and complete mortality of susceptible lines was noted after 13 days of incubation, when observations were recorded by using a modified 1-9 point scale. Reaction type 1-3 was considered resistant; 3.1-5, tolerant; and 5.1-9, susceptible. In one experiment, 11 chickpea lines were tested against 9 races of *A. rabiei*; ICC 1903 was resistant to 6 races--3072, 3492, 3522, 3744, 4064, and 4080--and tolerant to 2--3844 and 3904. Five lines, ICC 607, 1065, 1905, 1472, and ILC 2520, were resistant to 3 races (Table 1). Six lines, ICC 607, 1065, 1905, 1472, 4000, 5566, showed a tolerant reaction to several of the races tested.

In the second experiment, another 17 lines were screened against 7 races of *A. rabiei*; ICC 1467 was resistant to all. Three lines, GL 83119, GL 84097, and GG 828, were resistant to 2 races. P 2129 was tolerant to 6 races and resistant to 1 race. Three lines, GL 83119, GL 84098, and E 100 Y were tolerant to 5 races (Table 2).

References

Singh, G. 1982. Screening for resistance to ascochyta blight with special reference to efficient screening techniques. Presented at the Workshop on Rabi Pulses, All India Co-ordinated Pulse Improvement Project, 2-5 Oct 1982, New Delhi, India. 13 pp.

Singh, G. 1985. Biological specialization and other related studies on parasitization by *Ascochyta rabiei* on gram. Presented at the All India Co-ordinated Pulse Improvement Workshop, 1-5 Sep 1985, Srinagar, India. 11 pp.

Singh, G. 1986. Further screening of chickpea for resistance to ascochyta blight of chickpea. Presented at the International Food Legume Research Conference, 7-11 Jul 1986, Spokane, Washington, USA.

Singh, G., Singh, K., and **Kapoor, S.** 1982. Screening for sources of resistance to ascochyta blight of chickpea. International Chickpea Newsletter 6:15-17.

Singh, G., Verma, M.M., Gill, A.S., Sandhu, T.S., Brar, H.S., Sra, S.S., and **Kapoor, S.** 1984. Screening of gram varieties against ascochyta blight. Crop Improvement 11(2):153-154.

Verma, M.M., and **Singh, G.** 1987. Genetic control of chickpea blight (*Ascochyta rabiei*), a serious bottleneck in chickpea production. Pages 301-302 in Food Legume Improvement for Asian Farming Systems: proceedings of an International Workshop, 1-5 Sep 1986, Khon Kaen, Thailand (Wallis, E.S., and Byth, D.E., eds.). ACIAR Proceedings no.18. Canberra, Australia: Australian Centre for International Agricultural Research.

Bacterial Wilt of Chickpea Caused by *Xanthomonas campestris* (Pam.) Dowson

J. Raghava Reddy, K.M. Ahmed, and **B.K. Varma** (Plant Quarantine, ICRISAT)

Chickpea seeds received for export by the Plant Quarantine Unit of ICRISAT Center showed bacterial infection on the surface in blotters during a routine microscopic examination. Bacterial growth on the incubated seeds was yellow and slimy. Infected seeds turned black.

The bacterium was isolated on Nutrient Agar (NA) and SX Agar (SXA) from seed coat, cotyledons, and embryo. Isolations were also made from the roots, stems, leaves, and pods of some field-grown chickpea plants exhibiting wilt symptoms. Bacterial colonies on NA were yellow, convex, mucoid, transparent, and shiny; colonies on SXA were circular, mucoid, and translucent, with a bluish center. Cells were gram negative, short rods with rounded ends, motile by a polar flagellum. Cell size ranged from 0.6-0.9 x 0.96-1.9 μm. The bacteria isolated from the seeds and those from the plants were identical and appear to be one of the pathovars of *Xanthomonas campestris* (Pam.) Dowson.

