Fusarium wilt and other important diseases of chickpea in the Mediterranean area

Haware M.P.

in

Saxena M.C. (ed.), Cubero J.I. (ed.), Wery J. (ed.).
Present status and future prospects of chickpea crop production and improvement in the Mediterranean countries

Zaragoza : CIHEAM
Options Méditerranéennes : Série A. Séminaires Méditerranéens; n. 9

1990
pages 61-64

Fusarium wilt and other important diseases of chickpea in the Mediterranean area

M.P. HAWARE
FOOD LEGUME IMPROVEMENT PROGRAM
INTERNATIONAL CENTER FOR AGRICULTURAL RESEARCH IN THE DRY AREAS (ICARDA)
P.O. BOX 5466, ALEPPO, SYRIA

SUMMARY - Several diseases of chickpea are reported from different parts of the world. Only a few of them are of economically important. Fusarium wilt, ascochyta blight and stunt (bean leaf roll virus) are internationally important diseases of chickpea. In the mediterranean countries, ascochyta blight, fusarium wilt, stem rot, stunt and root rot are commonly observed in the farmer's fields. However precise information is not available on the losses caused by these diseases.

RESUME - "Le fusarium et d'autres maladies importantes du pois chiche dans le bassin méditerranéen". Plusieurs maladies du pois chiche sont connues dans le monde, mais peu d'entre elles ont une importance économique. La fusariose, l'anthracnose et le virus de l'enroulement (bean leaf roll virus) sont les principales maladies du pois chiche à l'échelle mondiale. Dans le bassin méditerranéen l'anthracnose, la fusariose, la pourriture des tiges, le virus de l'enroulement et la pourriture des racines peuvent être observés dans les champs des agriculteurs. Cependant nous n'avons aucune information précise sur les pertes de rendement causées par ces maladies.

Fusarium wilt

Fusarium wilt is a serious disease of chickpea in India, Iran, Pakistan, Nepal, Burma, Spain, Tunisia and Mexico. It has been observed in Morocco, Algeria, and Syria. An estimated annual loss of 12 million rupees was reported from Pakistan (Sattar *et al.*, 1953). Attempts were made to estimate loss in yield on a per plant basis. Earlier wilting caused more loss than late wilting. Seeds harvested from wilted plants were lighter and duller than those from healthy plants (Haware and Nene, 1980).

In a highly susceptible cultivar wilting can be observed within 25 days after sowing in the field. Seedlings that die due to the wilt disease can be confused with those that die to root rots if not examined carefully. The affected seedlings show drooping of the leaves and paler color. They may collapse and lie flat on the ground. The roots do not show any external rotting but look apparently healthy. Such roots, when split vertically from the collar region downward show a brown discoloration of the internal tissues.

Adult plant may show typical wilting which may occur in the field up to podding stage. The initial symptom is drooping of petioles and rachis along with leaflets. There is a slow fading of the green color and the plant looks dull green. Gradually all leaves turn yellow and straw colored. Roots of the wilted plant show no external rotting drying or discoloration, and when split vertically, show internal discoloration of the pith and xylem.

Pathogen: *Fusarium oxysporum* Schlecht. emend. Snyd. and Hans. f. sp. *ciceri* (Padwick) Snyd. and Hans.

Growth of the fungus on potato-sucrose-agar at 25 °C is delicate, white, cottony becoming felted and wrinkled in old cultures. Hyphae are septate and profusely branched.

Microconidia are borne on simple short conidiophores, arising laterally on the hyphae. Conidia are generally sparse on solid medium. They are formed abundantly on potato-sucrose broth. Microconidia are oval to cylindrical, straight to curved and 2.5-3.5x5.11μm. Macroconidia

are lesser in number than microconidia, borne on branched conidiophores, thin-walled, 3 to 5 septate, fusoid and pointed at both ends, and measure 3.5-4.5x25-65µm. Chlamydospores are formed in old cultures (20 d), smooth or rough walled, terminal or intercalary, and may form singly, in pairs or in chain.

