

HISTOPATHOLOGY OF *SYNCHYTRIUM* GALLS ON *SESAMUM INDICUM*

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Synchytrium sesamicola Lacy. causes warty galls of *Sesamum indicum* L. in Rohilkhand. The disease occurs regularly, inflicting heavy losses to the crop in low lying areas. Histopathological studies of galls reveal some specific changes in the anatomy of the affected parts. In general, the warty galls are formed of homogeneous tissue, without intercellular spaces, produced by enlargement (Hypertrophy) of the invaded, as well as neighbouring cells. Stem galls show a distinct lignified epidermal layer. Epidermis of gall tissue is invariably uniseriate. Possible role of trichomes in the infection is emphasised. Leaf galls are associated with the loss of chloroplast in hypertrophied mesophyll and distortion of midrib bundles. Severe infestation results in reduction of cortex, crushed phloem, inconspicuous endodermis and swollen trichomes. Infected calyx and corolla become massive, irregular and contain various reproductive stages of pathogen on outer peripheral side.

Key words : Histopathology, *synchytrium sesamicola*, *Sesamum indicum*, warty galls, Hypertrophy, Hyperplasia sesame galls.

Synchytrium is an intracellular, obligate parasite usually confined in the epidermal cells. The pathogen induces gall formation on various infected parts mainly on the stem and underside of the leaf blade. The mass of invaded host cells nearly hypertrophy and burst outward to form large blisters. While studying the Indian *Synchytrium* Lacy (1950) reported the warty galls on *Sesamum indicum* infected with *Synchytrium sesamicola*. The pathogen is highly destructive and causes considerable damage to the Sesame crop (Pavagi, 1978).

The histopathology of *Synchytrium* galls on different host plants has been investigated by several workers (Akai, 1951; Karling, 1954). Recently Prakasa Rao (1973) has also given a detailed account of anatomical anomalies caused by *Synchytrium* sp. on various host plants.

Host specificity has been regarded as an important character of *Synchytrium* sp. A perusal of literature reveals that the anatomical anomalies caused by *Synchytrium sesamicola* on *S. indicum* has been inadequately studied (Bhargava & Singh, 1979; Srivastava *et al.*, 1982). Most of the studies conducted in India and abroad are confined to the taxonomy of the species, development of resting sporangia and its germination (Kole, 1965; Lang & Olson, 1981 a & b; Variar & Pavgi, 1981 a & b; Hampson, 1986; Price, 1987).

MATERIALS AND METHODS

Infected material was collected during the survey

of Sesame diseases in Rohilkhand and was fixed and preserved in FAA and 70% alcohol respectively. Preserved material was dehydrated through graded series of alcohol. Blocks were prepared in customary way by embedding the dehydrated material. Microtome sections, 8-12 μ m thick were stained with safranin-fast green combination.

OBSERVATIONS

S. sesamicola produces a large number of small, granular, translucent galls on infected parts. Marked characteristic, localised swelling of diseased stem towards the lower unbranched region is caused by the densely aggregate galls (Fig. 1). Infected parts become rough, irregular and warty when dry. Galls are scattered on leaves, petioles and floral parts. Young branches, axillary buds and floral parts are greatly swollen and entirely hypertrophied in case of severe infection.

Anatomy of Stem : The stem is quadrangular in cross section having prominent protuberances due to ridges and furrows. The epidermis is single layered with glandular hairs and multicellular tapering trichomes, which are predominantly confined to the furrows of younger stem. Cortex is many layered thick and is differentiated into outer collenchyma and inner chlorenchyma. Collenchyma is several layers deep in the ridges. The vascular ring is complete showing normal secondary growth. Central pith is massive and parenchymatous (Fig. 2).

