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STUDIES ON THE WHEAT SEED GALL NEMATODE

by
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Summary. The reaction of 16 genotypes of wheat to infection by the seed gall nematode, *Anguina tritici*, and the development of "tundu" or spike blight disease was evaluated by artificial inoculation under field conditions for two successive seasons at Baghdad. The tested genotypes showed differential reactions to ear-cockle and tundu diseases. Cv. Saberbeg was found to be highly resistant. Surface sterilization of nematode inoculum reduced the incidence of ear-cockle disease, when juveniles but not when galls were used as inocula.

Anguina tritici (Steinb.) is an important parasite of wheat in some regions of the world including Iraq. Ear-cookle disease has been reported from most provinces of Iraq and all wheat cultivars that have been planted were susceptible, exept for cv. Saberbeg which is highly resistant (Al-Beldawi *et al.*, 1985).

Tundu or spike blight disease of wheat is caused when *A. tritici* and the bacterium *Clavibacter tritici* (Carlson and Vidavar, 1982) coinfection occurs (Cheo, 1942; Gupta and Swarup, 1972; Bird, 1981; Pathak and Swarup, 1984; Fattah, 1988). This disease complex was recently reported in Iraq in cv. Mexipak artificially inoculated with *A. tritici* under field conditions (Fattah, 1986).

A study was undertaken to evaluate the reaction of some wheat cultivars to both ear-cockle (wheat seed gall) and tundu (also known as spike blight or yellow ear rot) infections, and the effect of surface sterilization of nematode inocula on the development of ear-cockle disease.

Materials and methods

Sixteen genotypes of wheat (See Fig. 1) were evaluated for ear-cockle and tundu diseases by artificial inoculation under field conditions at Baghdad. The experiments were conducted for two successive growing seasons, 1987 and 1988.

Evaluation of wheat genotypes to ear-cockle infection

Twenty five seeds of each of the 16 genotypes of wheat were sown for two successive seasons on January 12, 1987 and December 14, 1987. Seeds were planted in plots (100 x 50 cm each) inside a 1m long and 4cm deep ditch made in the middle of each unit. Seeded ditches were covered to a depth of about 1 cm with 500 ml of sandy soil before inoculation with the nematodes. Aqueous suspension (50 ml) of second stage juveniles of *A. tritici*, extracted from 0.1 g seed galls, was spread evenly over each seed line. Inoculated lines were covered with soil to a height of 3 cm and watered with 250 ml of tap water. This method of inoculation produced the highest ear-cockle disease incidence (Fattah, 1988). Each treatment was replicated 3 times and randomized complete block design was followed. At spike maturation percentages of infected plants were calculated. A plant was considered infected when nematode seed galls were found in any of its spikes.

Evaluation of wheat genotypes to tundu disease

Twenty five-2 day old germinated seeds of each of the 16 wheat genotypes were sown on January 13, 1987 and December 14, 1987 for 1987 and 1988 cultivation seasons respectively. The sowing and inoculation procedures were as mentioned before except 0.3 g of intact seed galls per seed line was used as the nematode inoculum. Such inoculation method produced the highest percentage of tundu disease (Fattah, 1988).

Effect of surface sterilization of nematode inoculum on ear-cockle disease

In this experiment 5 seeds or 5-2 day old germinated seeds of cv. Mexipak were sown on December 15, 1986 in 10 cm diam plastic pots.

Inoculum densities of 5, 10 and 15 galls were either used intact or surface sterilized in 0.1% $HgCl_2$ solution for 3 min and washed three times with sterile water.

Juveniles extracted from 5, 10 and 15 seed galls were

either used intact or surface sterilized with $HgCl_2$ as was mentioned above and added as 10 ml water suspensions. Each treatment was replicated three times and the pots were maintained in the field.

Results

Evaluation of wheat genotypes to infection by A. tritici

Differences between genotypes to infection by *A. tritici* and the development of the nematode — bacterial complex (tundu) are shown in Fig. 1. The local cv. Saberbeg (No. 16 Fig. 1) was the most resistant and showed no evidence of infection by *A. tritici* or the bacterium *C. tritici* in both the 1987 and 1988 tests. A low percentage of nematode infection occurred on *T. aestivum* 179, Sab. x (Mex. x Abug. 3) and Sab x Araz, (Nos. 4, 8 and 12 respectively), for the two test seasons. Other genotypes such as *T. durum* 8, *T. aestivum* 149, Sab x (Mex. x Abug. 3) and Horani, (Nos. 1, 3, 9 and 15 respectively), were more resistant in 1987 than in 1988 seasons. Kokort C71, Araz, Gerardo and Mexipak were highly susceptible in both 1987 and 1988 seasons.

