J. Mycopathol, Res, ***48(2)*** *: 387—390 (2010)*

Club Root disease of rapeseed-mustard : A review

A. BISWAS[1]*
Pulses and Oilseeds Research Station, Berhampore 742 101, West Bengal

Most of the cruciferous crops are affected by club root disease, caused by an obligate biotrophic fungus *Plasmodiophora brassicae*. The disease has been reported to occur in several countries of the world, In India, it was reported from West Bengal and Orissa. It occurs more frequently in soils which are acidic and poorly drained. All the indigenous varieties belonging to *Brassica juncea*, and *B. napus* (toria, yellow sarson and brown sarson) are moderately to highly susceptible to the disease and causing losses in yield due to the disease is quite high (54%). Recent findings on various aspects of the disease are reviewed.

Key words : Rapeseed-mustard, *Plasmodiophora brassicae*, club-root

INTRODUCTION

The club-root or finger and toe disease is potentially the most dangerous disease of the family Cruciferae, especially rapeseed-mustard, cabbage, cauliflower, radish, turnip etc. Comprehensive description by Kolte (1985) has highlighted the importance of the disease. In the present paper, an attempt has been made to give an appraisal of recent advances on work done so far in India and abroad An intensive review is an urgent need to reinvestigate the various aspects of the disease as it may become a possible future threat and constraint to rapeseed-mustard production.

OCCURRENCE

On rapeseed-mustard, the disease is reported to occur in East Germany (Lembcke *et al.*, 1974), Malay (Anon, 1962), New Zealand (Gibbs, 1931 ; Lobb 1951), Poland (Nowicki, 1976), Sweden (Nilson, 1951), United Kingdom (Paterson, 1972), and U.S.A.(Martin, 1933 ; Stout *et al.*, 1954). The disease has been reported in rapeseed-mustard on the variety YSB - 9 (*Brassica napul L.,* cv. Benoy) from West Bengal (Laha *et al.*, 1985) and in *B. napus* var. *toria* from Orissa (Das *et at.*, 1987).

SYMPTOMS

Club root, as the name suggests is essentially a disease of the root system. At the initial stage, the affected plants show normal healthy growth, but as the disease develops, the earliest above ground symptoms are unthrifty development of the plants showing pale-green or yellowish with leaves, flagging in sunny days, as if the plants are suffering from lack of water. When such plants are pulled, overgrowth (hypertrophy and hyperplasia) of the main and lateral roots become visible in the form of small or large spindle or spherical shaped knobs, called clubs. In certain cases the galls are 8-20 per plant. Tap roots as well as lateral roots and rootlets develop galls. Depending on the type of root of *Brassica* species, the shape of the club varies. When many infections occur close together, most of the root system is transformed into various shaped of malformations. In advanced stages of the disease, due to increase in the size of galls, disruption of the outer tissues of the host takes place and a general decay of the galled tissue sets in and turn black. Gradually loss of foliage takes place. Plants of this kind are without or with very few small flowers and of inferior quality. Number and size of pods, if any, are reduced. Pods are thick and sometimes curved.

*1. Present Address Joint Director of Agriculture (Mycology and Plant Pathology), Rice Research Station, Bankura 722 101, West Bengal * Correspondence. E-mail : biswas.arunava@rediffmail com

YIELD LOSS

Laha *et al.* (1985) reported yield loss of 32.5 per cent in *Brassica napus* var. yellow sarson cv Benoy in West Bengal. Chattopadhyay (1991) was the first who made a systematic study on the disease and recorded losses up to 54 per cent in seed yield in single plant due to club root disease as reflected in pod yield of cv. Benoy in West Bengal.

CAUSAL ORGANISM

The pathogen is *Plasmodiophora brassicae* Woronin. It is an obligate biotrophic fungus. Woronin (1878) first scientifically investigated the disease on cabbage and the nature of its causal organism in Russia. He has worked out its general life history. Other aspects of its biology have been reviewed by Calhoun (1958) and Karling (1968).

The fungus has a plasmodial vegetative stage characterized by a naked, amoeboid, multinucleate protoplast without a definite cell wall. The plasmodium is produced only within the host cell and remains intracellular, with two distinct phases. The first, the primary one, usually results from infection by primary zoospores derived from the resting spores, and the secondary one results from infection by secondary zoospores derived from a zoosporangium. The life cycle has been described in detail by Webster (1970) and Dobson and Gabrielson (1983).

Tommercup and Ingram (1971) have studied the behaviour of the pathogen in callus tissue culture of *B. napus* and have presented the interpretation of the life cycle. Butcher *et al.* (1974), Linss (1978), Buczacki and Ockendon (1979) have studied the biochemistry behind the galling on roots of rapeseed-mustard plants.

