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RESEARCH PAPERS

Sources of resistance to Fusarium wilt of chickpea in Sudan

Mohamed Elfatih K. Ali[1*], Shinobu Inanaga[2] and Yukihiro Sugimoto[2]

[1]El-Obeid Research Station, Agricultural Research Corporation, P.O. Box 429, El-Obeid, Sudan
[2]Arid Land Research Center, Tottori University, Hamasaka 1390, Tottori 680-0001, Japan

Summary. A total of 330 chickpea genotypes were screened for resistance to Fusarium wilt caused by *Fusarium oxysporum* f. sp. *ciceris* in a wilt-infected plot at Hudeiba Research Station, Ed-Damer, Sudan. The released kabuli cv. Shendi (NEC 2491/ILC 1335) and Jebel Marra-1 (ILC 915), and the widely-grown kabuli cv. Baladi were highly susceptible to Fusarium wilt. Conversely, kabuli cv. ICCV 2 and UC 15, and desi genotypes FLIP 85-20C, FLIP 85-29C and FLIP 85-30C were highly resistant to the disease with less than 10% wilt incidence. 'ICCV 2' matured about 4 to 5 weeks earlier than the other four resistant chickpea genotypes. Two field trials were also conducted to verify the resistance of cv. ICCV 2 and the efficacy of the seed-dressing fungicides Tecto-TM and Quinolate Pro for control of Fusarium wilt. One trial was carried out in the wilt-infected plot and the other in farmers' fields with a history of high disease incidence. In both trials, cv. ICCV 2 showed a good level of resistance to wilt and produced twice the grain yield of cv. Baladi in farmers' fields. Both seed-dressing fungicides significantly increased seedling emergence in the wilt-infected plot, but neither significantly decreased the final incidence of dead plants, or increased the grain yield in the infected plot or the farmers' fields. Results of this study indicate that cv. ICCV 2 is a potential variety for release in the traditional chickpea-producing areas of Sudan where Fusarium wilt and late-season soil moisture stress are major constraints to production.

Key words: *Cicer arietinum, Fusarium oxysporum* f. sp. *ciceris*, Quinolate Pro, Tecto-TM.

Introduction

Fusarium wilt, caused by *Fusarium oxysporum* Schlechtend.:Fr. f. sp. *ciceris* (Padwick) Matuo & K. Sato, is a major constraint to chickpea (*Cicer arietinum* L.) production in many countries (Nene and Reddy, 1987; Haware, 1990; Jalali and Chand, 1992). Yield losses attributable to Fusarium wilt vary from 10 to 15% (Trapero-Casas and Jiménez-Díaz, 1985; Jalali and Chand, 1992) but can result in total loss of the crop under specific conditions (Haware and Nene, 1980; Halila and Strange, 1996). In Sudan, the disease is especially serious in the traditional production areas of the Wad Hamid basin in northern Sudan, where chickpea is grown on stored soil moisture after the flood waters of the Nile River subside (Ali, 1996; Faki *et al.*, 1996). In these areas, farmers do not practice crop rotation and the crop at the post-flowering stage is often subject to moisture stress in years of low flood.

Corresponding author: M.E.K. Ali
Fax: +81 857 296199
E-mail: melfatihali@hotmail.com
*Current address: Arid Land Research Center, Tottori University, Hamasaka 1390, Tottori 680-0001, Japan

The Fusarium wilt pathogen is seed-borne and can persist in the soil in the absence of chickpea for more than 6 years (Haware *et al*., 1992). Therefore, the most practical, effective and economical method of controlling Fusarium wilt of chickpea is through the use of resistant cultivars (Nene and Reddy, 1987; Haware *et al*., 1992; Kaiser *et al*., 1994). Both desi and kabuli chickpea cultivars with resistance to Fusarium wilt have been identified in different countries (Haware and Nene, 1980; Nene and Haware, 1980; Haware *et al*., 1981; Halila *et al*., 1984; Kumar *et al*., 1985; Buddenhagen and Workneh, 1988; Jiménez-Díaz *et al*., 1991; Haware *et al*., 1992).

