C.I.H. Descriptions of
Plant-parasitic Nematodes
Set 2, No. 27

RADOPHOLUS SIMILIS

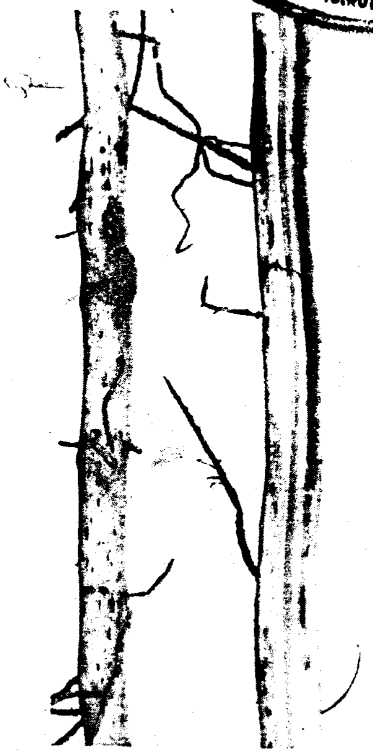

Fig. 2 Left: Banana root with surface lesions. Right: Root split longitudinally to show cortical lesions. The stele has not been invaded. (Photograph: Rothamsted Experimental Station.)

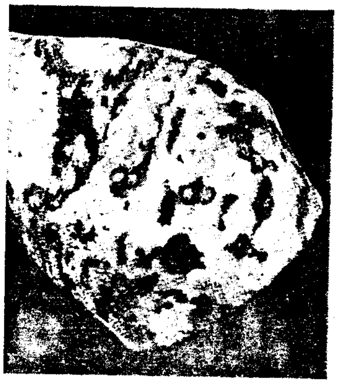

Fig. 3 Pared banana rhizome showing lesions surrounding embedded roots. (Photograph: after Stover, 1972.)

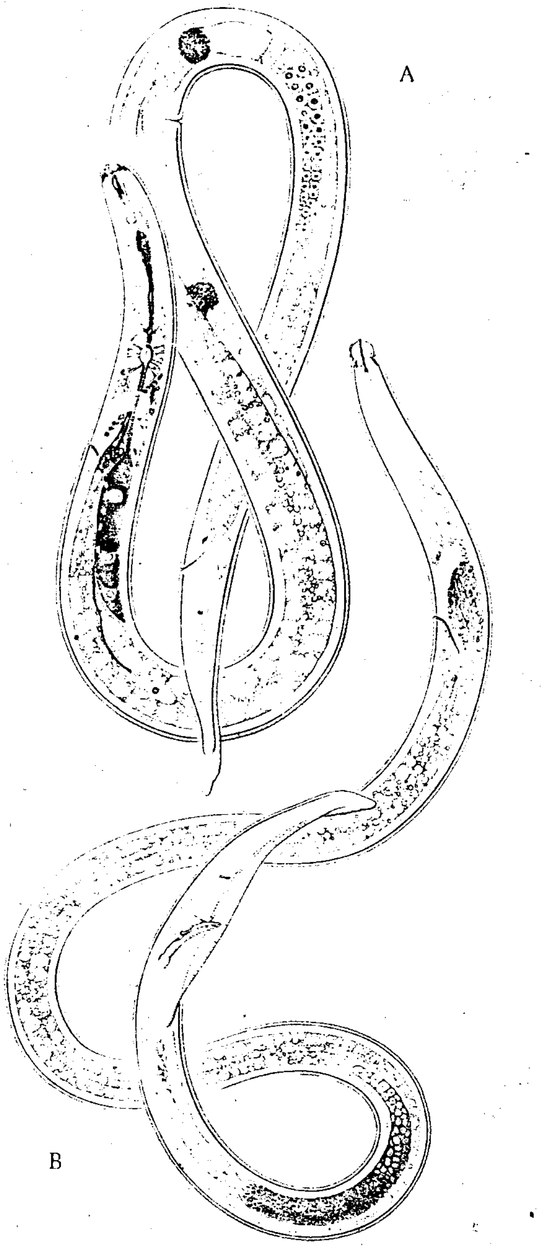

Fig. 1 *Radopholus similis*. *A*. Female. B. Male. (After Cobb, 1915.)

