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A Review of Ascochyta Blight of Chickpea*

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Abstract. Chickpea (*Cicer arietinum* L.) is an important grain legume crop in Asia, Africa and Central and South America. Ascochyta blight caused by *Ascochyta rabiei* (Pass.) Lab. is one of the most serious diseases of the crop and severe epidemics have been reported. The available literature on Ascochyta blight is reviewed in order to ascertain the present status of knowledge of the disease and to identify the areas of research which need immediate attention. Distribution, yield losses, symptoms, taxonomy, reproduction, epidemiology and control of the disease are discussed. Further information on sexual reproduction, epidemiology and control is needed.

Introduction

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.

Of the several diseases recorded on chickpea, Ascochyta blight is considered to be one of the most important. Severe epidemics of this disease have been reported from many chickpea growing countries. A workshop was held at the International Centre for Agricultural Research in Dry Areas (ICARDA) in Aleppo, Syria in May 1981 to ascertain the present status of knowledge and to identify priority areas of research. This paper reviews the available literature on Ascochyta blight of chickpea.

Historical

Ascochyta rabiei (Pass.) Labr., the causal fungus of the blight, was first named *Zythia rabiei* by Passerini in 1867 on the basis of unicellular and hyaline pycnidiospores (Khune and Kapoor, 1980). According to Khune and Kapoor (1980), Passerini's diagnosis was either overlooked or not accepted by later workers. Comes in 1891 identified the fungus as *Ascochyta pisi* Lib. and Prillieux and Delacroix in 1893 named it *Phyllosticta cicerina* (Khune and Kapoor, 1980). After studying Saccardo's material, Trotter in 1918 concluded that the fungus was not *Ascochyta pisi* but resembled *Phyllosticta* and later proposed the combination *P. rabiei* (Pass.) Trotter (Khune and Kapoor, 1980). Gonzalez (1921) agreed with Trotter's proposal. Labrousse (1930) described the fungus as *Phyllosticta rabiei* because he saw no bicellular spores on the host, though a few were observed in culture. However, a year later Labrousse (1931) suggested that the fungus be called *Ascochyta rabiei* as it produced 2–4% single septate spores on artificially inoculated plants. Although taxonomists still differ in their opinion about the name.

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Ascochyta rabiei is the name accepted by the majority of workers. The disease has always been considered as economically important. Perhaps the best documented account of blight epidemics exists for the former Punjab province of British India, a part of which is now in Pakistan, where the disease was first observed in 1911 (Butler, 1918). Records of subsequent epidemic years have been reviewed by Kausar (1965).

Geographical distribution

The disease has been reported from the following 26 countries: Algeria, Australia, Bangladesh, Bulgaria, Canada, Cyprus, Ethiopia, France, Greece, India, Iran, Iraq, Israel, Italy, Jordan, Lebanon, Mexico, Morocco, Pakistan, Romania, Spain, Syria, Tanzania, Tunisia, Turkey and the USSR (Nene, 1980). The disease is more frequently observed in Algeria, Bulgaria, Cyprus, Greece, Iraq, Israel, Jordan, Lebanon, Morocco, Pakistan, Romania, Spain, Syria, Tunisia, Turkey and the USSR.

Losses

There are many reports of serious losses caused by Ascochyta blight. A few examples will suffice. Labrousse (1930) reported that the disease was very destructive in Morocco in 1929. In what is now Pakistan, investigations on the disease were initiated in 1922 and it was found that annually 25–50% of the crop was destroyed (Sattar, 1933). According to Kovachevski (1936) 20–50% of the crop was lost annually in Bulgaria, while occasionally total loss occurred in certain fields. In the Dnepropetrovsk region of USSR, blight was severe in 1956 "sometimes causing 100% loss" (Nemlienko and Lukashevich, 1957). In Greece, 10–20% damage was reported during 1957–1958 (Demetriades *et al.*, 1959). According to Puerta Romero (1964) the disease "causes great losses of chickpea wherever it is grown in Spain". In Pakistan about 70% of the crop was lost in 1979 and 1980 (personal communication, Dr B. Ahmed Malik). Anyone who has seen the disease in the field will readily agree on the potential of this disease to cause serious losses in chickpea. In fact, epidemics of the disease have been reported by several workers (Benlloch 1941; Biggs, 1944; Kaiser, 1972; Kausar, 1965; Radulescu *et al.*, 1971; Zalpoor, 1963).