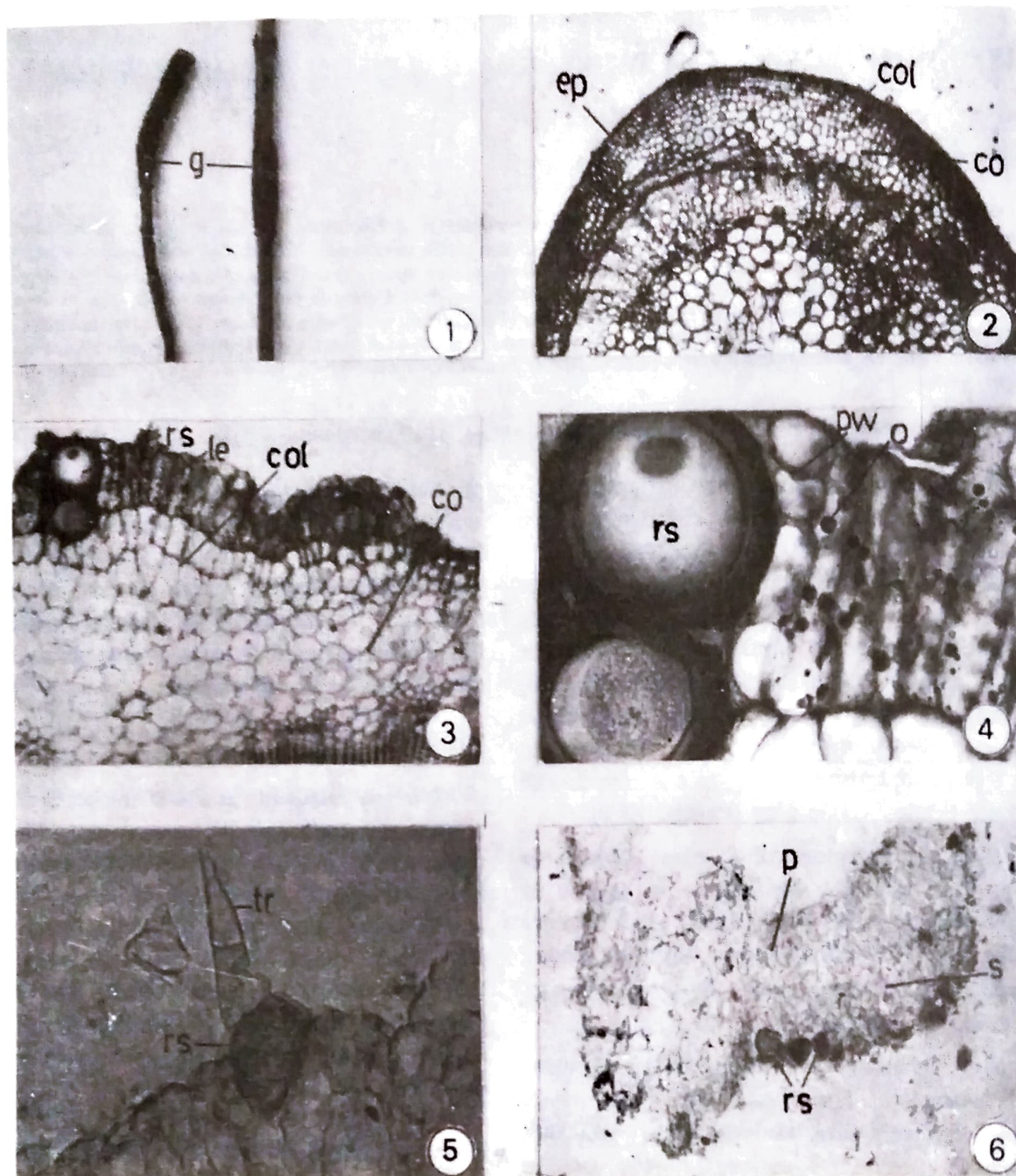


Fig. 1-6. *Synchytrium sesamicola* Lacy. on *Sesamum indicum* L. (ep-epidermis, col-collenchyma, co-cortex, tr-trichome, rs-resting spore, g-galls, p-palisade tissue, s-spongy parenchyma, o-oil globules, pw-periclinal wall, le-lignified epidermis, cl-calyx, ct-corolla tube).

1. Infected stem. 2. T.S. normal stem (a part). x 99. 3. T.S. infected stem- a portion showing uniseriate lignified hypertrophied epidermal layer and multiseriate hypertrophied collenchymatous layer. x 99. 4. T.S. infected stem- enlarged portion. Note periclinal walls in enlarged epidermal cell, oil globules and resting spores. x 396. 5. T.S. a part of infected stem, note the infection in basal cell of trichome. x 396. 6. T.S. infected leaf showing hypertrophy of spongy tissue and resting spores on lower side. x 99.