Radopholus similis **(Cobb, 1893) Thorne, 1949.**

Syn. *Tylenchus similis* Cobb, 1893; *Tylenchus* (*Chitinotylenchus*) *similis* Cobb, 1893 (Micoletzky, 1922); *Anguillulina similis* (Cobb, 1893) Goodey, 1932; *Tylenchus* (*Tylenchorhynchus*) *similis* Cobb, 1893 (Filipjev, 1934); *Rotylenchus similis* (Cobb, 1893) Filipjev, 1936; *Tylenchus granulosus* Cobb, 1893 (considered a senior synonym by Sher, 1968); *Anguillulina granulosa* (Cobb, 1893) Goodey, 1932; *Bitylenchus granulosus* (Cobb, 1893) Filipjev, 1934; *Tetylenchus granulosus* (Cobb, 1893) Filipjev, 1936; *Tylenchus acutocaudatus* Zimmermann, 1898; *Anguillulina acutocaudatus* (Zimmermann, 1898) Goodey, 1932; *Tylenchus* (*Tylenchorhynchus*) *acutocaudatus* Zimmermann, 1898 (Filipjev, 1934); *Tylenchus biformis* Cobb, 1909; *Anguillulina biformis* (Cobb, 1909) Goodey, 1932; *Tylenchus* (*Tylenchorhynchus*) *biformis* Cobb, 1909 (Filipjev, 1934).

MEASUREMENTS (Topotypes, after Sher, 1968) 12♀♀: L = 520–880 (690) μ; a = 22–30 (27); b = 4.7–7.4 (6.5); b′ = 3.5–5.2 (4.5); c = 8–13 (10.6); c′ = 2.9–4.0 (3.4); V = 55–61 (56); spear = 17–20 (19) μ; o = 12–20 (18).

5♂♂: L = 590–670 (630) μ; a = 31–44 (35); b = 6.1–6.6 (6.4); b′ = 4.1–4.9 (4.8); c = 8–10 (9); c′ = 5.1–6.7 (5.7); spear = 12–17 (14) μ; spicules = 19–22 (20) μ; gubernaculum = 8–12 (9) μ. (Topotypes, after