Pathogenicity was tested on 200 seeds of chickpea cultivar K 850. Seeds were surface-sterilized and soaked for 24 h in 200 mL of the bacterial suspension containing 2 x 10^7 cells mL^{-1}. Afterwards, seeds were dried at room temperature and sown in pots containing sterilized soil. The pots were incubated in mist chambers for 72 h at 100% relative humidity and 30°C. After incubation, pots were returned to greenhouse benches. Healthy plants, grown separately, were given the same treatments, except for the bacterial inoculum, to serve as control. The symptoms on the plants were recorded soon after germination, at three stages of plant growth.

In the first stage, the bacterium killed the seeds, resulting in loss of germination (Fig. 1a, 1b, 1c). The

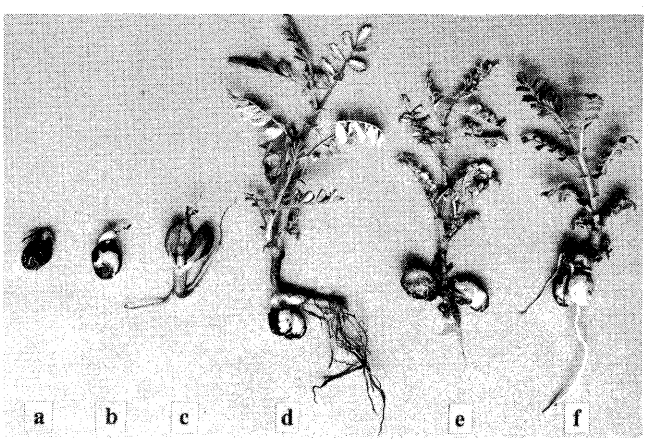

Figure 1. Symptoms of bacterial wilt on chickpea seedlings; a-e: infected seedlings; f: healthy seedling.

bacterium in the second stage caused sudden death of the seedlings within 5-15 days after emergence (Fig. 1d, 1e). Figure 1f shows a healthy seedling.

The third stage of the disease is characterized by the wilt symptoms on the plants. Initial symptoms can be seen on the lower leaves 15-20 days after emergence. On the leaflets, disease symptoms appear as small, irregular, white necrotic spots all over the surface, which can be very easily distinguished from the surface of a healthy leaf. These whitish spots soon coalesce to form larger areas surrounded by a water-soaked margin. Subsequently, the necrotic area covers the entire leaf, resulting in defoliation. The disease advances towards the upper leaves, and ultimately growing tips droop and the plant dies off (Fig. 2a, 2b). The entire process may take about 1 to 6 weeks. The main stem of the infected plant did not show any visible symptoms, except that it became pale green. No vascular discoloration was observed in the root or collar regions.

A few plants that survived the infection produced seeds carrying the bacterium to the next generation. Seeds harvested from such plants did not show any visible symptoms.

Koch's postulates were fulfilled for proof of pathogenicity. The bacterium is systemically pathogenic to chickpea seeds and plants. The disease is toxin-mediated. Wilt symptoms were reproduced when cell-free culture filtrate was used for inoculating healthy plants. Xanthomonads secrete a polysaccharide slime, which is said to be involved in the pathogenicity, acting as a wilt-inducing phytotoxin (Sutton and Williams 1970).

The symptoms and biochemical tests of the bacterium differed from bacterial disease caused by *Xanthomonas cassiae* (Rangaswamy and Prasad 1959), the only bacterial disease reported on chickpea plants in India. *X. cassiae* causes postemergence seedling rot and blight symptoms on petioles and leaflets and was found to cause leaf spot on its primary hosts, *Cassia*

