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Phytopathology

ETIOLOGY OF THE WHITE-TIP NEMATODE DISEASE IN RICE

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The white-tip nematode feeds on individual cells of tender leaf-sheaths, leaf-blades, outer wall and base of embryonic ovary, inner walls of lemma and palea and lodicules, causing necrosis and browning. In the vegetative stage of plants, the unfurling leaf carries the white-tip symptoms and the nematodes. Injury to flowers leads to the partial filling of kernels, and the hypertrophy and necrosis of cells lining the inner walls of lemma and palea causes shrivelling of seed coat in the ripening grain.

INTRODUCTION

The white-tip nematode (*Aphelenchoides besseyi* Christe, 1942) is ectoparasitic feeding on unfurled leaves of the rice plant. This results in withering of leaves from tip downwards giving them a whitish whip-like appearance during the wet season. Due to the semblance of the waving white-tips in fields to fire-flies, this disease is also known as fire-fly blast' in Japan (Ichinohe, 1973). Before heading stage of the crop, the leaf margins of infested plants are often curled, especially the flag leaf (Vuong, 1968). Plants become stunted with reduced tiller development and these symptoms have collectively been confused with calcium or magnesium deficiency in the USA (Feakin, 1970). At the heading stage of the crop, the flag-leaf emerges in a twisted manner, ear length reduced and some glumes are rendered partly or completely chaffy (Vuong, 1968; Haung & Huang, 1972; Nandakumar *et al.*, 1975). Abnormal development of spikelets was also reported as a result of nematode injury in flowers (Rao, 1976). Accumulation of nematodes at the base of florets suggests that the floral parts are also damaged, but no evidence of histological aberrations was detected (Haung & Huang, 1972).

With a view to investigate the etiology of the white-tip disease in rice, inoculations were made to healthy plants under green-house conditions.

MATERIALS AND METHODS

A. besseyi was cultured on mycelial mats of *Curvularia senegalensis* isolated from seeds of rice var I.R-8 harvested a month earlier. The fungus was grown on a substrate containing 4% oat meal, 2% agar and 1% sucrose. Nematodes, surface-sterilized in calcium hypochlorite 1.5% were inoculated to the substrate when the mycelia grew and spread to $\frac{1}{3}$ of the surface of substrate in Petri-plates (10 cm diam). Nematodes were collected 16 days following inoculation from the Petri-plates by the emthod described earlier (Nandakumar *et al.*, 1975).

Five days old sprouts of rice cult. CRM 13-3241 (NSJ 200 × *Padma*) were placed in an aqueous suspension of *A. besseyi* adjusted to give 200 nematodes/sprout for three days in the green-house. The sprouts were later removed and planted in sterilized soil in pots. Similarly, potted plants of CRM 13-3241 were inoculated at ear initiation stage with an injection syringe to provide 2000 nematodes in the site between the leaf-sheath and primordium of earhead in 24 plants. Inoculated plants were sampled six times at intervals of four days after inoculation to study the symptomatology and histopathology of tissues (Jensen, 1962).

Results and Discussion

Symptoms of nematode injury

The tip of the young folded leaf was turned pale yellowish green on the 8th day after inoculation as reported by Yoshii and Yamamoto (1950). Typical white-tip appeared on the 16th day as the manifestation of the earlier damage to the leaf in the primordium (Fig. 1 A). As the leaf unfurled, it carried the nematodes and the white-tip (Nandakumar *et al.*, 1975). In some inoculated plants (about 15%) the symptom of the white-tip was not manifest in spite of the prevalence of the nematode in the inner whorl of leaf-sheaths. The masking of the symptom may be due to the nematode attack on the leaf sheath but not on the embryonic leaf in the primordium. Such a phenomenon has been reported earlier (Muthukrishnan *et al.*, 1974). Plants inoculated at the ear initiation stage did not show the twisting of the leaf sheaths but, the flag leaf was twisted, confirming the earlier reports (Yoshii & Yamamoto,1951; Todd & Atkins, 1958; Fukano, 1962; Muthukrishnan *et al.*, 1974).