Symptoms

Several workers have described the symptoms of the disease as it occurs in different countries (Atanasoff and Kovacevski, 1929; Benlloch and Del Canizo, 1931; Labrousse, 1930; Luthra and Bedi, 1932). The descriptions are remarkably similar. All above-ground parts of the plant are attacked. On leaflets the lesions are round or elongated, bearing irregularly depressed brown dots, and are surrounded by a brownish red margin. On the green pods the lesions are usually circular with dark margins and have pycnidia arranged in concentric circles. Often the infected seeds carry lesions. On the stem and petiole, the lesions are brown, elongated (3–4 cm), bear black dots and often girdle the affected portion. When lesions girdle the stem, the portion above the point of attack rapidly dies. If the main stem is girdled at the collar region the whole plant dies. As the disease advances, patches of diseased plants become prominent in the field and slowly spread, involving the entire field. Labrousse (1930) stated in his description that "seeds do not appear to be attacked". This is certainly incorrect, and it is puzzling that he did not observe lesions on seeds. Recently Haware and Nene (1981) described a blight caused by *Phoma medicaginis* Malbr. & Roum. Symptoms of this blight resemble Ascochyta blight symptoms, but pycnidia in concentric circles are not present in the case of Phoma blight.

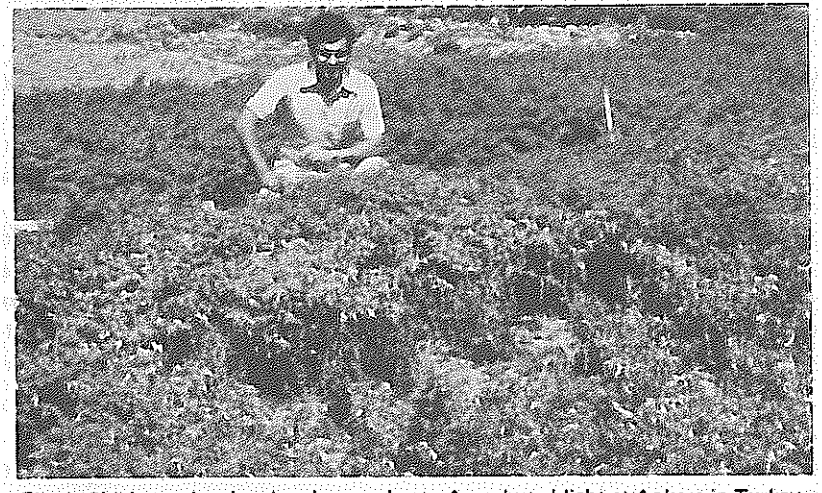

Fig. 1. Chickpea plot showing damage due to Ascochyta blight at Ankara in Turkey.

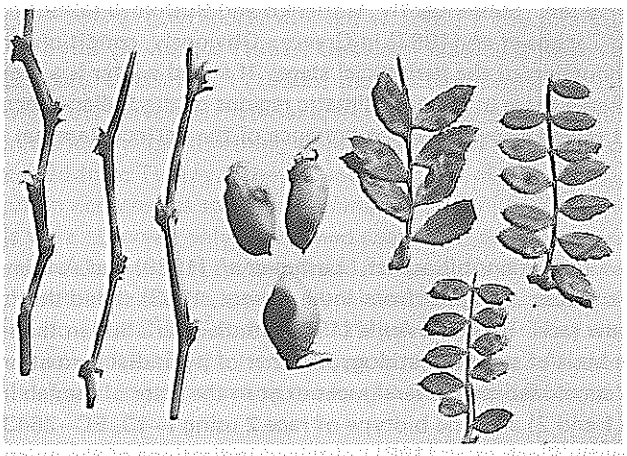

Fig. 2. Symptoms of blight on different parts of affected plants.

Pathogen

Taxonomy

Some taxonomic information has been given earlier under "historical". Although a large number of workers accept *Ascochyta rabiei* as the name of the fungus, some workers do not agree. They prefer the use of *Phyllosticta rabiei* (Luthra and Bedi, 1932; Aujla, 1960). Recently Khune and Kapoor (1980) suggested that the fungus be called *Phoma rabiei* (Pass.) on the basis of the presently accepted concept that *Phoma* contains about 5% bicelled pycnidiospores. The Commonwealth Mycological Institute, however, continues to call the fungus *Ascochyta rabiei*.

Reproduction

Asexual

The asexual or imperfect stage of the fungus is characterised 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 μm (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 μm. Kovachevski (1936) reported the spore size to be 6.0 to 16.0 x 3.4 to 5.6 μm on host and 4.8 to 14.0 x 3.2 to 5.2 μm 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. Observations at ICRISAT support 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 (1936) 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, which 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 μm in size. The asci were cylindrical-clavate, more or less curved, pedicellate and 48 to 70 x 9 to 13.7 μm in size. The ascospores (8/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 μm. 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 favourable 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 the agroclimatic regions of 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.

Races

There have been very few studies on this aspect. Luthra *et al.* (1939) and Arif and Jabbar (1965) did not find any evidence of the existence of races. A report from India (Anon., 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 behaviour 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 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 existence of races through results obtained from the Chickpea International Ascochyta Blight Nursery. Studies of races will have to be intensified to obtain a full picture of the race situation if we are to obtain stable host resistance to Ascochyta blight.

Epidemiology

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 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 two 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 two 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 depths of 10–40 cm. It is clear to me that 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. In the geographical regions where the climate between two chickpea seasons is dry this particular mode of survival will be important. However, 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. Does this actually happen in nature in India? There are no definite answers 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 to 1941. These studies revealed that years of high chickpea season rainfall coincided with 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-year period. 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 not significant.

