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Discussion

M. Aslam
What are the planting and harvesting time for spring and winter planted chickpeas and their relation with rainfall?

G.C. Hawtin
Planting of winter chickpeas in Syria depends on when the rains start; any time from October to December. Spring planting takes place towards the end of the rainy season in late February or March.

M. Aslam
Do you experience any mortality from wilt at crop maturity*time due to shortage of soil moisture?

K.B. Singh
Wilt and root rot disease complex makes comparatively more damage to spring sown crop than winter sown crop. However, the extent of damage by these diseases is not serious at the moment.

M. Kamal
Table 2 shows that the yield in winter planting even with local material can be upto 1700 kg/ha. Does that mean that we can avoid Ascochyta blight by planting at optimum date in certain location even by using susceptible cultivar?

G.C. Hawtin
Yields can be high on susceptible materials if there is no disease. When disease is severe, however, there can be zero yield. Delaying the planting date reduces the risk of disease, but the yield potential is also considerably lower.

A. Telaye
There are climatological differences of winter season in between ICARDA's working mandatory regions and Ethiopia. How could one reconcile the differences in integrating research activities?

G.C. Hawtin
In Ethiopia ascochyta blight has been reported to be a problem. Normally the crop is planted there, as here, at the end of the rainy season. Earlier planting during the summer rains in Ethiopia is analogous to winter planting, and it too may result in better use of available moisture.

CP03

Proceedings of the Workshop on Ascochyta Blight and Winter Sowing of Chickpeas (Saxena, M.C. and Singh, K.B., eds.), ICARDA, 4-7 May 1981, Aleppo, Syria

Second Session: Ascochyta Blight

A Review of Ascochyta Blight of Chickpea (*Cicer arietinum* L.)

Y.L. NENE
Leader, Pulses Improvement Program,
ICRISAT, Patancheru, A.P., INDIA

Chickpea (*Cicer arietinum* L.) is an important grain legume crop of dryland agriculture in Asia, Africa and Central and South America. The total cultivated area of chickpea in the world is about 10.4 million hectares and annual production is about 6.8 million tonnes (FAO 1978). The average yields per hectare are estimated to be around 700 kg. Chickpea is known by other names such as Bengal gram, gram, Egyptian pea, Spanish pea, Chestnut bean (all English), pois chiche (French), chana (Hindi), homos (Arabic), grao-de-bico (Portuguese), garbanzo or garavance (Spanish), etc.

Ascochyta blight is considered to be one of the most important diseases of chickpea. Severe epidemics of this disease have been reported from many chickpea-growing countries The very fact that the present workshop deals in a major way with ascochyta blight bears testimony to its international importance. Since the objective of the workshop is to ascertain the present status of knowledge and to identify high priority areas of research for the immediate future, a review of the available literature on the ascochyta blight of chickpea is presented in this paper.

Historical

Ascochyta rabiei (Pass.) Lab., the causal fungus of the blight, was first named *Zythia rabiei* by Passerini in 1867 on the basis of unicellular and hyaline pycni-

2. Reproduction

Asexual. The asexual or imperfect stage of the fungus is characterized by the production of the fruiting bodies (pycnidia) which produce spores (pycnidiospores). Pycnidia are visible as minute dots in the lesions produced on the host. Pycnidia are immersed, amphigenous, spherical to subglobose or depressed and generally vary in size from 65 to 245 u (Sattar 1934). Pycnidiospores (also called spores or conidia) are oval to oblong, straight or slightly bent at one or both ends, hyaline, occasionally bicelled, 8.2 to 10.0 x 4.2 to 4.5 μ. Kovachevski (1936a) reported the spore size to be 6.0 to 16.0 x 3.4 to 5.6 μ on host and 4.8 to 14.0 x 3.2 to 5.2 μ on an artificial medium.

Colonies of the fungus on artificial media (e.g., oat meal agar) are flat, submerged, with sparse mycelium, white at first and later turning dark and fumaceous. Bedi and Aujla (1970) reported that pycnidia developed best at pH 7.6 – 8.6 at 20°C on Richards' medium of double concentration. Besides oat meal agar, chickpea seed meal (4-8%) agar has been found to be a good medium for the growth of the fungus and pycnidial production (Kaiser 1973; Reddy and Nene 1979). Optimum temperature for growth, pycnidial production and spore germination has been found to be around 20°C (Bedi and Aujla 1970; Chauhan and Sinha 1973; Kaiser 1973; Maden *et al.* 1975; Zachos *et al.* 1963). Temperatures below 10°C and above 30°C have been found unfavourable to the fungus (Chauhan and Sinha 1973; Kaiser 1973; Luthra and Bedi 1932). Light affects growth of the fungus on artificial media. Kaiser (1973) reported that continuous light resulted in increased sporulation. Chauhan and Sinha (1973) reported reduced sporulation on infected plants in a glasshouse when continuous light was given. My own experience in ICRISAT supports Kaiser's findings. The incubation period between inoculation of plants and appearance of symptoms varies between 5 and 7 days depending on the temperatures provided (Chauhan and Sinha 1973; Zachos *et al.* 1963). It also varies with genotypes inoculated.