Anatomy of stem gall : The outline of the infected stem in cross section is highly irregular. The characteristic quadrangular shape, is not evident.

The epidermis of galls is uniseriate, radially stretched and lignified. The cells of 2-3 layered hypodermis are invariably enlarged (Fig. 3). In case of severe

infection, the gall reaction extends even to some cells of cortex. Elongated epidermal cells are lignified, having dense cytoplasm, conspicuous oil globules, lipid bodies and tannins. Distinct 2-4 periclinal divisions are noticed in the elongated epidermal cells, which are in direct contact of the infected cells. However such divisions are not marked in other cells (Fig. 4).

The young stem galls consist of two distinct zones-(1) The outer darker, single-layered zone, formed by the elongation of epidermal cells and (2) the inner transparent multilayered zone, formed by enlargement of hypodermal cells. The resting spores are restricted to the epidermal region of the gall. Hypertrophied collenchymatous cells are devoid of chlorophyll (Fig. 3).

A number of trichomes are associated with gall tissue. Interestingly resting spores are noticed within the basal cell of the trichome (Fig. 5). At later stage of infection, the trichomes become shrivelled due to the loss of protoplasmic contents. Vascular supply of stem is unaffected. Reduction of cortex and crushing of phloem were noted occasionally.

Anatomy of normal leaf : Both upper and lower epidermis of leaf are uniseriate and consist of barrel-shaped cells of unequal size. Stomata are present on lower side, while glandular non-glandular trichomes are distributed on both the sides. Palisade is double layered having compact and elongated cells. Spongy tissue is 2-3 layered with loosely arranged chlorenchyma. The vascular bundle is in the middle.

Anatomy of leaf gall : Most of the leaf galls are produced on the lower side and are associated with midrib or veins. Usually the leaf galls are formed by the radial elongation of epidermal cells and enlargement of spongy cells. In case of very severe infection the palisade tissue and upper epidermis are also affected. Lose of chlorophyll is well marked in hypertrophied cells of leaf (Fig. 6).

The vascular bundles in the veins are deformed due to severe infection. Many trichomes are also associated with leaf galls.

Anatomy of petiole : Epidermis of normal petiole is single layered consisting of rectangular cells of unequal size. Trichomes are usually three celled. Hypodermis is collenchymatous while cortex is parenchymatous. Vascular bundles are small and usually placed eccentrically.

Anatomy of petiole gall : Petiole gall is the product of enlarged epidermal, hypodermal and cortical cells. Gall tissue is homogeneous and lacks zonation, seen in stem galls. Vascular bundles remain unaltered.

Anatomy of floral parts : Outermost part of the flower is the calyx consisting of 5 sepals. They are composed of epidermis, surrounding the spongy mass of parenchyma and central vascular supply. Peripheral surface of sepal is hairy. Corolla tube is formed by the union of 5 petals having a mass of spongy cells enclosed within epidermal layers. Ovary is situated in the middle. Anthers are located outside the ovary. (Fig. 12).

Anatomy of infected floral parts : The flowers receive infection at bud stage through the rain splash. Pedicels and sepals are infected most severely. Rarely the infection reaches up to the petals. The galls on pedicel are formed by proliferation of epidermal and cortical tissue (Fig. 11).

Infection on the calyx is towards its outer peripheral surface which has scattered trichomes. Affected sepals become thick and massive. Gall tissues constitute hypertrophied epidermis and few layers of the spongy tissue. Affected cells are devoid of chlorophyll and other pigments. Trichomes on galls are shrunk and devoid of cell contents (Fig. 7, 8).

Corolla, when infected, becomes fleshy and irregular. Cellular elongation is restricted to the epidermis and some neighbouring cells of homogeneous parenchyma (Fig. 9).

Infection of stamens and ovary has not been observed.

