except the wild relatives of pigeonpea, *Atylosia* spp, which grow as weeds. Seed does not carry the fungus. However, it is not uncommon to see *Phytophthora* on pigeonpea in fields where no pigeonpea was planted at least for 5 preceding years. How does this occur? We have two kinds of soils at ICRISAT Centre : deep black Vertisols and shallow red Alfisols. Pigeonpea grown on Alfisols gets affected more by *Phytophthora* than that grown in Vertisols; why? Why is it that in our surveys Phytophthora blight was observed more at experiment stations than in farmers' fields?

Why is it that pigeonpea sterility mosaic occurs more in U.P., Bihar and Tamil Nadu than in other states? Why is it that in one year there is an epidemic of sterility mosaic in eastern U.P. and Bihar and the next year there is very little incidence? Again, why is it that Maharashtra (Vidarbha and Marathwada regions) and northern Andhra Pradesh have a very high pigeonpea wilt incidence compared to other parts of India? Is it that the cotton-pigeonpea intercrop, characteristic of this region, somehow favours the survival and incidence. Why is it that chickpea Fusarium wilt is most serious in vertisols of India, but not in other kinds of soils? This wilt fungus can survive in Vertisols for over 6 years (ICRISAT, 1985); is that true for all soils? Chickpea stunt, caused by pea leaf roll virus and vectored by aphids, occurs more in some parts of India; why? Mung bean yellow mosaic virus affects green gram, black gram, pigeonpea, and soybean and is vectored by *Bemisia tabaci*. The vector is present all over India. Then why is it that yellow mosaic on these crops (particularly soybean) is more widespread and severe in northern India than

in central India? Why is it that the pigeonpea cyst nematode (*Heterodera cajani*) occurs more in Vertisols than in other types of soils? However, the lance nematode (*Hoplolaimus seinhorstii*) is hardly observed in these Vertisols; why? Are we satisfied that we have adequate evidence to explain the annual recurrence of pea rust, lentil rust, pea powdery mildew? These are important diseases in northern India and we must understand their epidemiology.

DISEASE RESISTANCE

Undoubtedly disease resistant cultivars offer the best means of managing diseases of pulse crops. In recent years I have seen evidence of increased research effort towards identifying sources of resistance and their utilization in crop improvement programmes. Until a few years ago, we did not know any source of resistance to pigeonpea sterility mosaic, chickpea wilt and dry root rot, and pea powdery mildew. Now we have good sources of resistance to these diseases. I feel that pulse pathologists must focus a good deal of their attention to the identification of stable and durable sources of resistance to all diseases which are of real as well as potential importance. It is the job of pathologists, having identified sources of resistance, to continue to work with breeders until acceptable disease resistance cultivars are developed.

While for some diseases, efficient field and glasshouse screening techniques for the identification of resistance have been developed [Nene *et al.*, 1981(a); 1981(b)], there is a need to work out efficient screening techniques for other diseases such as chickpea stunt and Botrytis gray mold, green gram *Cercospora* and powdery mildew, black gram and green gram leaf curl, pea rust and powdery mildew, cowpea, mosaics, and root knot and cyst nematodes of various pulse crops. Also research is needed to develop nurseries for simultaneous screening of breeding material for multiple disease resistance.

Sometimes we are not able to find a good level of resistance to some diseases in the available world germplasm. Examples are *Ascochyta* and *Botrytis* on chickpea, *Phytophthora* on pigeonpea and yellow mosaic on green gram. In such cases it is necessary to screen the wild relatives of the cultivated species, to identify genes for resistance, and if genes are not discovered in the wild species, we must irradiate the seeds of moderately resistance genotypes in the hope of obtaining desired mutants. We now seem to have no alternative to mutation breeding in chickpea, to obtain better sources of resistance to *Ascochyta* and *Botrytis* than are available at present.

CULTURAL PRACTICES

An area of research which I think has immense scope in India, not only in pulse crops but in all crops, is that of what is now called solarization (Katan, 1981). This involves covering the soil with transparent polyethylene sheets for at least a month during the summer prior to rainy season (Kharif) plantings. Covering irrigated soil with polyethylene raises the soil temperature by at least 10°C over the ambient temperatures and this moist heat pasteurizes the soil. At ICRISAT Centre we found that we can effectively control both pigeonpea and chickpea wilt through solarization of Vertisols. We also found that the population of plant parasitic nematodes is drastically reduced. India is endowed with plenty of sunshine and it depends on our own innovativeness as to

how much of this free energy we use. It is true that the cost of polyethylene sheets today is probibitive, but can we do something to reduce it? Can we identify situations where the cost-benefit ratio is favourable even with the present high cost of polyethylene? In these calculations we must take into account that solarization not only controls many soilborne diseases, but also offers other advantages such as improved soil fertility, weed and insect control and, of course, significantly higher yields of crops.