Sexual. Kovachevski (1936a) was the first worker who observed the sexual stage of the fungus (in Bulgaria) and named it *Mycosphaerella rabiei* Kovachevski. The fruiting bodies, perithecia, were found exclusively on chickpea refuse, especially the pods, that had overwintered in the field. They were dark brown or black, globose or applanate, with a hardly perceptible beak and ostiole and were 76 to 152 x 120 to 250 μ in size. The asci were cylindrical-clavate, more or less curved, pedicellate and 48 to 70 x 9 to 13.7 μ in size. The ascospores (8 per ascus) were monostichous, rarely distichous, ovoid, divided into two very unequal cells, strongly constricted at the septum and measured 12.5 to 19 x 6.7 to 7.6 μ. Subsequently, Gorlenko and Bushkova (1958) confirmed the presence of the perfect stage in the USSR, and Zachos *et al.* (1963) in Greece. Obviously, conditions in eastern Europe and western Asia are favorable for the production of the perithecial stage. If a cold winter is a prerequisite for the production of perithecia, one may not observe these in agroclimatic regions represented in the Indian subcontinent where hot summers follow the chickpea season. It is well known that the presence of the perfect stage has a bearing on the production of new races.

3. Races

There have been very few studies on races. Luthra *et al.* (1939) and Arif and Jabbar (1965) did not find any evidence of the existence of races. A report from India (Anonymous 1963) stated that the cultivar C-12/34 lost its resistance probably due to a new race. Bedi and Aujla (1969) studied variation in the fungus isolates under controlled conditions. On the basis of symptomatology, manner of pycnidial formation on the host, and pathogenic behavior of 11 isolates, they concluded that several races exist in the state of Punjab in India. Vir and Grewal (1974b) identified two races (1 and 2) and one biotype of the race 2 using I-13, EC-26435, C-235, F-8 and V-138 cultivars as differentials. Recently Singh *et al.*, (1981) obtained indications of the existance of races through results obtained from the Chickpea International Ascochyta Blight Nursery. Intensified race studies to obtain a full picture of the race situation are needed if stable host resistance to ascochyta blight is to be achieved.

Epidemiology

1. Survival

The fact that there are so many reports of epidemics of this blight clearly indicates the existence of efficient mechanisms for the survival of the fungus from one season to another. Several workers have studied this aspect and reported that the fungus survives mainly in the diseased crop debris and in seeds from infected plants.

Crop Debris. The above-ground parts of the plants are infected and pycnidia are produced on these infected parts. Sattar (1933) could not determine the absolute importance of infected crop debris in fungus survival. Later, Luthra *et al.* (1935) considered infected debris to be an important source of primary infection in the following season because they found that the fungus survived for 2 years in infected tissues. However, they pointed out that the fungus will not survive if the infected debris is buried in moist soil at only 5 cm depth. Kaiser (1973) carried out systematic studies and confirmed that the fungus survived for over 2 years in naturally infected tissues at 10-35°C and 0.3% relative humidity at the soil

surface. However, the fungus lost its viability rapidly at 65-100% relative humidity and at soil depth of 10-40 cm.

This aspect of survival needs further attention. The fungus apparently survives in debris if conditions are dry and if the debris lies close to the soil surface. Which are those geographical regions where the climate between two chickpea season is dry? In such areas this particular mode of survival will be important. On the other hand, in countries such as India, infected crop debris should be of no importance because the chickpea season is followed by a monsoon season and the wetness of soil should not permit fungus survival in crop debris. However, does this actually happen in nature in India? We have no definite answer as yet.