The fungus is seed-borne (Haware *et al.*, 1987) and soil-borne. The fungus could survive on crop residues in soil for more then 6 years (Haware *et al.*, 1986). Lentil, pigeonpea and pea were identified as symptomless carriers of the pathogen (Haware and Nene, 1982). The primary infection is through chlamydospores or conidia. Soil temperature and moisture may affect the appearance of symptoms.

Haware and Nene (1982 a) have identified four physiologic races (1, 2, 3 and 4) from India on the basis of the reactions on 10 differentials. Using the same differentials 3 races (0, 1 and 5) were reported from Spain (Cabrera de la Colina *et al.*, 1987).

Since the fungus is soil-borne and can survive in the soil for long period, it is not possible to control disease through crop rotation. The seed-borne inoculum can be eradicated by seed dressing with Benlate T (benomyl 30% + thiram 30%) at 0.25% rate (Haware *et al.*, 1978).

At ICRISAT center, effective field-screening and laboratory procedure have been developed and wilt-resistance sources-identified (Nene and Haware, 1980). Some of them have additional resistance to dry root rot, sclerotinia stem rot and ascochyta blight. Fusarium wilt resistance has been incorporated into high yielding desi and kabuli backgrounds, using donors, identified as highly resistant at ICRISAT and screening breeding materials in wilt sick plot.

Recently chickpea wilt-screening facilities have been developed at Beja in Tunisia and Cordoba in Spain. In cooperative research between INRAT and ICARDA, wilt-resistant chickpea lines are developed in Tunisia. These lines should be used for testing in Mediterranean countries.

Earlier reports suggested that resistance to fusarium wilt in chickpea was conferred by a single recessive gene (Pathak *et al.*, 1975; Kumar and Haware, 1982). Recent studies have shown that resistance to race 1, appears to be controlled by at least 3 independent loci. Complete resistance is obtainable from crosses involving susceptible parents (late-wilting) (Harjit Singh *et al.*, 1987).

Verticillium wilt

Wilt caused by *Verticillium albo-atrum* in chickpea was first reported from California in USA (Erwin, 1958). The fungus is also associated with *Fusarium* in causing the widespread wilt in Tunisia (H. Halila, personal communication).

The foliage of the affected plant turns yellow before wilting. The xylem tissue shows light brown discoloration which is lighter than that caused by *F. oxysporum* f. sp. *ciceri* (Ewin, 1958). There is not much difference in symptoms produced by these 2 pathogens. Field screening at Beja in Tunisia has shown the progress in the development of combined resistant lines to both the pathogens.

Sclerotinia stem rot

Sclerotinia stem rot is reported from Australia, Chile, India and Iran. During the surveys in 1988, it was observed in Algeria, Morocco, Tunisia and Syria. In Morocco and Algeria, the disease was found widespread in chickpea.

The symptoms on adult plants are closely similar to wilt. When examined critically, symptoms are clearly seen on the stems which are attacked at any point from soil level to the top. A web of white mycelial strands appears on the stem and base of the branches. In absence of mycelium, greyish lesions can be seen on the stems. Occasionally whitish irregular sclerotial knots, or black sclerotia along with mycelial strands can be observed on stem. The above-ground symtoms are drooping of petioles and leaflets, which ultimately dry and turn straw-colored prematurely.

The disease appears mostly on adult plants when they form a thick canopy and soil remains wet for a long period. The disease is favoured by moderately cool and wet weather, excessive vegetative growth and heavy dew.

Pathogen: *Sclerotinia sclerotiorum* (Lib.) de Bary.

The fungus causes a stem rot of many annual and perennial plants. Sclerotia are formed in abundant white mycelium on the surface of the stem. They are very irregular in shape and size, black on surface and white within. The sclerotia survive in the soil. In the following season the sclerotia germinate by producing apothecia and ascospores. Ascospores infect the stem (Singh and Singh, 1983). Aeration, availability of light, a suitable temperature and light-textured soils are considered favourable for germination of sclerotia.