DISEASE CYCLE

Survival and transmission of the pathogen : The fungus survives in the form of resting spores in soil. After the death of the galls, the resting spores are released in the soil, and the pathogen thus becomes soil-borne and dispersed in soil as resting spores through farm implements, footwear, flood water, wind blown soil and moving animals. In areas where pond water is used for irrigation and the ponds receive run off water from infested fields, such pond and irrigation water serve as source of primary inoculum for healthy fields (Datnoff *et al.*, 1984).

The pathogen can also survive on cruciferous weeds like Sphepherd's Purse (*Capsella bursa-pastoris* (L.) Medik) Some of the non-cruciferous hosts are also affected by *P. brassicae*. They are *Agrostis* sp. *Dactylis* sp. , *Holcus* sp. , *Lolium* sp. , *Papaver* sp. , and *Rumex* sp. Whether these noncruciferous plants play any part in maintaining the continuity of the disease in the absence of a cruciferous host is not known.

Infection, environmental relations and disease development : Although prolonged resting period is not required for germination of the resting spores, many of them remain viable in soil for 10 years or longer without the presence of host plants. Infected subterranean plant parts disintegrate in the soil, releasing the free spores. While the spores germinate with little or no resting period, a large percentage of them remain dormant for many years. The fungus is one of the most persistent soil invaders. The spores germinate poorly or not at all in alkaline media (pH 7.2 - 7.4). Infection can occur at soil temperatures from 9° - 30°C. Optimum temperature for infection is 17°-25° C. Root hair and cortical infection decreases with decreasing availability of soil water. Larger watered soil pores help in movement of primary zoospores and their fusion to form large secondary zoospores necessary for cortical infection (Dobson, *et al.*, 1982). While infection is limited by low soil moisture (below 45 per cent of water holding capacity) conditions even a temporary rise in soil water, such as just after irrigation, can facilitate infection.

The incidence of the disease is very scanty in the gangetic alluvial region, the major rapeseed-mustard growing areas of West Bengal, but quite severe over a large area of western lateritic tract and its adjacent areas in West Bengal. Loam and clay soils are generally less prone to harbour club root than light soil. The soil pH of the area showing incidence is varying between 5.2 - 6.8. Usually low-lying, poorly drained soil favour club-root, and well drained soils inhibit it (Chattopadhyay and Bagchi, 1989 ; Chattopadhyay, 1991). Disease cycle has been described in detail by Kolte (1985) and many other workers.

VARIABILITY

P. brassicae shows a lot of variation in pathogenicity. Using the European Club root Differential (ECD) set, consisting 15 differential host varieties (five each of *B.campestris, B. napus* and *B. oleracea*), 34 physiologic races have been identified in Europe (Buczacki *et al.*, 1975). In New Zealand, eight races have been reported by Lammerink (1986). B-54 (*B. napus Lina*, toria) was claimed to be resistant from Orissa (Das *et al.*, 1987), but the same variety was found to be highly susceptible (45% infection) in West Bengal (Chattopadhyay and Bagchi, 1989) which indicates a possible existence of separate physiologic race of *P. brassiace* causing club root of rapeseed- mustard in the plains of West Bengal and Orissa. However, it is to be scientifically documented.

MANAGEMENT

The disease can be managed effectively by integrating certain cultural practices which are as follows : (i) eradication of crucifier weeds such as Shepherd's Purse (*Capsella bursa-pastoris*) (ii) growing the crop in fields known to be infected with the pathogen should be avoided, (iii) necessary soil drainage facilities are to be made to avoid water-logging in the field and (iv) very long (over ten years) crop rotation with non-cruciferous crops should be adopted.

Spores of *P. brassicae* do not germinate or germinate very poorly in alkaline soils. On this basis, amendment of infested soil with calcium compounds have been reported by many workers (Fletcher *et al.*, 1982 , Dobson *et al.*, 1983 ; Campbell *et al.*, 1985) Raising the soil pH to the level of 7.2 - 7.4 from initial pH of 5,7 or less by soil amendment with lime (CaO) @ 3 t/ha, 30 days before sowing has been effective (Chattopadhyay, 1991). The soil pH above 7.2 reduces infection and clubbing because thalli abort before producing zoospores (Meyers and Campbell, 1985).