Some interesting work on this aspect has been done in Pakistan by Kausar (1965). He studied the influence of winter rainfall during the chickpea-growing season (October to April) and of the preceding summer rainfall (May to September) on the development of epidemics. He studied correlations between the incidence of blight (percentage of crop area failed due to blight in Campbellpur subdistrict) and winter rainfall during the chickpea-growing season (October to April) and the preceding summer rainfall (May to September) in respect of the years 1906-1941. These studies revealed that years of high chickpea season rainfall coincided with a high incidence of blight. The incidence of blight was more than 50% during 15 years that received on an average more than 150 mm of rainfall. More than 150 mm rainfall was received in 26 years out of 35 and the incidence of blight was more than 10% during the 27 years periods. In another analysis it was found that chickpea seasons with low incidence of blight were followed by a summer of high rainfall. The correlation, however, was nonsignificant.

Seed. A good deal of research work has been done on the survival of the fungus through seed. Luthra and Bedi (1932) were probably the first to demonstrate the seed-borne nature of the pathogen. They showed that the seed coat and cotyledons of infected seeds contained mycelium and that the infected-seed weight was less than healthy-seed weight. Halfon-Meiri (1970) confirmed the presence of the fungus in the seed coat and cotyledons, and of pycnidia in lesions. Sattar (1933) demonstrated the surface contamination of seed with fungus spores and their role in causing infection. He found that 50% of such spores survived on seed for 5 months at 25-30°C, but only 5% of spores survived for 5 months at 35°C. Zachos (1952), Gobelez (1956) and Khachatryan (1961) also confirmed the seed-borne nature of the pathogen.

Lukashevich (1958b) showed that the fungus can behave as a saprophyte and spread to noninfected tissues if the harvested material is stored for some time before threshing. He found 1.5 to 2-fold increases in seed infection during prethreshing storage. Maden *et al.* (1975) carried out a detailed study in Denmark on the seed samples received from Turkey. They found that 70% of this

seed from Central Anatolia was infected by *A. rabiei*. The inoculum occurred as spore contamination and mycelium in the seed coat alone or in the seed coat and embryo. Pycnidia were observed only in the seed coat of seeds having deep lesions. Whole-mount preparations and microtome sections showed that the inter- and intra-cellular mycelium was localized in lesions. Pycnidia were subepidermal and contained mature spores. Pycnidiospores obtained from the seed surface and pycnidia from 14-month old seed stored at 3° ±1°C, showed 33% germination. They established that both superficial and deep infections were equally potent in the transmission of the disease.

All these studies considered together clearly establish the role of seed in perpetuating the fungus from one season to the next.

2. Spread

The spread of the disease has been attributed to the pycnidiospores produced at the foci of primary infection, either through crop debris or infected seed. Most workers seem to agree that temperatures of 20-25°C are best for the build up of infection (Askerov 1968; Chauhan and Sinha 1973; Zachos *et al.* 1963). Chauhan and Sinha (1973) in a glasshouse study found 85-98% relative humidity and 20°C temperature to be most favorable, provided this humidity was maintained for at least 84 hours. They found the incubation period under these conditions to be 6 days. Khachatryan (1962), working in Armenia, reported over 60% relative humidity, with 350-400 mm rain during summer and an average daily temperature of not less than 15°C, to be congenial for the incidence and spread of the disease. According to Luthra *et al.* (1935) the primary infection foci in a field are limited and isolated, but windy and wet conditions help in the rapid spread of the disease. They suggested that infected debris, broken off from brittle diseased plants, could be transported by wind for several hundred meters. Disease spreads rapidly if wet and windy conditions occur in February and March when temperatures are around 22-26°C.

Everyone knows that this disease spreads rapidly, sometimes too rapidly, and causes epidemics in extensive areas. Existing information on the epidemiology does not fully explain the occurrence of widespread epidemics of this disease at different times in different years and in some years but not in others, in spite of favorable weather.

3. Host Range

Most workers have reported *Cicer* spp. to be the only hosts of *A. rabiei* (Bondartzeva- Monteverde and Vassilievsky 1940; Gorlenko and Bushkova 1958; Sprague 1930). However, Kaiser (1973) reported that the fungus could infect cowpea (*Vigna sinensis*) and bean (*Phaseolus vulgaris*) when inoculated artificially. He

observed small reddish brown spots on the stems, petioles and leaves of cowpea and on the leaves of bean, but the lesions did not increase in size. However, Sprague (1930) found no symptoms on *Phaseolus vulgaris* when inoculated artificially. Kaiser's finding is very interesting and needs to be confirmed. Information on other hosts of *A. rabiei*, if any, is lacking and research efforts in this direction need to be intensified.