DISCUSSION

Synchytrium galls are hyperplastic in which the invaded host cells are induced to enlarge

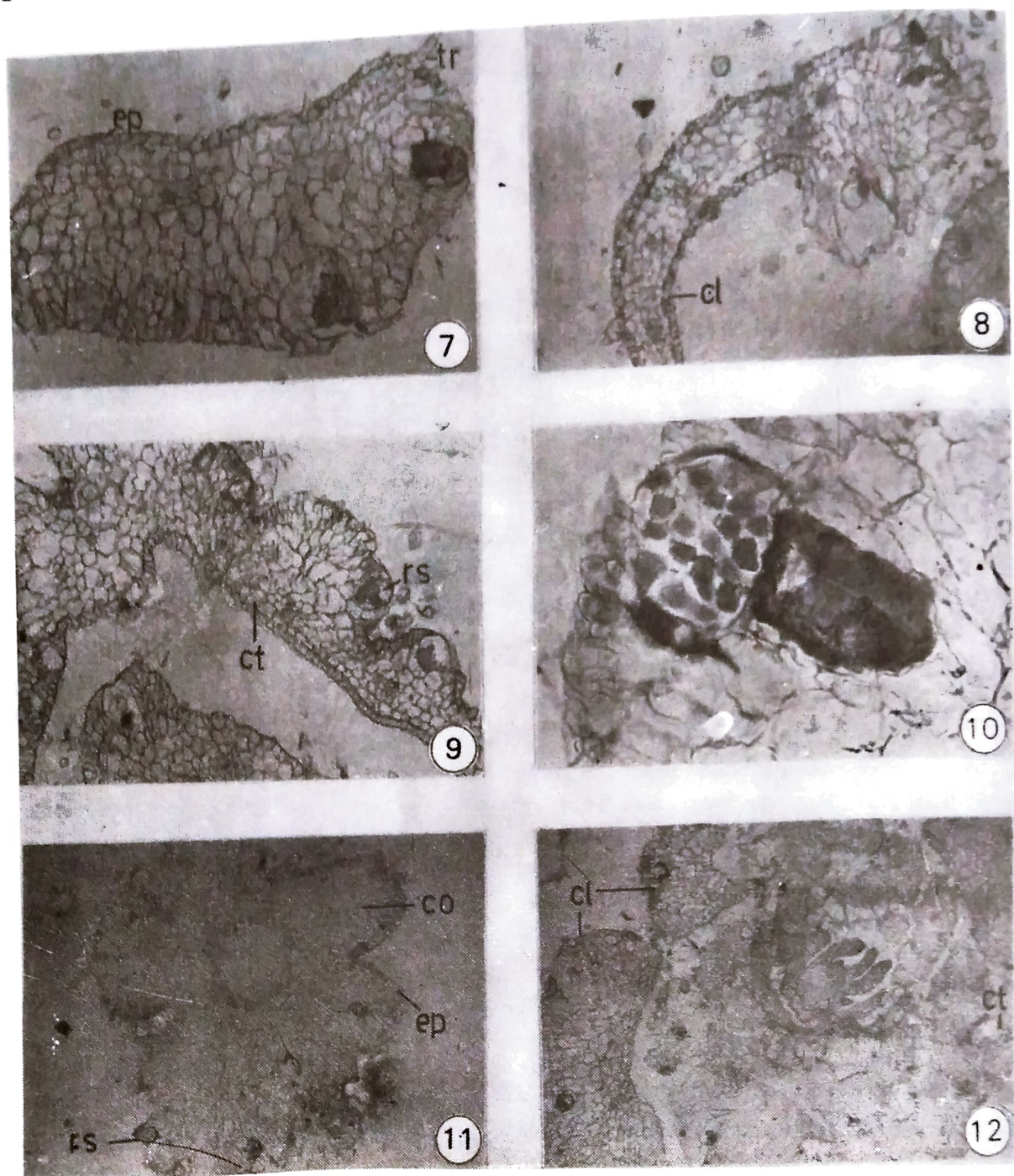


Fig. 7-12. *Synchytrium sesamicola* Lacy. on *Sesamum indicum* L.

7. T.S. infected sepal, note enlarged outer peripheral tissue. x 99. 8. T.S. infected sepal showing partial infection. x 99. 9. T.S. infected corolla tube showing hypertrophy in outer peripheral tissue. x 99. 10. T.S. a part of infected calyx, note the sorus produced. x 396. 11. T.S. infected pedicel. x 40. 12. T.S. infected floral bud, note parasite in peripheral parts of sepals and corolla tube. x 40.