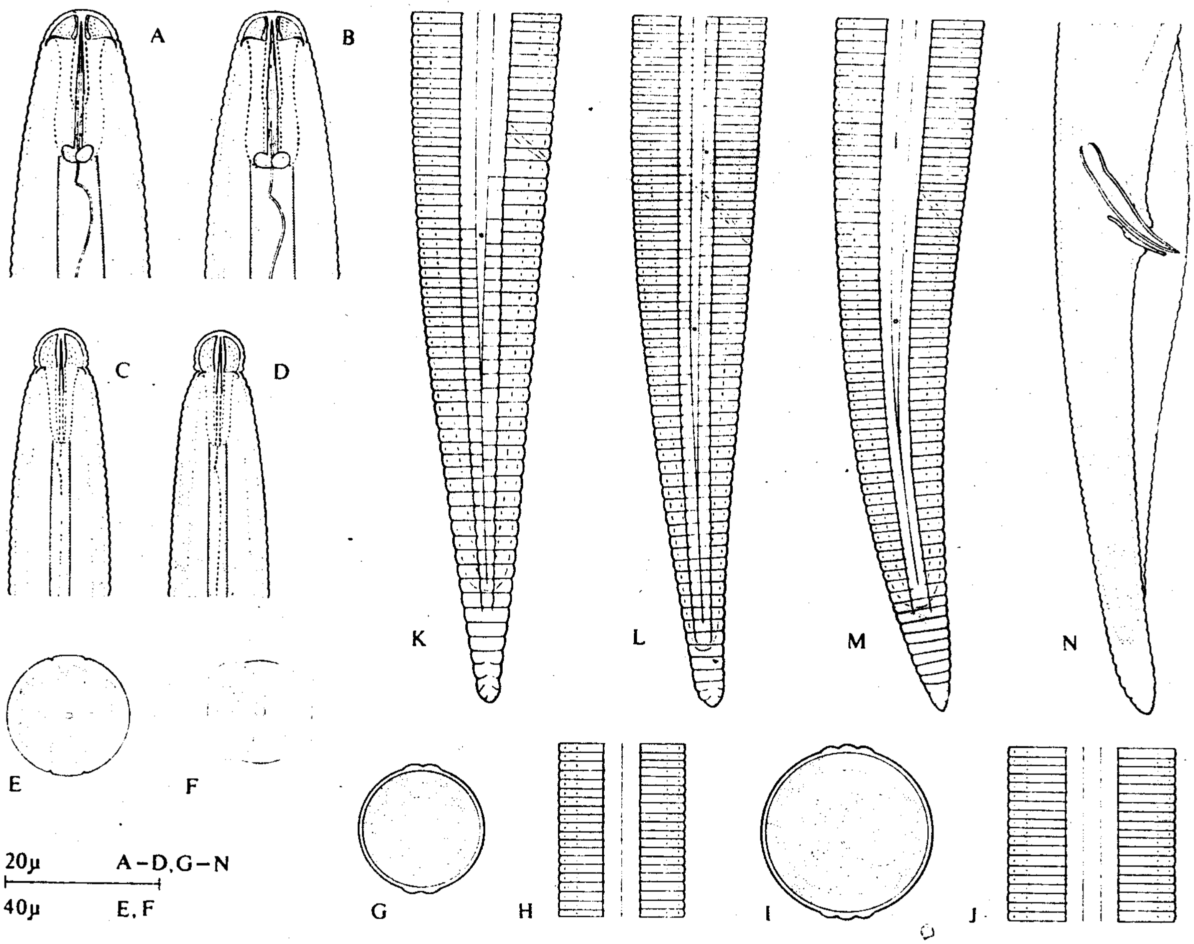

Fig. 4. *Radopholus similis.* Topotypes: A–B. Female, anterior end. C–D. Male, anterior end. E. Female, face view. F. Male, face view. G. Juvenile, cross section near mid-body. H. Juvenile, lateral view near mid-body. I. Female, cross section near mid-body. J. Female, lateral view near mid-body. K–M. Female, posterior region. N. Male, posterior region. (After Sher, 1968: courtesy of The Helminthological Society of Washington.)

Taylor, 1969): 20♀♀ (young): L = 540–660 (605.2) μ; V = 53–58 (55.9); tail length = 57–77 (66.9) μ; spear = 18 μ; phasmids from tail terminus = 44–61 (53) μ.
6♀♀ (gravid): L = 610–745 (685.8) μ; V = 52–57 (55.3); tail length = 52–74 (59.8) μ; spear = 18 μ; eggs = 50–68 (56.1) μ × 19–30 (23.3) μ.
12♂♂: L = 535–650 (585) μ; tail length = 64–86 (72.8) μ; spear = 12 μ; spicules = 18–19 (18.2) μ; gubernaculum = 10–11 (10.3) μ; phasmids from tail terminus = 46–58 (53.7) μ.

DESCRIPTION **Female:** Body straight to slightly arcuate ventrally; cuticle distinctly annulated. **Lateral field with 4 incisures,** not areolated except towards extremities, arising from near median oesophageal bulb and ending near tail terminus; **inner incisures coalescing near middle of tail.** Lip region hemispherical, sometimes offset, **usually with 3–4 annules**; sclerotization strong; dorsal and ventral arms of framework not wider than submedians; lips 6, equal. Anterior cephalids just posterior to labial sclerotization. **Spear about 18 μ long, with well developed round basal knobs which are usually indented anteriorly**; dorsal knob sometimes appearing larger than subventrals. Median oesophageal bulb well developed, round to oval, valvular apparatus prominent. Oesophageal glands 3, in separate lobes, overlapping intestine dorsally and dorso-laterally; dorsal gland anteriormost. Hemizonid 3 annules long, just anterior to excretory pore which is at or just behind the level of the oesophago-intestinal valve. Vulva prominent, just postequatorial. Reproductive organs paired, opposed, outstretched. **Spermathecae spherical, usually packed with small rod-shaped sperms.** Ovaries generally with a single row of oocytes. Intestine filled with spherical granules, indistinctly overlapping rectum. **Tail somewhat elongate-conoid with a narrow rounded or indented terminus.**