Treatment of chickpea seed with protectant or systemic fungicides was reported to increase seedling emergence and to reduce pre-emergence damping-off (Verma and Vyas, 1977; Kotasthane and Agrawal, 1978; Shukla *et al*., 1981; Jiménez-Díaz and Trapero-Casas, 1985). In an earlier work, two seed-dressing fungicides, Tecto-TM and Quinolate Pro, were reported to reduce seedbed losses and increase seedling emergence of chickpea in wilt-infected plots (Ali, 1996).

The objectives of this study were: (i) to identify chickpea genotypes resistant to Fusarium wilt in Sudan; and (ii) to verify the resistance of cv. ICCV 2 and the efficacy of two fungicide seed dressings, Tecto-TM and Quinolate Pro, for control of Fusarium wilt in naturally infected farmers' fields.

Materials and methods

Screening for resistance

A uniform Fusarium wilt-infected plot covering an area of 1 ha was set up in a 3-year period at Hudeiba Research Station, Ed-Damer, Sudan by incorporating chopped wilted chickpea plants into the soil and planting with the susceptible chickpea cv. JG-62 (Nene and Haware, 1980; Haware *et al*., 1992). From 1989 to 1995, a total of 330 chickpea genotypes obtained from the International Crops Research Institute for the Semi-Arid Tropics (ICRISAT) and the International Center for Agricultural Research in the Dry Areas (ICARDA) were screened for resistance to Fusarium wilt. The released kabuli cv. Shendi (NEC 2491/ILC 1335) and Jebel Marra-1 (ILC 915), and the widely-grown kabuli cv. Baladi were included as controls. Test entries were arranged in a randomized complete block design with two replications. Throughout the years of screening, chickpeas were sown in the first week of November. Each genotype was sown in a single 2-m-long ridge. Inter- and intra-ridge spacing was 60 and 10 cm respectively, with two seeds per hill. The susceptible chickpea cv. JG-62 was sown after every two entries to check for uniformity of the disease in the plot and increase pathogen inoculum. The plot was irrigated every 10 days. Nitrogen fertilizer was applied as urea at the rate of 43 kg N ha^{-1} before the second irrigation.

Stand counts for the chickpea genotypes were made 3 weeks after sowing. Observations on wilt incidence (total number of dead plants/total number of plants at stand count) were made regularly at 3 weeks after sowing, flowering, and physiological maturity. Disease reactions were classified according to the percentage of dead plants (Haware and Nene, 1982) as resistant (0–20%), moderately susceptible (21–50%), and susceptible (≥51%). The percentage of dead plants at physiological maturity represented the reaction score of each genotype (Nene and Haware, 1980). At each recording date for wilt incidence, a random sample of 50 dead plants was collected from different spots in the wilt-infected plot for identification of the causal organisms. Collar and root tissues were washed in running tap water, cut into 5-mm-long pieces, surface disinfected with 2.5% sodium hypochlorite for 1 min, plated on potato dextrose agar and incubated at 23°C for 7 days. Fungi growing from the tissues were identified and their isolation frequencies were determined.

Verification of resistance and efficacy of fungicide seed dressings

Resistance of cv. ICCV 2 to Fusarium wilt and efficacy of the fungicide seed dressings Tecto-TM and Quinolate Pro were verified in two trials. One trial was conducted in the wilt-infected plot in the 1993 growth season and the other one in farmers' fields with a history of high disease incidence in the traditional chickpea-producing areas of the Wad Hamid basin in northern Sudan in the 1994 season.

Wilt-infected plot trial

Treatments consisted of a factorial combination of two chickpea cultivars, ICCV 2 and Shendi, and two seed-dressing fungicides, Tecto-TM (16.5% thi-

abendazole + 36.5% thiram) and Quinolate Pro (10% oxine copper + 10% carbendazim), applied at 3 g product kg^{-1} seed as a slurry and a dry powder respectively. An untreated control from each cv. was included for comparison. The six treatments were arranged in a randomized complete block design with three replications. Gross plot size for each treatment was 15 m^2 with a net harvested area of 7.2 m^2. Chickpea was sown on 25 November 1993. Inter- and intra-ridge spacing was 60 and 10 cm respectively, with two seeds per hill. The crop was irrigated at 10-day intervals from sowing to maturity. Nitrogen fertilizer was applied as urea at the rate of 43 kg N ha^{-1} before the second irrigation.