Since the potential of the disease to devastate the crop was not realised, no efforts were made to identify the resistance to stem rot in chickpea. Limited screening for resistance is being carried out by ICRISAT, and a few chickpea lines with moderate resistance have been identified.

Dry root rot

Dry root rot of chickpea has been reported from India, Iran, Australia, Ethiopia, Pakistan, Spain and USA. The importance of this disease in Mediterranean region is not known.

Drying of the plants appear suddenly in the field at flowering and podding. There is no drooping which is commonly seen in the plants affected by wilt. The leaves and stems of affected plants are straw-colored. The roots are usually dry, unless the soil is wet. The tap root is quite brittle, show shredding of the bark and can be broken easily. Minute dark black sclerotial bodies can be seen with the aid of a 10 x hand lens on the surface of the root, as well as in the pith.

The disease appears suddenly when ambient temperatures are between 25 and 30 °C.

Pathogen: *Rhizoctonia bataticola* (Taub) Butler=*Macrophomina phaseolina* (Maubl.) Ashby.

The fungus lacks fruiting bodies and spores. The mycelium is light-brown, thick in which black sclerotia are formed. Sclerotia are variable in form, small and loosely connected by mycelial threads.

The fungus is soil-borne and may survive in the soil in the form of sclerotia for long time. The fungus has a wide host range and can infect the roots of several crops. Therefore crop rotation is not effective to control the disease.

Since the disease is soil-borne the development of sick plot is essential for resistance screening. However the presence of other soil-borne pathogens in sick plot cannot be avoided. To confirm resistance specifically to *R. bataticola* it would be necessary to follow laboratory technique.

The blotter paper technique (Nene *et al.*, 1981) is routenly used at ICRISAT to confirm resistance in the lines identified promising in the field. In this technique 5 to 7 day-old seedlings, raised in sterilized sand are root inoculated with inoculum and seedlings incubated at 32-35 °C for 8 days. At the end of the incubation, seedlings are examined for the extent of root damage and scored on 1-9 point scale.

At ICRISAT center several lines resistant to dry root rot were identified. Some of these lines have the resistance to both, wilt and dry root rot.

The inheritance of resistance to dry root rot of chickpea seems to be monogenic with resistance dominant over susceptibility. (Ananda Rao and Haware, 1987).

Stunt

Pea leaf roll virus producing wilting in chickpea was first reported from Iran (Kaiser and Danesh, 1971). Nene and Reddy (1976) described it from India, the cause of which was established as the bean leaf roll virus. The disease is prevalent in North Africa and West Asia. Author has observed it to be particularly serious in Lebanon, Morocco and Tunisia.

Affected plant is stunted due to shortened internodes. The leaflets are smaller.Virus infected plants can be easily spotted in the field by their yellow, orange or brown discolored foliage. The tips and margins of leaflets became chlorotic before turning brown. The foliage discoloration is more prominant in desi than in kabuli types. The leaflets are stiffer and thicker than normal ones. A horizontal cut through the collar region reveals a brown ring of discolored phloem. Many plants die prematurely if they are infected in seedling stage.

Pathogen: Bean Leaf Roll Virus (BLRV).

The disease is caused by virus. The virus particles as in infected pea, are isometric, 27 nm in diameter. The host range of the stunt virus is confined to leguminous plants, such as beans, clovers, fababean, lentil, pea and chickpea. Alfalfa is a symptomless carrier of the virus. The virus is transmitted by *Aphis craccivora* Koch (Kaiser and Danesh, 1971; Nene and Reddy, 1976).

Identification of resistance to stunt is difficult. Screening of chickpea germplasm to stunt is done at ICRISAT subcenter in northern India (Hissar). Planting of susceptible chickpea lines and mixture of legumes is done one month earlier than the normal planting to build up the inoculum and vector population in the disease nursery. A few chickpea lines with moderate resistance to stunt have been identified.