Sen (2005) reported that application of mixture of N(80 kg/ha), P (40 kg/ha), K (40 kg/ha) and three nutrients namely calcium carbonate @ 80 kg/ha, boron @ 20 kg/ha and ammonium molybdate @ 2 kg/ha in an integrated way before sowing may reduce the club root disease severity and enhance the host tolerance in *Brassica juncea* cv. RW 85-89 in West Bengal. Boron, molybdenum alongwith calcium have some antagonistic effect on the pathogenesis of *P. brassicae.*

In Taiwan, Yang and Hsieh (1985) reported good control of club root disease by amending the soil with S - H mixture which consisted of sugarcane bagasse, rice husk, oyster shell powder, urea, potassium nitrate, calcium superphosphate and mineral ash. The last ingredient contained oxide of calcium, magnesium, aluminium, iron and silicon. The mixture was applied @ 0.5 -1 per cent by weight of soil. Mineral ash alone or mixed with oyster shell power was equally effective.

Pseudornonas fluorescens and *Streptomyces graminofasciens* introduced into soil check root hair infection process and club formation in the club root disease of rapeseed-mustard caused by *P.brassicae* (Bhattacharya and Pramanik, 1998).

An alternative biological management process of club root disease by soil application of *Trichoderma harzianum* + *T. viride* (1 : 1) @ 2.5 kg/ha and seed treatment with garlic + clove extract (5%, w/v) before sowing of rapeseed-mustard has been reported by Bhunia and Biswas (2008)

Although certain chemicals like benomyl, quintozene, and other soil fumigants are known to be effective against *P. brassicae*, such disease management methods are not feasible and economical because of the high cost of chemicals and application. Moreover, most of such chemicals are banned in India and other countries due to their soil pollution nature.

Among different management methods, use of resistant varieties appears to be most feasible. According to Chattopadhyay *et al.* (1991,2001) all the indigenous varieties belonging to *Brassica juncea*, *B. napus* (*toria, yellow sarson and brown sarson*) and *B. chinensis* are moderately to highly susceptible to the club root disease. Exotic cultures belonging to B. napus (Tower, Midas etc.), *B. nigra* (ACCBN-479) and *B. carinata* (HC-1, HC-4, HC-05 etc.) are either free from disease or are resistant. Recently, WBBN-1 (Kalyan) a club root resistant variety, has been recommended for the club - root prone western lateritic tract and its adjacent areas of West Bengal.

REFERENCES

Anonymous. 1962. Quarterly Report of the Plant Protection Committee for the South East Asia and Pacific Region, FAO Publ. Bangkok, Thailand, p.12.

Bhattacharya, I. and Pramanik, M. 1998. Effect of different antagonist Rhizobacteria and neem products on club-root of crucifers, *Ind. Phytopath.* **51** : 87

Bhunia, C.K. and Biswas, A. 2008. Biological management of club-root disease of rapeseed-mustard. *Pestology* **32** (2) : 32 - 35.

Buczacki, ST., Toxopeaus, H., Mattusch, P., Johnston, T. D., Dixon, G. R., and Hobolth, LA. 1975, Study of physiologic specialization in *Plasmodiophora brassicae* : proposals for attempted rationalization through international approach. *Trans. Br. Mycol. Soc.* **65** (2) : 295- 303

Buczacki, ST. and Ockendon, J.G. 1979. Role of glucosinolate incidence and clubroot susceptibility of three cruciferous weeds. *Trans. Br. Mycol. Soc.* **72** : 156.

Butcher, DM., El-Tigani, S., and Ingram, D. 1974. The role of indole glucosinolates in the club-root disease of the Cruciferae. *Physiol. Plant Pathol.*, **4 :** 127.

Calhoun, J. 1958. Club-root disease of Crucifers caused by *Plamodiophora brassicae. Commonw. Mycol. Inst. Phytopathol. Paper*, **3** : 108.

Campbell, R. N. , Greathead, R.S., Meyers, D.F. and Boer, G.J. Jr. 1985. Factors related to control of club-root of crucifers in the Salina Valley of California. *Phytopath.* **75**; 665-670.

Chattopadhyay, A.K. 1991. Studies on the control of club root disease of rapeseed-mustard in West Bengal *Ind. Phytopath.* **44** : 397 - 398.

Chattopadhyay, A.K. and Bagchi, B.N. 1989. Occurrence of club root disease on rapeseed-mustard in West Bengal. *Ind. J. Mycol Res.* **27** (1) : 83 - 84.

Chattopadhyay, A.K., Bagchi, B.N. and Roychoudhury 1991. Reaction of some cultures of rapeseed-mustard against club-root disease. *Ind. Phytopath.* **44** : 238-239.

Chattopadhyay, A.K.Moitra, A.K. and Bhunia, C.K. 2001. Evaluation of *Brassica* species for resistance to *Plasmodiophora brassicae* causing club-root of rapeseed-mustard. *Ind. Phytopath.* **54**(1) :131 -132

Datnoff, L.E. , Lacy, G.H. and Fox, J.A. 1984. Occurrence and population of *Plasmodiophora brassicae* in sediments of irrigation water sources. *Plant Dis.* **68** : 200-203.