Stand counts (plants m^{-2}) were determined 3 weeks after sowing from an area of 7.2 m^2 in each plot. Wilt incidence as a percentage of dead plants (total number of dead plants/total number of plants at stand count $\times$100) was recorded regularly in the same area at three weeks after sowing, flowering, and physiological maturity. Grain yield was also determined from the same area in each plot. Data were subjected to analysis of variance using the MSTAT-C Statistical Package developed by Michigan State University. Percentages of dead plants were transformed to arcsine before statistical analysis. Data presented in Table 2 are the means of three replications.

On-farm trial

This trial was conducted in four farmers' fields with a history of high disease incidence at Wad Hamid basin, the largest chickpea-producing area in Sudan, where Fusarium wilt is a major constraint to production. Treatments consisted of a factorial combination of two chickpea cv. (ICCV 2 and Baladi) and a slurry seed treatment with the seed-dressing fungicide Tecto-TM at 3 g product kg^{-1} seed including controls. The four treatments were arranged in a randomized complete block design replicated in four previously flooded farmers' fields. Each of the four fields served as a separate replication. These fields were selected on the basis of their previous disease history. Plot size for each treatment was 300 m^2 in each field. Chickpea was sown in mid-November 1994 after the flood water of the river had subsided, and the crop was grown on stored soil moisture till harvest. The seeding rate for both cv. was 60 kg ha^{-1}. Crops were established according to the farmers' practice in which the seeds are dibbled along furrows made by an ox-driven plow that automatically covers the seeds of the preceding furrow.

Stand counts (plants m^{-2}) were determined 3 weeks after sowing from three 4 m^2 spots randomly marked in each plot. Wilt incidence as the percentage of dead plants (total number of dead plants/total number of plants at stand count 100) was recorded regularly in the same spots at 3 weeks after sowing, flowering, and physiological maturity. Grain yield was determined from a net harvested area of 180 m^2 in each plot. Data were subjected to analysis of variance using the MSTAT-C Statistical Package developed by Michigan State University. Percentages of dead plants were transformed to arcsine before statistical analysis. Data presented in Table 3 are the means of four replications.

Results

Screening for resistance

During all the years of screening, *F. oxysporum* f. sp. *ciceris* was the primary fungus isolated from the collar and root tissues of wilt-affected plants, with a mean frequency of 98.7% (data not shown). In a very few cases (1.3%), *Rhizoctonia bataticola* or *R. solani* were isolated together with *F. oxysporum* f. sp. *ciceris*. All plants of the susceptible cv. JG-62 were killed throughout the plot in about 3 to 4 weeks from sowing. Of the 330 chickpea genotypes screened for resistance to Fusarium wilt in the wilt-infected plot from 1989 to 1995, 279 genotypes, including the two released kabuli cv. Shendi and Jebel Marra-1, and the widely-grown kabuli cv. Baladi, were susceptible. 'Shendi' consistently showed 100% mortality, while 'Jebel Marra-1' and 'Baladi' had more than 85% wilt incidence (Table 1). For more than 4 years of screening, the two kabuli genotypes ICCV 2 and UC 15 and the three desi genotypes FLIP 85-20 C, FLIP 85-29 C and FLIP 85-30 C consistently showed high wilt resistance, with less than 10% incidence (Table 1). 'UC 15' and 'ICCV 2' had larger seed size than the desi genotypes. However, 'ICCV 2', matured about 4 to 5 weeks earlier than 'UC 15' and the resistant desi genotypes (Table 1). In the 1995 season, 46 other chickpea genotypes with less than 20% wilt incidence were identified. Resistance of these genotypes, however, needs further confirmation in coming seasons.