Conclusion

Diseases are the major constraints in the cultivation of chickpea. During the last 10 years, considerable information became available on chickpea diseases. Progress has been made in identifying sources of resistance to important disease of chickpea through screening of germplasm. However concentrated efforts are needed to utilize the information which is available for the improvement of chickpea crop in the Mediterranean countries. Disease surveys in Mediterranean countries are needed to understand the distribution, severity and crop losses caused by the diseases in chickpea. Epidemiological studies of the important diseases in the region will help to formulate effective integrated disease management.

References

ANANDA RAO, P.K. and HAWARE, M.P. (1987): Inheritance of dry root rot (*Rhizoctonia bataticola*) resistance in chickpea. Plant Breeding *98:* 349-352.

CABRERA DE LA COLINA, J., TRAPERO CASAS, A. and JIMENEZ DIAZ, R.M. (1987): Pathogenic variation of *Fusarium oxysporum* f. sp. *ciceri* and *F. solani* to chickpea in Andalusia, southern Spain. Pages 121-123 *in* Proceedings of 7th Congress of the Mediterranean Pathological Union, Sept. 20-26, 1987, Granada, Spain.

ERWIN, D.C. (1958): Verticillium wilt of *Cicer arietinum* in southern California. Plant Dis. Reptr. *42:* 1111.

HARJIT SINGH, KUMAR, J., SMITHSON, J.B. and HAWARE, M.P. (1987): Complementation between genes for resistance to race 1 of *Fusarium oxysporum* f. sp. *ciceri* in chickpea. Plant Pathology *36:* 539-543.

HAWARE, M.P. and NENE, Y.L. (1980): Influence of wilt at different stages on the yield loss in chickpea. Tropical Grain Legume Bulletin *19:* 38-44.

HAWARE, M.P. and NENE, Y.L. (1982): Symptomless carriers of the chickpea wilt fusarium. Plant Disease *66:* 250-251.

HAWARE, M.P. and NENE, Y.L. (1982a): Races of *Fusarium oxysporum* f. sp. *ciceri*. Plant Disease *66:* 809-810.

HAWARE, M.P., NENE, Y.L. and RAJESHWARI, R. (1978): Eradication of *Fusarium oxysporum* f. sp. *ciceri* transmitted in chickpea seed. Phytopathology *68:* 1364-1367.

HAWARE, M.P., NENE, Y.L. and NATARAJAN, M. (1986): Survival of *Fusarium oxysporum* f. sp. *ciceri* in soil in the absence of chickpea. National seminar on management of soil-borne diseases of crop plants. TNAU, Coimbatore, 8-10 Jan. 1986. P.1 (Abstr.).

KAISER, W.J. and DANESH, D. (1971): Biology of four viruses affecting *Cicer arietinum* in Iran. Phytopathology *61:* 372-375.

KUMAR, J. and HAWARE, M.P. (1982): Inheritance of resistance to fusarium wilt in chickpea. Phytopathology *70:* 1035-1036.

NENE, Y.L. and REDDY, M.V. (1976): Preliminary information on chickpea stunt. Tropical Grain Legume Bulletin *5:* 31-32.

NENE, Y.L. and HAWARE, M.P. (1980): Screening chickpea for resistance to wilt. Plant Disease *64:* 379-380.

NENE, Y.L., HAWARE, M.P. and REDDY, M.V. (1981): Chickpea diseases, resistance-screening techniques. Information Bulletin No. 10. International Crop Research Institute for the Semi-Arid Tropics (ICRISAT), Patanohen, India.

PATHAK, M.M., SINGH, K.P. and LAL, S.B. (1975): Inheritance of resistance to wilt (*Fusarium oxysporum* f. sp. *ciceri*) in gram. Indian Farm Sci. *3:* 10-11.

SATTAR, A., ARIF, A.G. and MOHY-UD-DIN, M. (1953): Effect of soil temperature and moisture on the incidence of gram wilt. Pakistan Journal of Scientific Research *5:* 16-21.

SINGH, U.P. and SINGH, R.B. (1983): The effect of soil texture, soil mixture, soil moisture and depth of soil on carpogenic germination of *Sclerotinia sclerotiorum*. Journal of Plant Disease and Protection *90:* 662-669.