Male: Oesophagus and spear degenerate; median bulb and valvular apparatus indistinct, spear without distinct knobs. **Lip region elevated, 4-lobed, with lateral lips considerably reduced,** not strongly sclerotized, with 3–5 annules posteriorly. Hemizonid just anterior to excretory pore which is usually 2–3 body widths behind median oesophageal bulb. Single testis, outstretched anteriorly; spermatocytes in 3 rows followed by 5; spermatozoa rod-like. **Bursa coarsely crenate, enveloping about 2/3 of tail. Spicules strongly cephalated, 18–22 μ long,** with pointed distal ends. **Gubernaculum rod-like, protrusible, with distinct sharp claw-like titillae at distal end.**

Larva: Taylor (1969) found that 7 larvae were 315–400 μ in length with spears 13 or 14 μ long. Tail tapering to a bluntly rounded terminus; hyaline portion much shorter than in female. Genital primordium near mid-body.

Note: Cobb (1893) published the descriptions of *Tylenchus granulosus* n. sp. and *Tylenchus similis* n. sp. from diseased banana plant material sent to him in New South Wales from Fiji in July, 1891. *T. granulosus* is the female and *T. similis* the male of the same species, *T. granulosus* having page priority over *T. similis*. To preserve the well known name *similis* for this widely distributed economic pest, Sher (1968) proposed its retention, regarding *T. granulosus* as a senior synonym.

TYPE HOST AND LOCALITY Soil about diseased banana plants, possibly *Musa* (AAA Group, Cavendish Subgroup) Dwarf Cavendish (see Taylor, 1969); Fiji.

SYSTEMATIC POSITION Tylenchida: Tylenchoidea: Pratylenchidae: Radopholinae: *Radopholus* Thorne, 1949: type-species.

DISTRIBUTION AND HOSTS *R. similis* is widespread in tropical and sub-tropical regions of the world and present in glasshouses in Europe. Although described by Cobb in 1893 from banana in Fiji where disease symptoms had been observed since 1890 (Cobb, 1915), it has only recently been recognized as the major banana root pathogen, probably present in most banana-growing areas (Wehunt & Edwards, 1968, list countries) and of great economic importance in Australia, Central and South America, parts of Africa and the Pacific and Caribbean Islands. It causes what is variously called root rot, blackhead, blackhead toppling disease, toppling disease and decline. Many banana and plantain cultivars are attacked (see Luc & Vilardebó, 1961a; Wehunt *et al.*, 1965) as well as abaca (*Musa textilis*) and other *Musa* species. In Florida, USA, *R. similis* causes spreading decline of citrus, an important disease known since about 1928 but not linked with the nematode until much later (Suit & DuCharme, 1953). Most citrus types act as host to varying degrees. *R. similis* was responsible for yellows disease of black pepper (*Piper nigrum*) which had destroyed 90% of the pepper crop on Bangka, Indonesia, by 1953 (for details see Thorne, 1961). It has been recorded from sugarcane in the USA, Cuba, India, Java, the Philippines and Australia (see Williams, 1969), and found causing decline in avocados in Florida (DuCharme & Suit, 1953) and tea in Ceylon (Sivapalan, 1968). Other hosts include: coffee, maize, vegetables, ornamentals, trees, grasses and weeds. Poucher *et al.* (1967) list 244 hosts and 40 non-hosts. Edwards & Wehunt (1971) give a host list for Central America.