Verification of resistance and efficacy of fungicide seed dressings

Wilt-infected plot trial

'ICCV 2' and 'Shendi' differed significantly (P<0.001) in wilt incidence (Table 2). 'ICCV 2' showed a good level of resistance and produced an average grain yield of 1,343 kg ha^{-1}, while plants of 'Shendi' were all killed by the disease before the flowering stage. Seed-dressing with Quinolate Pro and Tecto-TM significantly (P<0.01) increased seedling emergence compared to the untreated control; however, they did not significantly decrease the final incidence of dead plants or increase grain yield (Table 2).

Table 1. Wilt incidence, reaction scores and some agronomic characteristics of selected chickpea genotypes in a wilt-infected plot at Hudeiba Research Station, Ed-Damer, Sudan, screened from 1989 to 1995.

Genotype	Germplasm	Mean wilt incidence (%)[a]	Reaction score[b]	Days to 50% flowering	Days to maturity	HSW[c] (g)
ICCV 2	Kabuli	9.4	R	34	80	26
UC 15	Kabuli	7.2	R	65	108	43
FLIP 85-20 C	Desi	5.9	R	73	112	14
FLIP 85-29 C	Desi	7.7	R	73	115	15
FLIP 85-30 C	Desi	0	R	75	115	15
JG-62	Desi	100	S	-	-	-
Shendi	Kabuli	100	S	-	-	-
Jebel Marra-1	Kabuli	87.6	S	-	-	-
Baladi	Kabuli	91.2	S	-	-	-

[a] Percentage of dead plants (total number of dead plants/ total number of plants at stand count 3 wk after sowing ×100) at physiological maturity. Mean of two replications throughout the years of screening.
[b] R, resistant (0–20% mortality) and S, susceptible (≥51% mortality) according to Haware and Nene (1982).
[c] HSW, 100-seed weight.

Table 2. Effect of chickpea cultivar and fungicide seed treatment on crop stand, wilt incidence and grain yield in a wilt-infected plot at Hudeiba Research Station, Ed-Damer, Sudan in 1993.

Treatment	Plants m^{-2} at 3 w.a.s.[a]	Final incidence of dead plants (%)[b]	Grain yield (kg ha^{-1})
Cultivar			
Shendi	29	99.9	0
ICCV 2	29	9.5	1343
Mean	29	54.7	672
SE ±	ns	0.18***	-
Seed treatment			
Tecto–TM	30	54.6	1382[c]
Quinolate Pro	31	54.6	1324
Untreated control	28	54.9	1321
Mean	30	54.7	1342
SE ±	0.52**	ns	ns

[a] Weeks after sowing.
[b] Percentage of dead plants (total number of dead plants/ total number of plants at stand count 3 weeks after sowing 3100) at physiological maturity. Mean of four replications. Percentage data transformed to arcsine.
[c] Mean yield of cv. ICCV 2.
ns, not significant (P>0.05); **, significant at P<0.01; ***, significant at P<0.001.

Table 3. Effect of chickpea cultivar and fungicide seed treatment on crop stand, wilt incidence and grain yield in farmers' fields at Wad Hamid basin, northern Sudan in 1994.

Treatment	Plants m^{-2} at 3 w.a.s.[a]	Final incidence of dead plants (%)[b]	Grain yield (kg ha^{-1})
Cultivar			
Baladi	20	47.6 (54.53)	560
ICCV 2	16	0.6 (0.01)	1092
Mean	18	24.1	826
SE ±	ns	4.7 ***	59.1***
Seed treatment			
Tecto–TM	21	23.2 (15.52)	891
Untreated control	16	25.1 (17.99)	762
Mean	18	24.1	826
SE ±	ns	ns	ns

[a] Weeks after sowing.
[b] Percentage of dead plants (total number of dead plants/ total number of plants at stand count 3 weeks after sowing ×100) at physiological maturity. Mean of four replications. Percentage data were transformed to arcsine. Numbers in parentheses are actual percentages.
ns, not significant ($P>0.05$); *** significant at $P<0.001$.