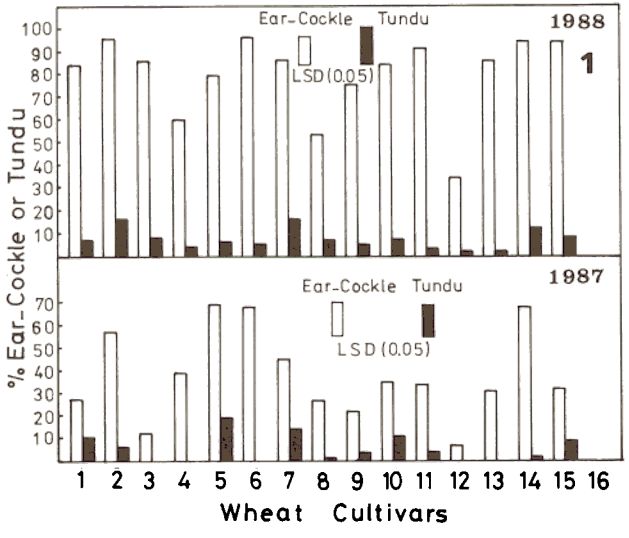
The incidence of ear-cockle disease was generally higher in 1988 than in 1987 for all the test genotypes. Some genotypes showed no tundu infection in 1987 but all developed various degrees of tundu disease in the 1988 test. Tundu incidence never exceeded that of ear-cockle for any particular genotype tested in 1987 or 1988 season.

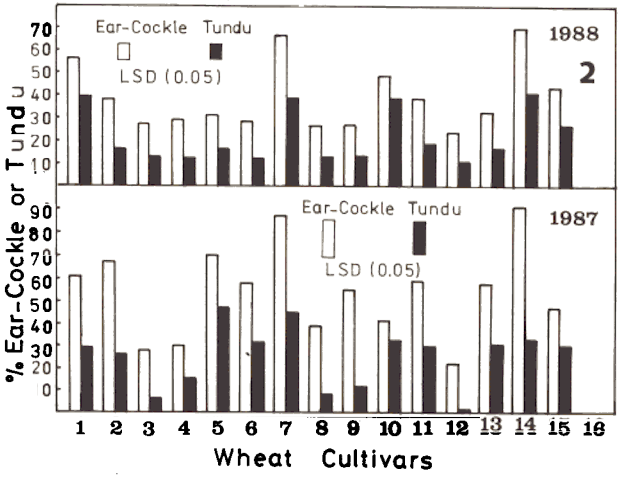
Evaluation of wheat genotypes to tundu disease

Significant (P = 0.05) differences between genotypes in percentages of infected plants by both pathogens are shown in Fig. 2. The genotype Saberbeg (16) remained symptom-free of both diseases in 1987 and 1988 trials. Low incidence of tundu disease appeared on the genotypes *T. aestivum* 149 (3), *T. aestivum* 179 (4), Sab. x (Mex x Abug. 3) (8), Sab. x (Mex. x Abug. 4) (9) and Saberbeg (12) in the two trials. High incidence of tundu disease was observed on *T. durum* 8 (1), Sinaljamal (7), *T. durum* 138 (10) and Mexipak (14) in 1987 and 1988 trials. The genotypes Sinaljamal (7) and Mexipak (14) showed high incidence of ear-cockle disease in the two test seasons. The highest tundu incidence (about 50%) was recorded on cv. Araz (5) and cv. Sinaljamal (7) in 1987 trial and (about 40%) on *T. durum* 8 (1), Sinaljamal (7), *T. durum* 138 (10) and Mexipak in 1988 trial. In general most of the test genotypes showed higher incidence of tundu and ear-cockle in 1987 than in 1988. Similarly as in the previous test, in this test, tundu incidence for any particular genotype has also never exceeded that of ear-cockle (Fig. 2).

Effect of surface sterilization of nematode inoculum on the incidence of ear-cockle disease

Surface sterilization of nematode inocula reduced the incidence of ear-cockle disease when juveniles but not when galls were used to inoculate seeds or germinated seeds of cv. Mexipak (Figs. 3, 4). Significantly (P = 0.05) higher disease incidence occurred when higher inoculum density of unsterilized juveniles (extracted from 10 or 15 seed galls) than when a lower juvenile concentration (extracted from 5 seed galls) regardless of whether seeds or germinated seeds were used. The same trend was apparent for the sterilized inocula; however, the results were not significantly different (Fig. 3). Whereas, when seed galls were used as inocula, inoculum concentration, plant type (seeds or germinated seeds) and surface sterilization





Figures 1 and 2: Incidence of ear-cockle and tundu diseases on wheat genotypes caused by inoculation with juveniles of *Anguina tritici* extracted from 0.1 g seed galls (Fig. 1), or by inoculation with 0.3 g seed galls (Fig. 2). The genotypes are: *Triticum durum* 8 (1), Kokort C71 (2), *T. aestivum* 149 (3), *T. aestivum* 179 (4), Araz (5), Gerardo (6), Sinaljamal (7), Saberbeg x (Mexipak x Abughraib 3) (8), Saberbeg x (Mexipak x Abughraib 4) (9), *T. durum* 138 (10), Abughraib 3 (11), Saberbeg x Araz (12), Nouri 70 (13), Mexipak (14), Horani (15) and Saberbeg (16).

showed no significant effects on the development of ear-cockle disease (Fig. 4).