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 five months at 25–30°C, but only 5% of spores survived for five 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 non-infected tissues if the harvested material is stored for some time before threshing. He found 1.5–2-fold increases in seed infection during the pre-threshing storage. Maden *et al.* (1975) carried out a detailed study in Denmark on 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 localised 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.

Spread

The spread of the disease has been attributed to the pycnidiospores produced at the foci or 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 to be most favourable, provided this humidity was maintained for at least 84 h. 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 metres. Disease spreads rapidly if wet and windy conditions occur in February and March when temperatures are around 22–26°C.

It is well known that this disease spreads rapidly and causes epidemics in extensive areas. I believe the information that exists to date on 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 favourable weather.

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 unguiculata* (L.) Walp.) and bean (*Phaseolus vulgaris* L.) 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 *P. 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 using host resistance, adopting cultural control practices including sanitation and using chemicals to treat seeds and for foliar application. Literature on these aspects is briefly reviewed in the following paragraphs.

Host resistance

Screening techniques

Labrousse (1931) was perhaps the first scientist to make 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) published a paper describing their field inoculation procedure. They 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 was 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 and Reddy *et al.* (1980) worked out an efficient field screening procedure from the results of workers. This involved 1. planting a row of a susceptible line after every 2–4 test rows to serve as an infector row, 2. spraying plants with a spore suspension prepared from diseased plants, 3. scattering infected debris collected in the previous season, and 4. maintaining high humidity through sprinkler irrigation.

Reddy and Nene (1979) used a glasshouse procedure for screening germplasm. This involved the use of Isolation Plant Propagator (Burkard Manufacturing Co. Ltd, Rickmansworth, Herts, UK). Ten seedlings of each germplasm line were grown in one pot. Two-week old seedlings were inoculated by spraying them with an aqueous 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 infection; 1 = a few minute localised 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. 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 nine 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 programme.

Reddy and Nene (1979) developed a 9-point scale for glasshouse 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, we need to further simplify the rating scale and adopt a uniform scale for use by all chickpea research workers.

Sources of resistance

Many reports on the 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 glasshouses. A long list would be required if I were to attempt to mention all the cultivars that have been reported to be resistant. The majority of the reports are from the Indian subcontinent (Ahmad *et al.*, 1949; Anon., 1963; Azis, 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 (Anon., 1963). Azis (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 Romania), 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 Kostrinski (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 programme on a wide scale and good progress has already been made. It is important to identify good reliable sources of resistance, but it is more important 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 favoured 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 germ tube 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 and 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 higher peroxidase activity, higher L-cystine content, and more phenolic content and higher catalase activity after inoculation. According to them, these biochemical differences should explain the resistance of I–13.

Cultural practices

Sattar (1933) suggested the removal and destruction of dead plant debris, crop rotation and the 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* L.), etc. to reduce disease spread in the crop season. Lukashevich (1958a) suggested the application of potassium fertilizers (45 kg(?)/ha) to reduce disease severity. Reddy and Singh (1980) reported no effect of interrow spacings on disease incidence. Adopting specific cultural practices will help, particularly when there is group action by all the farmers of a region.

Fungicides

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

Seed dressing

Sattar (1933) was the first to make efforts to eradicate seed-borne inoculum. He reported good control with the immersion of seed for 10 min in 0.5% copper sulphate, or the pre-soaking of seed in water at 20°C for 6 h followed by immersion in hot water (53°C) for 15 min. Zachos (1951), however, found that hot water treatment adversely affected seed germination. He found a 2-h immersion of seed in malachite green (0.005%) or a 4-h immersion in formalin, eradicated seed-borne inoculum. Zachos *et al.* (1963) subsequently found that a 12-h immersion in pimaracin (150 µg/ml) eradicated the inoculum completely. Various fungicides have been reported to reduce seed-borne inoculum. These include Granosan (ethylmercury chloride) (Lukashevich, 1958a), thiram (Khachatryan, 1961), benomyl (Kaiser *et al.*, 1973) and Calixin M (11% tridemorph + 36% maneb) (Reddy, 1980). Calixin M 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 over emphasised. 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 (chlorothalonil) (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 reduce significantly 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

Based on this review it is thought that, in the near future, scientists working on this disease should address themselves to the following questions.

1. Sexual reproduction (perfect stage) occurs in *A. rabiei.* Do we know the conditions under which this stage is produced? What is its role, if any, in producing new races?

2. Are we satisfied with the available evidence on the existence of races of *A. rabiei?* How should we intensify research work 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? Are we satisfied that Calixin M seed dressing is 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 do we explain the occurrence of epidemics in large, geographically continguous regions in certain years, but not in others?

6. Is *A. rabiei* specific only to the species of *Cicer?*

7. Are we satisfied with the efficacy of resistance screening techniques 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. Are we satisfied with the performance of 'resistant' lines that have been identified so far?

9. Should we not look for a systemic fungicide which would control the disease with only one or two foliar sprays as a standby in case 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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