Control

Measures to control this disease have been sought ever since it was first described. Measures that have been claimed to be effective are (a) utilizing host resistance, (b) adopting cultural control practices including sanitation, and (c) using chemicals to treat seeds and for foliar application. Literature on these aspects is briefly reviewed below.

1. Host Resistance

This aspect is discussed in greater depth in another paper in this workshop. Many reports on the identification of resistance can be seen in the literature.

Screening Techniques. Labrousse (1931) was perhaps the first scientist who made an effort to identify resistance through artificial inoculations. He scattered infected tissues on test plants and carried out repeated sprinklings with an aqueous suspension of spores. Luthra *et al.* (1938) repeated what Labrousse (1931) had done except that they used infected debris from the previous year to scatter on the test plants. Sattar (1933) had earlier suggested that the best time to carry out inoculations was when plants were flowering and podding. Sattar and Hafiz (1951) suggested broadcasting small bits of blighted plants on test plants after ensuring that the infected debris contained viable pycnidiospores. According to these workers, infection occurred after rain even if it were received months after inoculation. They claimed the method to be as effective as that in which aqueous suspensions of spores were applied. Vedysheva (1966) suggested spreading infected debris over soil both in autumn and spring. Taking a clue from the methods described above, Reddy *et al.* (1980) worked out an efficient field screening procedure. This involved (a) planting a row of susceptible line after every 2-4 test rows, to serve as an infector row, (b) spraying plants with a spore suspension prepared from diseased plants, (c) scattering infected debris collected in the previous season, and (d) maintaining high humidity through sprinkler irrigation.

Reddy and Nene (1979) used a glasshouse procedure for screening germplasm. This involved the use of an Isolation Plant Propagator (Burkard Manufacturing Co. Ltd., Rickmansworth, Herts, England). Ten seedlings of each germplasm line were grown in one pot. Two-week old seedlings were inoculated by spraying them with an aqeuous suspension of spores (20,000 spores/ml). Humidity was maintained by covering the plants with plastic covers for 10 days. This method proved very useful for confirming field results.

Disease Rating Scales. Vir and Grewal (1974b) suggested a 5-point scale for use in pot screenings as follows:

0 = No infections;
1 = A few minute localized lesions on stem and/or up to 5% foliage infection;
2 = Stem lesions 2-6 mm long which may girdle the stem and/or 5-25% foliage infection;
3 = Stem lesions bigger than 6 mm and girdling the stem and/or 25-75% foliage infection;
4 = All young shoots and leaves killed.

They (Grewal and Vir 1974) also suggested that the same scale be used in field screening.

Morall and McKenzie (1974) developed a 6-point scale for use in the field, as follows:

0 = No lesions visible on any plant in the plot;
1 = A few scattered lesions on the plants, usually found only after careful searching;
2 = Lesions common and readily observed on plants, but defoliation and damage not great, or in only one or two patches in plot;
3 = Lesions very common and damaging, severity intermediate between 2 and 4;
4 = All plants in plot with extensive lesions, defoliation and dying branches; but few, if any, plants completely killed;
5 = All plants, or all but parts of a few, completely killed.

Singh *et al.* (1981) extended the scale to 9 points having five defined categories of severity, as follows:

1 = No disease visible on any plant (highly resistant);
3 = Lesions visible on less than 10% of the plants, no stem girdling (resistant);
5 = Lesions visible on up to 25% of the plants, stem girdling on less than 10% of the plants but little damage (tolerant);
7 = Lesions present on most plants, stem girdling on less than 50% of the plants, resulting in the death of a few plants and causing considerable damage (susceptible);
9 = Lesions profuse on all plants, stem girdling present on more than 50% of the plants and death of most plants (highly susceptible).

This scale has been used by them for evaluating materials in a large-scale breeding program.

Reddy and Nene (1979) developed a 9-point scale for greenhouse screening in a propagator, as follows:

1 = No lesions;
2 = Lesions on some plants, usually not visible;
3 = A few scattered lesions, usually seen only after careful examination;
4 = Lesions and defoliation on some plants, not damaging;
5 = Lesions common and easily observed on all plants but defoliation/damage not great;
6 = Lesions and defoliation common, few plants killed;
7 = Lesions very common and damaging, 25% of the plants killed;
8 = All plants with extensive lesions causing defoliation and the drying of branches, 50% of the plants killed;
9 = Lesions extensive on all plants, defoliation and drying of branches, more than 75% of the plants killed.

Each of these rating scales has merit; however, there is a need to further simplify the rating scale and adopt a uniform scale for use by all research workers.