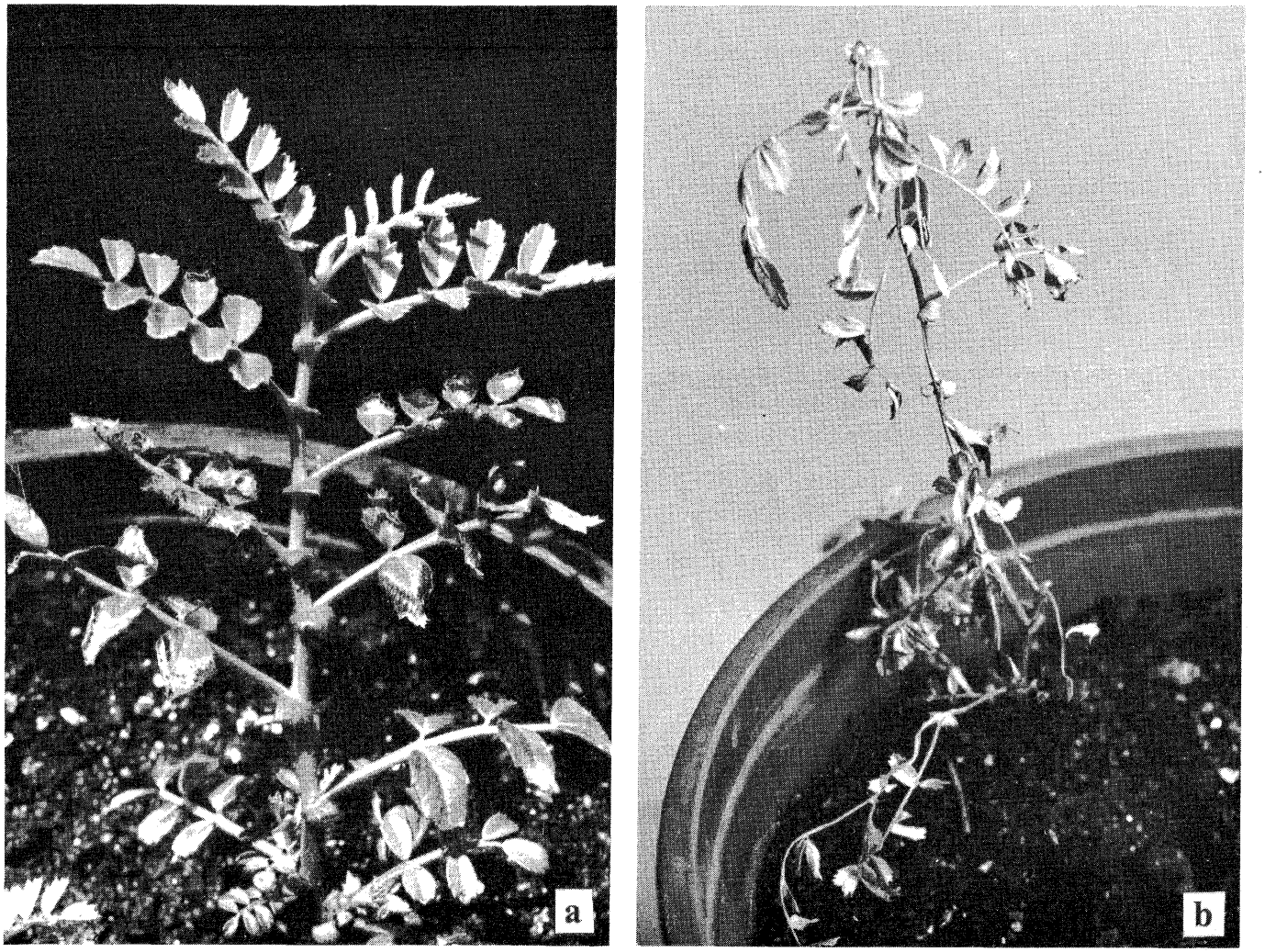

Figure 2. Symptoms of bacterial wilt on full-grown chickpea plants; a: symptoms advance upwards; b: plant dies.

tora L. and *C. occidentalis* L., in nature. The bacterium that we have intercepted did not produce disease symptoms on these primary hosts. Therefore the symptoms we have described appear to be caused by a pathovar of *X. campestris* (Pam.) Dowson.

