Stem Gall/Tumor of Coriander

Pathogen: *Protomyces macrosporous*

Taxonomic position of the pathogen: Sub Division: Ascomycotina; Class: Hemiascomycetes; Order: Protomycetales Family: Protomycetaceae

Distribution & Importance: Stem gall/tumor of coriander is a widely distributed everywhere there the coriander is grown. The disease was first reported by Unger (1833) in Great Britain, and the causal organism was described as *Protomyces macrosporus*. In India, stem gall disease of coriander was reported for the first time from Pusa (Bihar) by Sydow and Butler (1911).This disease has been reported from all coriander growing areas of the India. This is a common and widespread disease of coriander causing heavy damage to the crop, especially when associated with wilt. The yield loss up to 33-36% and deteriorates quality of seeds.

Symptoms: The disease appears in the form of gall/tumor like swellings of leaf veins, leaf stalks, peduncles, stems as well as fruits. The swellings of the veins give a swollen hanging appearance to the leaves. The tumors are variable in size and smooth and glossy at first but later rupture and become rough. The tumors are about 3 mm broad and up to 12.5 mm long. Badly affected plants may be killed. Infected plant remains stunted. In the presence of excessive soil moisture, especially under shade, when the stem fails to harden and remain succulent, the tumors are numerous. When inflorescence is attacked seed production is reduced considerably. Shape of coriander seeds change due to effect of the disease.

Pathogen: The pathogen of the disease is *Protomyces macrosporous.* The fungus forms galls of variable sizes on the young, green succulent stems which contain resting spores of the pathogen. Mycelium is composed of intercellular, closely septate and broad hyphae. Hyphal branching is irregular and chlamydospores are formed by swelling scattered hyphal cells. These ellipsoid or globose chlamydospores are surrounded by three layered wall which is thick and hyaline.

Disease Cycle: The pathogen perpetuate through soil and seed. The pathogen survives in the soil in the form of chlamydospores and this act as the primary source of infection. The *P. macrosporous* has a complex life cycle including ascospores and chlamydospores. Spores reach the hosts via air movements and are spread from the galls that develop on the petioles, midrib veins, and lamina; they will only germinate on the correct host. In soil the fungus overwinters in the form of chlamydospores. The chlamydospores germinate in the presence of water by rupturing the outer wall. The inner wall is pushed out to form vesicle, which appears in continuation with the mouth of the crack. The protoplasm passes into this vesicle and the nucleus divides several times forming 100 to 200 daughter nuclei. The protoplasm also divides by cleavage into several uni-nucleated masses. This structure is known as “Synascus”. On maturity, these spores separate and collect in the center of the vesicle. The latter bursts and the spores are set free. These spores further multiply by budding in yeast like fashion and cause infection of the host.

Favourable condition: High soil moisture along with shade, pH 7.4 to 8.4.

Management:

1. Use clean and healthy seeds for sowing. Infected seed having gall should not be used for sowing
2. Field sanitation and removal of infected plants from field.
3. Soil solarisation: Cover the soil beds with polythene sheet of 45 gauge (0.45 mm) thickness for three weeks before sowing for soil solarisation which will help in reducing the soil-borne inoculum.
4. Use of resistant varieties.
5. Rotate the crop with non-host crops.

6. Seed treatment with captan 50 SD or thiram 75 SD @ 2g per kg seed before sowing.
7. Spray 0.1% solution of carbendazim when the symptoms start appearing and repeat the spraying at an interval of 20 days.