Intercropping and rotation, though out of fashion at present, cannot, and should not, be forgotten. These are centuries-old practices that have stabilized our agriculture. There contributions to disease control in legumes have not been meausred but are well-recognized. In many areas of India, sorghum-pigeonpea intercropping is an age old practice and for very good reasons, one of which is the significant reduction of Fusarium wilt of pigeonpea (Natarajan *et al.*, 1984). Likewise, where the inoculum does not survive too long in soil, it is possible to obtain substantial reduction of disease incidence through crop rotation. Even though we find that crop rotations are recommended routinely for most soilborne diseases, it is surprising for how few such diseases experimental information is available. A few years ago I would have accepted crop rotation as a possible management practice for the Fusarium wilt of chickpea, but not after we found that the causal fungus can survive for more than 6 years in the deep Vertisols (ICRISAT, 1985).

Planting short duration pigeonpeas offers us the benefit of reduced Fusarium wilt incidence and avoidance of frost damage.

BIOLOGICAL CONTROL

This has been talked about for a long time, but it is only recently that its potential as practical tool is being realized seriously. Some laboratories in India have intensified their research in this area. *Trichoderma* and some bacteria can be used in the control of a few soilborne pathogens, provided production and delivery systems are worked out. We need not wait for foreign technology to arrive on the scene. We can take the initiative and in collaboration with chemical engineers develop our own production and delivery systems. I would strongly recommend reading the article by Papavizas (1985) for those who have an inner urge to work on biological control. Most pulse crops have their own share of soilborne disease problems and research on biocontrol offers opportunities to manage some of these.

Recently at ICRISAT we observed that a vesicular-arbuscular mycorrhiza can reduce the incidence of Phytophthora blight on pigeonpea. We are fortunate that we have at least two centres (Bangalore and Hissar) where research on mycorrhizae is in progress. We must make use of this resource and critically examine relationships between mycorrhizae and diseases of pulse crops.

CHEMICAL CONTROL

A large number of papers have appeared recently in the literature indicating the possibility of managing some of the pulse crop diseases through the use of chemicals. We must realize that chemicals have a place in effective disease management. Where host resistance fails or where it is not of a high level, to manage diseases better we have

to integrate other methods in the total package. For example, today Ascochyta blight and Botrytis gray mold of chickpea cannot be managed through host resistance alone. Both fungi are seed-borne. Recent research (Reddy *et al.*, 1982; Grewal, 1982) has indicated that it is possible to eradicate the seedborne inoculum through appropriate seed dressing fungicides.

Resistance to *Phytophthora* is not yet available in pigeonpea cultivars. We must think of combining good soil drainage with the use of foliar sprays of metalaxyl when necessary. We should be able to control seedling diseases of lentil through seed dressing with appropriate fungicides. If sterility mosaic resistance is not available in a locally adapted and high yielding cultivar of pigeonpea, there should be no hesitation in managing the disease by controlling its vector, the eriophyid mite *Aceria cajani* through the use of appropriate pesticides. Thus I see no reason to be discouraged from working on chemical control of pulse crop diseases, even after hearing remarks by plant breeders, agronomists, and administrators, that chemical control will not be acceptable to farmers. I can tell you, from my personal experience, that farmers often show less resistance to the adoption of chemical control measures than our own fellow scientists and administrators!

VIRAL DISEASES

Although I have alluded to viral diseases in the preceding paragraphs, I wish to speak a little more on the subject. In spite of the small number of pathologists working on viral diseases of pulse crops, we have done well in generating information on most of the pathological aspects such as surveys, symptomatology, host range, properties of the virus in sap, methods of transmission including the identification of arthropod vectors, host resistance, vector control and, to some extent, diagnosis based on serology. Of the papers published between 1979 through 1985, more than 50% on black gram, 40% on cowpea, and 35% on green gram relate to viral diseases. However, we have not been able to do a good job of research in the identification and relationship of viruses based on electron microscopy, in identifying virus strains, or in evolving good purification procedures. Wherever possible, we should seek active collaboration with Indian virologists who are working in western countries where good facilities for basic virology work exist.

CONCLUDING REMARKS

I requested our library to do a literature search for this lecture to give me a list of publications by Indian pathologists working in India, on different pulse crops published between 1979 through 1985. This revealed that 531 papers have been published but some are undoubtedly duplicates. Their cropwise break up Is : chickpea 110, green gram 92, pea 89, cowpea 81, pigeonpea 77, black gram 58, lentil 13, moth bean 6, and horse gram 5. I have not compared these statistics with any cereal or other crops but even with no comparison, one can see how inadequate the research effort has been. These research papers include *all* aspects, some of which I did not allude to in my lecture (e.g. seed pathology, biochemical studies, etc.).

The contribution of pulses to our total food grain production is estimated to be about 10% (personal communication--Dr. R. N. Bhargava). If my lecture contributes, at

least in a small way, towards increasing the number of Indian Phytopathological Society members working mainly on pulse crop diseases to 10% (120 out of about 1200 members, which would require a 6-fold increase over the current strength), I would consider my effort today worthwhile and rewarding. Many of you may not know that we have been importing pulses to the tune of 200,000 tonnes each year for the last few years. My calculations indicate that this must be costing us at least Rs. 40 crores which is very close to the loss caused by pigeonpea wilt alone. If we succeed in managing even one serious disease of each of the pulse crops, we can help stop the import. That would be a contribution that we pathologists can easily make. We must work closely with agronomists, breeders, and entomologists to make India fully self-sufficient in its requirements of pulses.

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