Spread of *R. similis* in soil through root contact, or near contact, is estimated at 10–20 ft per year in Central America (Stover, 1972) and 50 ft per year in Florida (DuCharme, 1968). However, the wide distribution of *R. similis* is due to the transference of infected plant material (especially banana sets) from country to country. The Australian banana industry for instance was established from infected Fijian sets imported between 1890–1920 (Blake, 1972). Ornamentals from the USA transferred the nematode to France (Scotto la Massèse, 1967). For detailed reviews of *R. similis* on banana see Wehunt & Edwards (1968), Blake (1969, 1972) and Stover (1972); and on citrus, DuCharme (1968, 1969) and Cohn (1972).

BIOLOGY AND LIFE-HISTORY *R. similis* is a migratory endoparasite spending its adult vermiform life in the root, but capable of emerging in adverse conditions. The life cycle of a banana population studied on *Tephrosia candida* took 20–25 days at 24–32°C (Loos, 1962). In citrus the life-cycle was completed in 18–20 days at 24–27°C (DuCharme & Price, 1966). All larval stages and females are infective. Fertilization is usual but parthenogenesis does occur. For variation in progenies of a single female, embryology and post-embryological development see van Weerdt (1958, 1960). At least 2 biotypes of *R. similis*, a "banana race" attacking banana but not citrus, and a "citrus race" pathogenic to both were demonstrated by DuCharme & Birchfield (1956). No morphological differences were found; this has been confirmed by van Weerdt (1958) and Sher (1968). The citrus race is at present confined to Florida where it also has many non-citrus hosts; the banana race has a more limited host range. However, neither host range has been fully determined and the existence of other biotypes is strongly indicated (DuCharme & Birchfield, 1956; Edwards & Wehunt, 1971). For fuller aspects of biology see reviews under Distribution and Hosts.

HOST-PARASITE RELATIONSHIPS The most obvious symptom of attack on banana is the toppling over of plants especially those bearing fruit. Other symptoms include lack of vigour, premature defoliation and reduction in bunch weight, size and number of leaves. The histopathology of banana roots attacked by *R. similis* was studied by Blake (1961, 1966). On entering the root the nematodes occupy an intercellular position in the cortical parenchyma where they feed on the cytoplasm of nearby cells, so destroying them and causing cavities to develop. These cavities coalesce and are continually enlarged by the nematodes feeding and tunnelling laterally and towards the endodermis, producing the characteristic reddish brown lesions throughout the cortex (Fig. 2). 3–4 weeks after infection, when extensive cavities have formed, one or more deep cracks with raised margins appear on the root surface. The endodermis seems to act as a barrier to nematodes; the stele is not invaded. Fungal invasion of the lesions causes necrosis which penetrates the stele resulting in root atrophy. Eventually the root system may be reduced to a few short stubs. In a large rhizome roots may pass through 3–4 inches of cortex before emerging. *R. similis* migrates from the root to the rhizome cortex causing diffuse black lesions surrounding embedded roots (Loos & Loos, 1960b). Paring the rhizome clearly reveals these lesions (Fig. 3), which Ashby (1915) termed blackhead disease. Occasionally *R. similis* attacks leaf bases near the soil line (Wehunt & Edwards, 1968).

Declining citrus trees have fewer and smaller leaves and more dead twigs than healthy trees. There is a tendency to wilt, seasonal growth flushes are weak, fruit set sparse and yields low, but death is not usual. Trees show the same degree of decline in an area which increases in size each year (Poucher *et al.*, 1967). DuCharme (1959) studied histopathology of affected citrus. As in banana, *R. similis* feeds on and burrows in the cortex forming extensive cavities. In citrus, unlike banana however, nematodes enter the stele via endodermal passage cells and accumulate in the phloem and cambium which in time may be completely destroyed leaving a nematode-filled cavity separating the remains of the stele from the cortex. Cell reaction includes wound gum deposition in the cortex, hyperplasia and tumour formation in the pericycle. External cracks appear over the lesion 3 weeks after infection. DuCharme & Price (1966) isolated up to 739 individuals from one lesion and estimated that a 20-year-old tree supported 100,000–800,000 nematodes. Avocado trees show similar decline symptoms and root lesions to citrus (DuCharme & Suit, 1953). Yellows disease of pepper is characterised by the gradual abnormal yellowing of foliage, especially young growth. Plant growth ceases, there is leaf drop, die back, and eventual death. When first symptoms show, few plants may be affected but the disease spreads, eventually destroying the entire crop (Christie, 1957). Attack results in destruction of small feeder roots followed by extensive necrosis of the larger laterals.