Sources of Resistance. Many reports on identification of resistance to ascochyta blight have appeared in the literature during the last 50 years. Many of these reports were based on observations made during natural epidemics while several were based on artificial inoculation tests in the field or in greenhouses. The majority of the reports are from the Indian subcontinent (Ahmad *et al.* 1949; Anonymous 1963; Aziz 1962; Bedi and Athwal 1962; Grewal and Vir 1974; Luthra *et al.* 1938; Padwick 1948). One of the cultivars that was identified as resistant was 4F32 (renamed F-8 by Luthra *et al.* 1938) which was traced to France. Subsequently, C-12/34 became a popular resistant cultivar and was obtained by crossing F-8 with Pb-7. Padwick (1948) noted that the resistance of F-8 remained effective. Around 1950, C-12/34 "lost" its resistance, but another resistant cultivar C-235 was developed and made available to farmers (Anonymous 1963). Aziz (1962) reported C-727 to be resistant, Grewal and Vir (1974) identified P-1528-1-1 (from Morocco) as immune and I-13 (from Israel) as resistant, and Singh (1978) reported resistance in Galben (from Rumania), E.C.-26414, -26435 and -26446. However, these sources of resistance have apparently not been used by breeders so far.

From regions other than the Indian subcontinent one finds fewer reports of resistance. Solel and Konstrinski (1964) identified the cultivar "Bulgarian" as immune and Kaiser (1972), working in Iran, found one black-seeded accession from Israel highly resistant to Iranian isolates of the fungus, but not to isolates from Pakistan. It is not certain if I-13 of Grewal and Vir (1974) is the same as the black-seeded accession of Kaiser (1972). Radkov (1978) reported from Bulgaria no. 180 and no. 307 to be resistant, high yielding and suitable for mechanical

cultivation. Also from Bulgaria, Ganeva and Matsov (1977) reported the cultivars Sovkhoznyi 14, Kubanskii 199, VIR-32, no. 222 (from the USSR) and Resusi 216 to be highly resistant.

With the inclusion of chickpea in the mandate of ICRISAT and subsequently in that of ICARDA, it has now become possible to carry out a systematic resistance breeding program on a wide scale and good progress has already been made. This work will be covered in another paper of this workshop.

It is important to identify good reliable sources of resistance, but what is more important is to use these sources to combine resistance with high yield.

Inheritance of Resistance. All the reports published so far (Eser 1976, Hafiz and Ashraf 1953; Vir *et al.* 1975) indicate that the resistance is governed by a single dominant gene. Thus incorporation of resistance into a high-yielding background should be fairly simple and easy.

Mechanism of Resistance. Sattar (1933) considered that more malic acid secreted by leaves at flowering/podding time favored infection. In contrast, however, Hafiz (1952) claimed that a resistant cultivar (F-8) secreted more malic acid than a susceptible cultivar (Pb-7) and that malic acid was inhibitory to spore germination and germtube development. Work carried out at ICRISAT (Reddy and Nene, unpublished) has not confirmed Hafiz's claim.

Hafiz (1952) found no difference in cuticle thickness between resistant and susceptible types, but found higher numbers of stomata in resistant types. Very little difference was found in the acidity of sap collected from resistant and susceptible types.

Ahmad *et al.* (1952) reported that resistant types (F-8 and F-10) were significantly taller, possessed a large number of hairs per unit area of stem and leaf, and had a smaller number of tertiary branches than the susceptible types (Pb-7, C-7).

In a series of papers Vir and Grewal (1974a; 1974c; 1975a; 1975b) compared biochemically a resistant cultivar (I-13) with a susceptible cultivar (Pb-7). They found that the resistant cultivar showed (a) higher peroxidase activity, (b) higher L-cystine content and (c) more phenolic content and higher catalase activity after inoculation. According to them, these biochemical differences should explain the resistance of I-13.

2. Cultural Practices

Sattar (1933) suggested the removal and destruction of dead plant debris, crop rotation, and deep-sowing of seed to prevent infected seeds from emerging, as methods to reduce the blight. Luthra *et al.* (1935), in addition to sanitation, suggested intercropping chickpea with wheat, barley, mustard (*Brassica campestris*), etc. to reduce disease spread in the crop season. Lukashevich (1958a)

suggested the application of potassium fertilizers to reduce disease severity. Reddy and Singh (1980) reported no effect of inter-row spacings on disease incidence. Adopting specific cultural practices will help, particularly when there is group action by all the farmers of a region.

3. Fungicides

Several reports on the use of chemicals for seed dressing and foliar spraying have appeared in the literature.