Das, S. N., Mishra, S.K. and Swain, P.K. 1987. Reaction of some toria varieties to *Plasmodiophora brassicae. Ind. Phytopath.* **40** : 120.

Dobson, R.L., Gabrielson, R.L. and Baker, AS. 1982. Soil water matric potential requirements for root hair and cortical infection of Chinese cabbage by *Plasmodiohora brassicae, Phytopath.* **72** : 1598- 1600.

Dobson, R.L., Gabrielson, R.L. and Baker, AS. and Bennett, L. 1983. Effect of lime particle size and distribution and fertilizer formulations on club-root disease by *Plamodiophora brassicae. Plant Dis.* **67** : 50 - 52.

Dobson, R.L., and Gabrielson, R.L. 1983. Role of primary and secondary zoospores of *Plamodiophora brassicae* in the development of club-root in Chinese cabbage *Phytopath.* **73** : 559-561.

Fletcher, J.T., Hims. M.J. Archer, F.C. and Brown, A. 1982. Effect of adding calcium and sodium salts to field soils on the incidence of club-root. *Ann. Appl. Biol.* **100** : 245 - 251.

Gibbs J.G. 1931. Club-root in cruciferous crops, investigations by Plant Research Station. *N.Z.J. Agnc* 13 : 1.

Karling, J.S. 1968. *The Plasodiophorales*, 2nd ed.Hafner, New York, p.256.

Kolte, S. J. 1985. Rapeseed-mustard Diseases, *In 'Diseases^of Annual Edible Oilseeds Crops', Vol-II, Rapeseed-mustard and sesame diseases,* CRC Press, Inc Boca Raton, Florida. 53 pp

Laha, J.N., Naskar, I., and Sharma, B.D. 1985. A new record of club root disease on mustard *Curr. Sci.* **54** : 1247.

Lammerink, J. 1986. Identification of an eighth race of *Plasmodiophora brassicae*, the cause of club-root, in New Zealand. *N.Z.J.Argic. Res.* **29** : 101 -104.

Lembcke, G., Muller, W and Schlogel, H. 1974. The importance of club root in winter rape cultivation with reference to the Gadebusch area (Schwerin district), Abstr. *Rev. Plant. Pathol.* **53** : 58.

Linss, H. 1978. Investigation on changes of the metabolism of rape plant after infection with *Plasmodiophora brassicae Wor. Abstr. Rev. Plant Pathol.* **5**(5) : 215.

Lobb. W.R. 1951. Resistant type of rape for areas with club root. *N.Z.J. Agric,*. **82** : 65.

Martin, W.H 1933. Plant Pathology - Fifty-third and Fifty- fourth Annual Reports for the 2 year Period ending June 30, 1933, New Jersey Agnc. Exp. Stn. **57**.

Meyers, D. F. and Campbell, R.N. 1985. Lime and the control of club root of crucifers : effect of pH, calcium, magnesium and their interaction. *Phytopath.* **75** : 670 - 673.

Nilson, L. 1951. Some crop diseases in Scania in 1950, Abstr., *Rev. Appl. Mycol.* **30** : 360.

Nowicki, B. 1976. Susceptibility of crucifers grown in Poland to cabbage club root (*Plasmodiophora brassicae* Wor.), Abstr. *Rev. Plant Pathol.* **55** : 2417.

Paterson, W.G.W. 1972. A new race of *Plasmodiophora brassicae* Woronin. *Plant Pathology* (U.K.) **21** (3) : 145.

Sen, P. 2005. Antagonistic effect of Ca, B and Mo on Club-root disease of rape-mustard. *Ind. Agric.* **49** (1 & 2) : 13-16.

Stout, G.L-Altstatt, G.E. and Nakayama, R. M. 1954. Club root of Crucifers. *Bull. Dept. Agric. Calif.* **42** (3) .113.

Tommerup, I.C. and Ingram, D.S. 1971. The life cycle of *Plasmodiophora brassicae* Woronin in *Brassica* tissue cultures and in intact roots. *New Phytologist* **70** : 327.

Webster, J. 1970. *Introduction to Fungi.* University Press, Cambridge.

Woronin, M 1878. Plasmodiophora brassicae, Urheberder der Kohlpflanzen - Hernie. *Jahrb. Wiss. Bot.* **11** : 548 English Trans, by C. Chupp, in *Phytopathof, Classics,* **4,** 1934.

Yang, C.H. and Hsieh, 1985. Effect of soil amendments on club root of crucifers. *Plant Prot. Bull., Taiwan* **27** : 225-231.

(Accepted for publication June 15, 2010)