Data on crop losses are given by Wehunt & Edwards (1968) for Central America. Uninfested banana plots yielded up to 17,000 lb/acre per year more fruit than infested. In Surinam, Maas (1969) found that on estates where 100% of bananas were infected yield was 30 tons/ha per year compared with 73 tons/ha per year where infection was 3%. Spreading decline reduces yield by 40–70% in oranges and 50–80% in grapefruit. After a 10-year comparison between 2 citrus groves, one healthy, the other infected, crop production was 535 boxes/acre and 25 boxes/acre respectively (Poucher *et al.*, 1967).

ASSOCIATIONS WITH OTHER PATHOGENS Incidence of Panama wilt caused by *Fusarium oxysporum* f. *cubense* doubled in Gros Michel bananas when *R. similis* was added to the soil (Newhall, 1958) and wilt symptoms appeared faster on *R. similis*-infected bananas (Loos, 1959). *F. oxysporum* alone is unable to invade intact banana root cells but colonized cortical parenchyma cells wounded mechanically or by the nematode. In plants inoculated with both *F. oxysporum* and *R. similis* the fungus was able to grow through the endodermis, causing necrosis of the stele and eventual atrophy of the whole root distal to the point of stelar invasion (Blake, 1966). The syndrome of spreading decline of citrus involves an interaction of *R. similis* with *Fusarium*, *Sclerotium* and other soil-inhabiting organisms (DuCharme, 1968).

CONTROL Loos & Loos (1960a) described a method of paring banana sets involving removal of all discol tissue. Using paring and a DBCP mixture as a dip, infection was reduced from 89% to 1% after 8 months. Similar techniques have been used at Kew (Peachey & Hooper, 1963) and in the Windward Islands (Edmunds, 1969). However phytotoxicity may be encountered, especially with pared sets (see Vilardebó & Robin, 1969), or nematicide penetration prove insufficient for complete kill (Blake, 1961). Hot water treatment of sets has become standard practice in Central America and Australia. Immersion of small sets at 55°C for 20 min (25 min if planting follows immediately) is effective (Blake, 1961, 1963). Paring increases the likelihood of deinfestation. Flood fallow for 5–6 months eliminated *R. similis* from fields in Honduras and Panama (Loos, 1961). Flood fallowing is practised in Surinam where bare fallowing is unsuitable (Maas, 1969). A 2-year fallow period with a cover crop (*Panicum maximum* var. *trichoglume*) was recommended in Queensland, Australia (Colbran, 1964) while in New South Wales *R. similis* was not detected in soils 6 months after infested stools were mechanically uprooted (Blake, 1969). Rotations with sugarcane have met with some success (Loos, 1961). Soil fumigation with DD, EDB and DBCP was investigated by Luc & Vilardebó (1961b). DBCP was most satisfactory, the authors recommending applications of 40 litres/ha at planting during May–June, 25 litres/ha in October and 15 in March each following year. This programme gave excellent control and has been widely adopted in the Ivory Coast. DBCP experiments in Central America (Wehunt & Edwards, 1968) gave yield increases of 14–86%.

Because spreading decline of citrus is confined to Florida a state-run control programme is possible (see Poucher *et al.*, 1967). *R. similis* has been virtually eliminated from glasshouses and nurseries in California by strict measures including quarantine of rooted plants from Florida, Hawaii and Puerto Rico (French, 1970). For resistant or tolerant citrus rootstocks see Poucher *et al.* (1967). For resistance in *Musa* see Wehunt *et al.* (1965).

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