(Hypertrophy) or divide (Hyperplasia) in response to pathogenic activity. The metabolites produced by the pathogen alter the physiological status of the affected cells, which subsequently enlarge and/

or divide producing characteristic swellings termed galls.

The galls produced by *Synchytrium sesamicola* on *Sesamum indicum* (Sesame) are warty and scattered

on the infected parts. Stem galls are mostly aggregated locally to form a characteristic knot-like swellings of the axis (Fig. 1). The galls on other infected parts are mostly scattered and do not exhibit such localization.

Stem galls of Sesame are chiefly the product of hypertrophy. Histopathological study reveals that soon after infection, the infected as well as neighbouring cells of epidermis get radially stretched having the infected cell as activation centre. Soon after, the collenchymatous cells of hypodermis in the affected area are enlarged to form 2-3 layered gall tissue below epidermis. Involvement of outer cortical tissue was noticed only in cases of very severe infection in branches, petiole and pedicels. Hypertrophied gall tissues are invariably without intercellular spaces. Major role of hypertrophy in production of *Synchytrium* galls on various hosts has also been emphasised by Prakasa Rao (1973).

Developmental study of galls reveals that the elongation of stem epidermis is accompanied with the thickening of cell wall and accumulation of oil-globules and tannins in them. Enlarged hypodermal tissues are however thin walled and without oil-globules. The tissue of young stem gall is distinctly divisible into two zones-

1. The outer uniseriate dark zone, formed by radially elongated and thickened epidermal cells, filled with oil-globules and tannins. The pathogenic stages are confined only to this zone.
2. The inner multiseriate colourless zone formed by the enlarged hypodermal/cortical cells, which are thin walled with less dense cytoplasm.

The zonation makes the Sesame warts tissue heterogeneous in nature which has not been reported in any other host studied so far. Thickening of elongated epidermal cells may be attributed to the deposition of lignin formed from polymerization of phenolic alcohols. The lignification of epidermal cells in the stem gall results in definite zones. Such type of zonation has not been noticed in galls of leaf, petiole, pedicel and floral parts. Lignification and suberization of epidermis in

Synchytrium galls has also been reported by Akai (1951) and Prakasa Rao (1973).

Gall epidermis in all the infected parts of Sesame is uniseriate. The finding is in contrast to the observations of Prakasa Rao (1973), who noticed biseriate or triseriate epidermis in *Peristrophe*, *Justicea* and *Launea* infected by *Synchytrium* sp. Quite distinct 2-4 periclinal walls are observed only in the elongated epidermal cells, which are in direct contact with infected cell bearing resting spores (Fig. 4). Such periclinal divisions are not noticed in other affected cells.

Trichomes play a significant role during infection of *Synchytrium* on Sesame. Occurrence of resting spore in the basal cell of trichome, association of large number of trichomes with gall tissue and the initiation of infection on stem mainly from within the furrows, having abundant trichomes, show the direct role of trichomes in pathogenesis. The trichomes provide a suitable site for the settlement of fungal zoospores which subsequently cause infection. Increase in the number of trichomes in the gall tissue has also been reported by Prakasa Rao (1973) in the *Synchytrium* galls of *Leucas* and *Peristrophe*.

Galls of leaf, sepals and petals are homogeneous, formed mostly by the enlargement of epidermis and few spongy parenchyma cells. Gall tissues are without intercellular spaces. Loss of chloroplast and other pigments is uniformly observed. Mostly the galls are produced on lower side of the leaf and outer peripheral side of calyx having trichomes. Corolla is occasionally infected. In case of very severe infection, hypertrophy is noticed up to palisade and upper epidermis of leaf. Distortion of vascular bundles is observed only in highly diseased leaves. These findings are in conformity with the observations of Prakasa Rao (1973) on *Justicea* and *Leucas*. All the reproductive stages of the pathogen are located in the infected parts. Resting spores are mostly noticed in stem and leaves, while sporangial stages are predominantly present within the gall tissue of infected floral parts. The sori contain 16-32 sporangia (Fig. 10).

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