On-farm trial

Cv. ICCV 2 had significantly ($P<0.001$) lower wilt incidence and higher grain yield than cv. Baladi (Table 3). 'ICCV 2' showed less than 1% wilt incidence, while 'Baladi' had more than 50% incidence. The grain yield of 'ICCV 2' was almost twice that of 'Baladi'. Moreover, 'ICCV 2' matured about 1 month earlier than 'Baladi' (data not shown). Seed dressing with Tecto-TM improved seedling emergence, but differences between fungicide treated and untreated plots were not statistically significant ($P>0.05$). The effect of seed treatment on the final incidence of dead plants and grain yield was also not significant ($P>0.05$).

Discussion

The 100% mortality of plants in all the ridges sown with the susceptible cv. JG-62 indicates that the Fusarium wilt pathogen was uniformly spread throughout the plot and that the screening method was reliable (Haware *et al*., 1992). Moreover, the high frequency of isolation (98.7%) of *F. oxysporum* f. sp. *ciceris* from the collar and root tissues of dead plants indicated that the wilt pathogen was the most predominant fungus in the plot and the primary cause of plant death.

Results of the screening clearly showed that the released kabuli cv. Shendi and Jebel Marra-1, and the widely-grown kabuli cv. Baladi were highly susceptible to Fusarium wilt. On the other hand, the kabuli cv. ICCV 2 and UC 15 and three desi genotypes FLIP 85-20C, FLIP 85-29C, and FLIP 85-30C consistently showed high resistance to Fusarium wilt. Resistance of these genotypes to wilt had already been reported from other countries (Kumar *et al*., 1985; Buddenhagen and Workneh, 1988; Jiménez-Díaz *et al*., 1991). The stable resistance exhibited by these genotypes in several countries, including Sudan, where different races of the wilt pathogen exist (Haware and Nene, 1982; Cabrera de la Colina *et al*., 1985; Phillips, 1988; Ali, 1996; Suliman, 1999/2000; Navas-Cortés *et al*., 2000) suggest that these genotypes possess a broad-based resistance (Nene *et al*., 1989).

Results of the verification trials conducted in the wilt-infected plot and the farmers' fields in the Wad Hamid basin confirmed the resistance of cv. ICCV 2 and susceptibility of cv. Shendi and Baladi to Fusarium wilt. Although 'ICCV 2' was resistant and 'Baladi' susceptible in both the wilt-infected plot and the farmers' fields, the percentage of dead plants in both cultivars was higher in the wilt-infected plot than in the farmers' fields (Table 1 and 3). The high level of inoculum and the uniformity of the disease in the wilt-infected plot may account

for the higher incidence of dead plants in the wilt-infected plot than in the farmers' fields.

In both the wilt-infected plot and the farmers' fields, cv. ICCV 2 produced satisfactory yields (Tables 2 and 3). The higher yield of 'ICCV 2' in the wilt-infected plot than in the farmers' fields may have been due to differences in soil fertility, moisture conditions, methods of planting or climatic conditions.

Seed dressing with Quinolate Pro and Tecto-TM significantly increased emergence of chickpea seedlings in the wilt-infected plot (Table 2). Similar results were previously reported for other fungicide seed dressings (Verma and Vyas, 1977; Kotasthane and Agrawal, 1978; Shukla *et al*., 1981; Jiménez-Díaz and Trapero-Casas, 1985). In the farmers' fields, however, the improvement with Tecto-TM was not significant (Table 3). This lack of significance in these naturally infected fields could be due to environmental conditions (Jiménez-Díaz and Trapero-Casas, 1985) and/or the presence of other soil-borne pathogens. In agreement with other researchers (Kotasthane and Agrawal, 1978; Jiménez-Díaz and Trapero-Casas, 1985), we found that neither Quinolate Pro nor Tecto-TM controlled Fusarium wilt in the wilt-infected plot or the farmers' fields.

Because of its resistance to Fusarium wilt, its earlier maturity and its higher grain yield than the widely-grown cv. Baladi, cv. ICCV 2 is a potential variety for release in the traditional chickpea-producing areas of Sudan where Fusarium wilt and late-season soil moisture stress are major constraints to production.

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