Histopathology

Nematodes reached the interspace between the outer and the fourth leaf sheath and coleoptile, and fed ectoparasitically on the cells. As a result, necrotic cells developed in the inter-veinal region (Fig. 1 B). The coleoptile contained nematodes and due to necrosis at feeding sites, the coleoptile turned putrescent (Fig 1 C). Nematodes reached the inner whorl of imbricate leaf sheaths to feed, by the 12th day after inoculation. The cells immediately below the inner epidermis were necrotic and necrosis spread to palisade cells some of which were either empty or partly filled with chloroplasts (Fig. 1 D). At 16-20 days after inoculation, several nematodes (80%) reached the leaf blades and others still remained in the interspaces between the leaf sheaths, more particularly in the innermost leaf sheath. The tender cells of leaf sheaths showed hyperplastic cells (Fig. 1 D). In older leaf sheaths, the nematodes fed on cells below the epidermis causing lesions and the cells immediately below th deamaged cells also turned brown (Fig. 1 E). The cell discolouration spread towards the nearest vascular bundle which also turned necrotic and later black (Fig. 1 E). Occasionally, nematodes entered the leaf tissue moving inside the cavities formed by puncturing and feeding of cells. Due to damage, this portion of leaf emerged in a longitudinally rolled up fashion, a symptom resembling infestation by thrips in rice.