Discussion

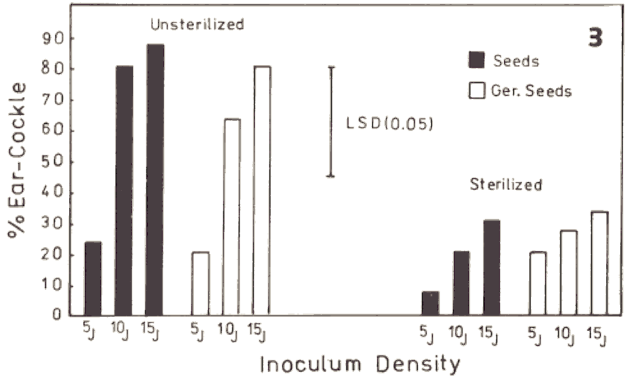
Previous studies (Al-Beldawi *et al.*, 1985) have shown that wheat genotypes differ in their susceptibility to infection by *A. tritici*. Similarly, our results showed differences between 16 test genotypes to infection by the wheat seed nematode. This differential reaction was mainly related to the genetic variation of the test genotypes. The high resistance of cv. Saberbeg (Al- Beldawi *et al.*, 1985) was confirmed by our results and indicated that it could be used in breeding programmes or in rotations to reduce damage by *A. tritici*. The effect of the high resistance of cv. Saberbeg is evident in the high level of resistance shown by the genotype Saberbeg x Araz and to tundu (Figs 1, 2).

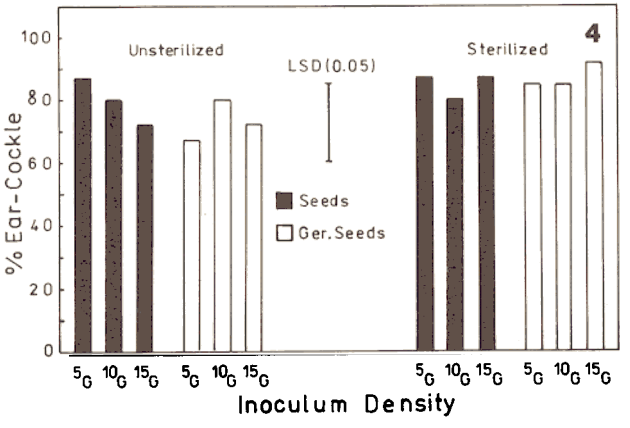
The low degree of tundu infection (Fig. 2) compared with that of ear-cockle, comfirms previous work and may also indicate the importance of nematode infection for the development of tundu disease (Pathak and Swarup, 1984). Furthermore, the occurrence of *C. tritici* with *A. tritici* may reduce the incidence of ear-cockle and points to the importance of using nematode inoculum which is free from the bacterium *C. tritici* for *A. tritici* resistance screening studies. The contamination of the nematodes with *C. tritici* was reported to cause *A. tritici* to aggregate and to reduce mobility (Bird, 1981; Fattah, 1986) and hence, limits the infection ability of the nematodes.

The high susceptibility of cv. Araz and cv. Sinaljamal to tundu is mainly due to their high susceptibility to infection by *A. tritici* (Fig. 1, 2). Tundu incidence was higher when 0.3 g seed galls (Fig. 2) were used intact than when 0.1 g seed galls were used as juveniles. This result supports previous studies (Cheo, 1942; Gupta and Swarup, 1974; Fattah, 1986, 1988) and indicated the importance of the co-occurrence of *C. tritici* and *A. tritici* within seed galls for the successful development of tundu disease.

The reduced disease incidence in the 1987 trial (Fig. 1) compared with that of 1988 trial could possibly relate to the relatively delayed planting time and/or the extreme weather conditions of 1987 season. In this test, seeds were inoculated with juveniles and the low temperatures in January may have delayed seed germination compared to that during December (1988 trial); some inoculum may have been lost while waiting for host availability. Conversely when galls were used to inoculate germinated seeds the cold weather in January increased the time of host availability for nematode infection and hence, increased the disease percentage which was observed in that test (Fig. 2).

Surface sterilization of second stage *A. tritici* juveniles reduced their infectivity (Fig 3), possibly due to the toxic effect of $HgCl_2$ and this may limit the value of surface sterilization in screening for *A. tritici* resistance. However, surface sterilization for 30 min in 0.1% $HgCl_2$ did not adversely affect the viability and infectivity of the second stage juveniles of *A. tritici* (Gupta and Swarup, 1972; Pathak and Swarup, 1984). Our results also indicated that surface sterilization of the seed galls caused no reduction in nematode infectivity (Fig. 4). This is possibly because the daur second stage juveniles of *A. tritici*, inside the seed galls are less sensitive to $HgCl_2$ than the active juveniles, extracted from the galls. It may also be because the gall wall reduces direct contact of the nematode with $HgCl_2$.





Figures 3 and 4: Effect of surface sterilization (3 min in 0.1% $HgCl_2$) of *Anguina tritici* on the incidence of ear-cockle disease on cv. Mexipak 5J, 10J and 15J were juveniles extracted from 5, 10 and 15 seed galls respectively (Fig. 3). 5G, 10G and 15G were 5, 10 and 15 seed galls respectively (Fig. 4).

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