Laboratory investigations carried out to eradicate seedborne inoculum have shown that dipping infected seeds in hydrogen peroxide (30%), alone and in combination with streptocycline (1000 ppm), gave complete control of the bacterium without affecting germination.

Acknowledgement. The authors are grateful to Dr. Y.L. Nene, Director, Legumes Program, ICRISAT, for his advice and guidance in the investigations.

References

Rangaswamy, G., and **Prasad, N.N.** 1959. A bacterial disease of *Cicer arietinum* L. Indian Phytopathology 12:172-175.

Sutton, J.C., and **Williams, P.H.** 1970. Comparison of extracellular poly-saccharide of *Xanthomonas campestris* from culture and from infected cabbage leaves. Canadian Journal of Botany 48:645-651.

Botrytis Gray Mold Epiphytotic of Chickpea in Nepal

M. V. Reddy[1], **Onkar Singh**[1], **M.P. Bharati**[2], **R.P. Sah**[2], and **S. Joshi**[2] (1. ICRISAT; 2. Grain Legume Improvement Program, Ministry of Agriculture, Kumaltar, Kathmandu, Nepal)

A severe epiphytotic of botrytis gray mold (*Botrytis cinerea* Pers. ex Fr.) occurred on chickpea in the Terai region of central and western Nepal in the 1987/88 season. In late March 1988, we visited seven experiment stations and farmers' fields at three locations to estimate probable yield losses from the disease (Table 1).

The highest levels of disease and the most severe damage, with up to 100% yield loss, were found on experiment station crops, perhaps because these have high plant populations, with dense canopy and high humidity, which foster incidence and severity of the disease, whereas farmers' crops have low plant populations and poor vegetative growth (Fig. 1).

If chickpea yields are to be improved in Nepal, it is essential that farmers should increase the plant population and improve inputs to encourage better growth of their crops. However, this may lead to increased levels of botrytis gray mold attack unless canopy effects on the crop microclimate can be modified. We suggest that compact genotypes with upright growth habit be tried, and the planting pattern be modified; for instance, by increasing interrow spacing and decreasing intrarow spacing to maintain optimal populations yet prevent undue humidity within the crop canopy.

Table 1. Visually estimated yield losses due to botrytis gray mold in chickpea on research farms and in farmers' fields in Nepal, 1987/88.

Location	Site	Yield loss (%)
Central region		
Hardinath	Research farm	100
Bellichapi	Research farm	80
Parwanipur	Research farm	80
Nawalpur	Research farm	50
Midwestern region		
Bhirahwa	Research farm	50
Udarapur	Farmers' field	20
Mankamanapur	Farmers' field	15
Nepalganj	Research farm	25
Western region		
Rampur	Research farm	80
Shivpur	Farmers' field	10

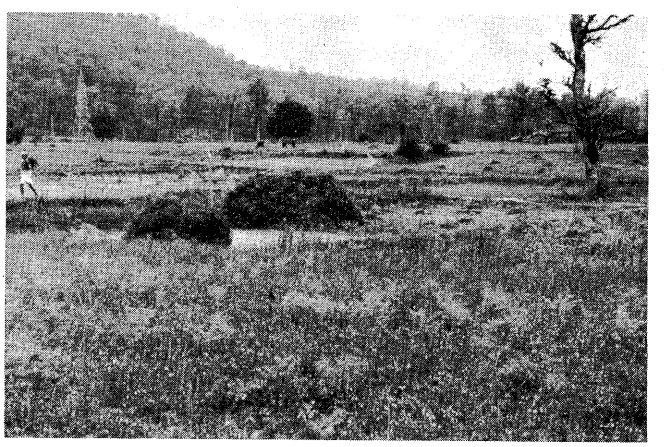

Figure 1. A farmer's chickpea field 20 km west of Nawalpur in central Nepal, showing very poor stand and many weeds. The weed with white flowers is *Leucas aspera*; it was infested by root-knot nematodes (photo from Dr. H.A. van Rheenen).