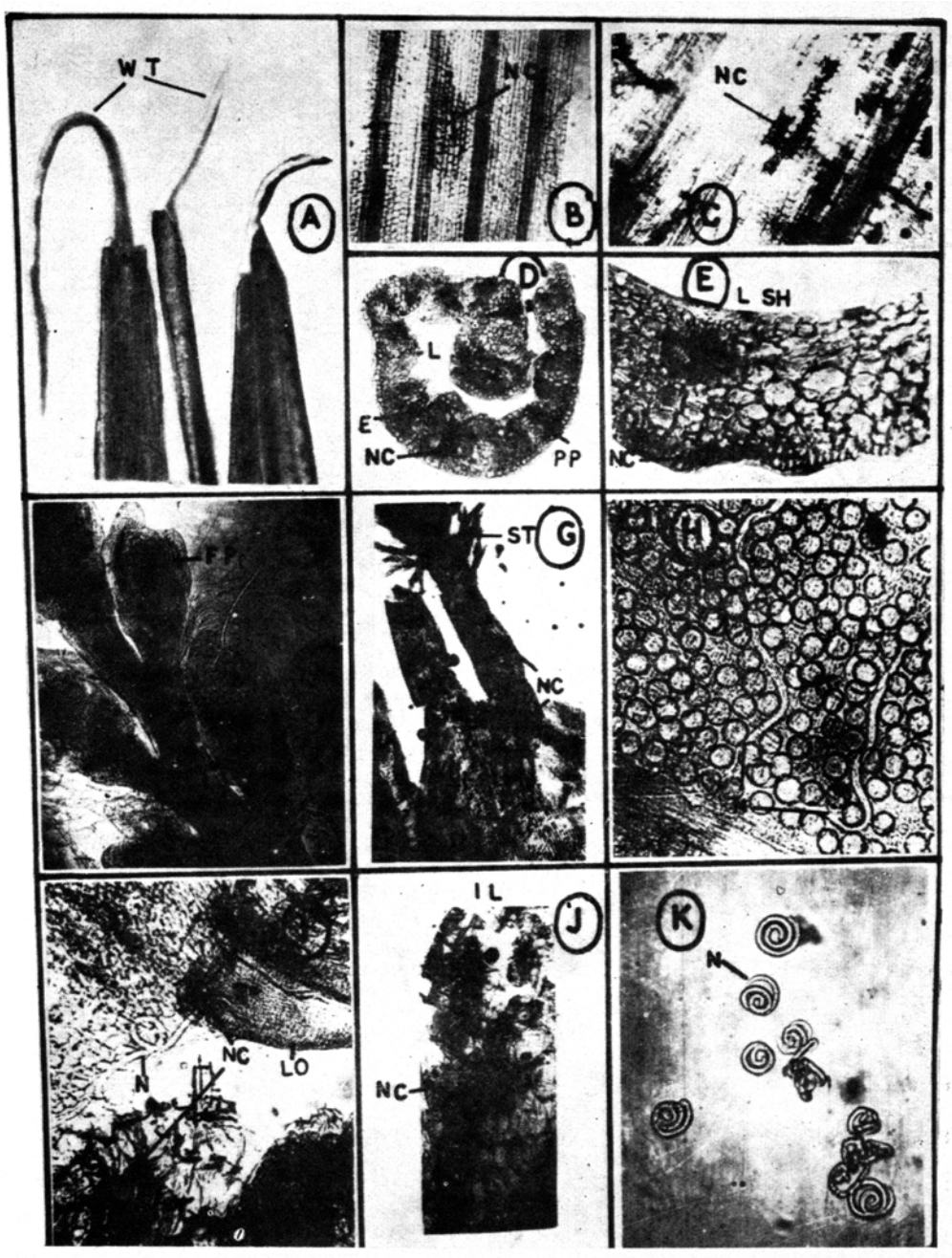

Fig. 1. Damage due to white-tip nematode in rice. *A*, White-tip (WT) on leaves; *B*, Necrosis (Nc) in the inter-veinal area of leaf; *C*, Spread of necrosis (Nc) to vascular tissue of leaf sheath; *D*, C.s tender leaf (*E*- Epidermis, *L*- inner surface of leaf, *Nc*-Necrotic cells, *PP*-Palisade parenchyma). *E*, C.s leaf sheath, showing spread of necrosis into tissues (Nc); *F*, Developing flowers. (*Fp*, Floral primordium, with nematodes (N) penetrating inside); *G*, Ovary. (*Nc*,Necrotic cells, *St*, Stylet); *H*, Pollen sac, with nematodes; *I*, Lodicule (LO) attacked by nematodes (N). Necrotic cells (Nc) in the inner wall of palea; *J*, Inner wall of lemma (IL) showing necrotic cells (Nc); *K*, Nematodes (N) coiled up in resting stage.

Inoculation to ear primordia indicated that the nematodes entered the developing flowers through the narrow opening at the tip of the embryonic lemma and palea (Fig. 1 F). Nematodes fed indiscriminately on the tender floral parts. Before the flower opened, the nematodes attacked the outer wall of the ovary from its base to its tip, the style stigma, the lodicules and the inner walls of lemma and palea. Cells damaged were devoid of contents and were brownish to black in colour (Fig. 1 G). After anthesis, the nematodes fed on the rotting pollen and moved freely in the mass of pollen cells inside the pollen sac (Fig. 1 H). By the 12th day, the lodicules showed necrotic cells (Fig. 1 I). The inner walls of the lemma and palea showed hypertrophy of cells which were also necrotic (Fig. 1 J). As a result of this damage, the lemma or palea of the ripening grain caved in and appeared crinkled or shrivelled up due to unequal growth of tissues of inner and outer walls.

The nematodes caused ill-developed grains apparently due to excessive damage to the cells at the base of ovary and lodicules. Cell damage in the region below the ovary rendered accumulation of starch in the kernel difficult and damage to lodicules resulted in the poor closure of lemma and palea after anthesis and fertilization of the flower, exposing the developing ovary to dehydration. Complete chaffiness was due to the infestation by *A. besseyi* during floral morphogenesis, particularly at the time of kernel development. At this stage, damage to even a few cells interferes with ovogenesis and accumulation of starch and other associated processes of grain development as a result of which the glumes become devoid of kernels. As the earheads ripened, the nematodes entered into resting stage and remained at the base of the lemma and palea in a crinkled and dry stage (Fig. 1 K).

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