Seed Dressing. Sattar (1933) reported good control with the immersion of seed for 10 minutes in 0.5% copper sulphate, or the presoaking of seed in water at 20°C for 6 hours followed by immersion in hot water (53°C) for 15 minutes. Zachos (1951), however, found that hot water treatment adversely affected seed germination. He found that a 2-hour immersion of seed in malachite green (0.005%) or a 4-hour immersion in formalin eradicated seed-borne inoculum. Zachos *et al.* (1963) subsequently found that a 12- hour immersion in pimaracin (150 μ/ml) eradicated the inoculum completely. Various fungicides have been reported to reduce seed-borne inoculum. These include Granosan (Lukashevich 1958a), Phenthiuram (Ibragimov *et al.* 1966), thiram (Khachatryan 1961), benomyl (Kaiser *et al.* 1973) and Calixin M (Reddy 1980). Calixin M (11% tridemorph + 36% maneb) seems to eradicate the seed-borne inoculum completely, and this offers an excellent opportunity to treat the seed effectively. The need to find an effective and simple seed treatment cannot be overemphasized. On the one hand, such a treatment will be useful in controlling the disease and, on the other, it will facilitate free international movement of seed.

Foliar Sprays. Foliar applications of various fungicides have been reported to reduce disease spread significantly. These fungicides include Bordeaux mixture (Kovachevski 1936), wettable sulphur (Lukashevich 1958a), zineb (Solel and Kostrinski 1964), ferbam (Puerta Romero 1964), maneb (Retig and Tobolsky 1967), captan (Vir and Grewal 1974d) and Daconil (Se, Nycirek *et al.* 1977). Foliar sprays are generally ineffective under epidemic situations. Even under moderate disease situations, four to six sprays become necessary to significantly reduce the disease. The rapidity with which the disease spreads makes it very difficult to follow the application schedule. It is obvious that foliar application with presently available fungicides has limited scope at present.

Looking Ahead

It is proposed that, in the near future, the scientists working on this disease should address themselves to the following questions:

1. Sexual reproduction (perfect stage) occurs in *A. rabiei*. What are the conditions under which this stage is produced? What is its role, if any, in producing new races?
2. Is the available evidence on the existence of races of *A. rabiei* satisfactory? Should research work be intensified to get a complete global picture of the occurrence of races of this fungus?
3. To what extent does the diseased crop debris play a role in the perpetuation of *A. rabiei*? Does it play a role in some regions but not in others?
4. The role of infected seed in the perpetuation of *A. rabiei* is established beyond doubt. Is it important to determine the numerical threshold value (minimum percentage of seed infection) required for initiating an epidemic under favourable weather conditions? Is Calixin M seed dressing adequate to eradicate seed-borne inoculum? Is it likely to help in controlling the disease later in the season?
5. How does the disease spread? How far does the inoculum move? How is the occurrence explained of epidemics in large, geographically contiguous regions gions in certain years, but not in others?
6. Is *A. rabiei* specific only to the species of *Cicer*?
7. Is the efficacy of the resistance screening techniques satisfactory that have been developed so far? Are the presently used disease rating scales simple enough? Is there a need to develop a standard rating scale?
8. Is the performance satisfactory of "resistant" lines that have been identified so far?
9. Should a systemic fungicide be looked for that would control the disease with only one or two foliar sprays as a standby in case the resistance "breaks down"? As an example, such a fungicide is now available for controlling the downy mildews of several crops.
10. There is an increased interest now in growing chickpeas in non-traditional areas mainly because this crop requires low cultivation inputs. What steps should be taken to avoid introduction of *A. rabiei* into areas where it does not exist at present?

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Discussion

M. Aslam
What is your idea about the frequency of occurrence of ascochyta blight in Punjab (India), that is, whether the disease is increasing there or decreasing?

Y.L. Nene
The disease was not actually increasing, but there are areas in Punjab (India) like Gurdaspur where the disease can occur and has been seen to occur.

T.S. Sandhu
There was a severe epidemic of blight during 1967-68, but after that it has not been observed.

K.B. Singh
In Punjab State of India blight epidemic in chickpea occurred in a form during the 1967-68 season that the farmers did not even harvest the crop. But the next year and subsequent years this disease was not even seen. So I feel that epidemiology is not fully understood and more needs to be known.

Y.L. Nene
I would like to say that there is no increase in the frequency of ascochyta blight epidemic in India. The disease is endemic in Gurdaspur, and depending upon weather conditions, it reaches epidemic proportions in certain years.