COLLAR AND BRANCH CANKER IN YOUNG TEA CAUSED BY *PHOMOPSIS THEAE* PETCH

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Early History

In 1927 Gadd observed a die-back of young shoots on a field that was recovering from pruning and tentatively identified the pathogen as *Leptothyrium theae*, a fungus that Petch (1925) had found earlier on tea stems. Petch apparently did not consider the fungus to be of any great importance. Gadd reported the disease again in 1929, and in 1935 he was able to prove by inoculation experiments that the fungus responsible for the disease was indeed *L. theae*. According to him, the fungus gains entry into green shoots before they have grown to any appreciable length. The fungus then grows towards the base of an infected shoot, killing the cortex as it advances, and thereafter into the parent branch. The shoot is ringed at the base and death follows.

In a few instances he also observed that the fungus had killed young bushes by encircling and ring-barking the main stem. As *L. theae* can infect green stems only, he felt that the main stem must have been infected when green, or via a young branch arising from it.

Recent Developments

Since these early observations very little was known or heard about the disease until 1957, when an estate in Dimbula reported the death of a few bushes in its multiplication plot. Webster (1957) who examined specimens concluded that death was due to *L. theae*. A later examination of the same plot showed that about 300 bushes were affected and on Webster's recommendation these were grubbed and destroyed as a precaution against further spread. Similarly, in 1959, a row of 10 bushes of clone Brunswick EM 9 had to be uprooted from a multiplication plot at St Coombs on account of severe collar and branch cankers (F. H. Kehl, 1959).

The first record of the disease in a new clearing came from Dambatenne Estate, Haputale (Power, 1960). This estate reported the death of approximately 100 bushes in a clearing of 22 acres in its fifth year of planting. It is not certain, however, whether all these deaths were due to Collar Canker.

The following year at Nayabedde Estate, Bandarawela, several bushes of clone TRI 2024 and NB 3 were found to be affected by the disease and about 10 bushes died (Burnet, 1961). These deaths occurred in a 5-year-old clearing of about 3½ acres.

By this time reports began to reach us of similar trouble on other estates in the neighbourhood. Most of these were concerned with death of young plants in new clearings. In the meantime, Venkata Ram (1959, 1960) in South India made a significant observation. He observed that *L. theae* can attack tea not only in its first year from pruning but also in its second and third years. He later noticed that the same fungus can cause die-back in young tea as well.

Mulder, who was Pathologist at this time, attributed all damage in new clearings to *L. theae*, and put forward certain hypotheses to explain its sudden outbreak in the Uva District. He also suggested certain tentative measures to prevent further occurrences of the disease (Mulder, 1962).

Figure 1.—Two young bushes of clone KW 16/3 showing yellow foliage and dying due to Collar Canker caused by *Phomopsis theae*.

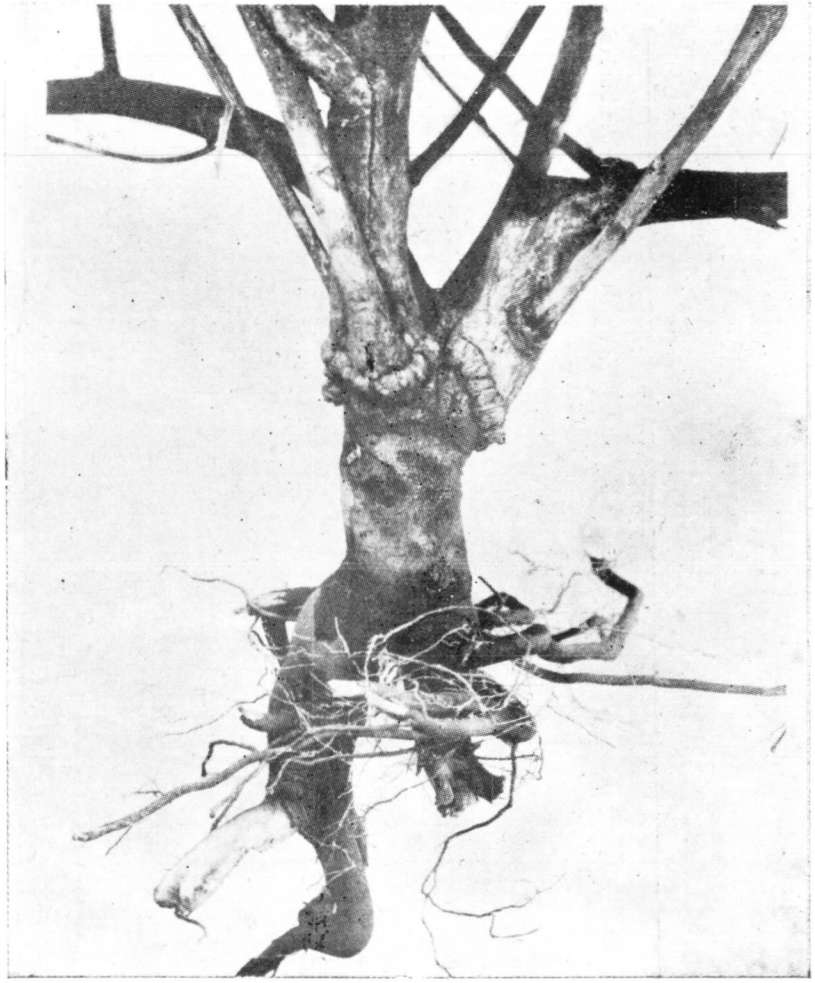

Figure 2 —A young bush of clone Brunswick EM 9 girdled at the collar.

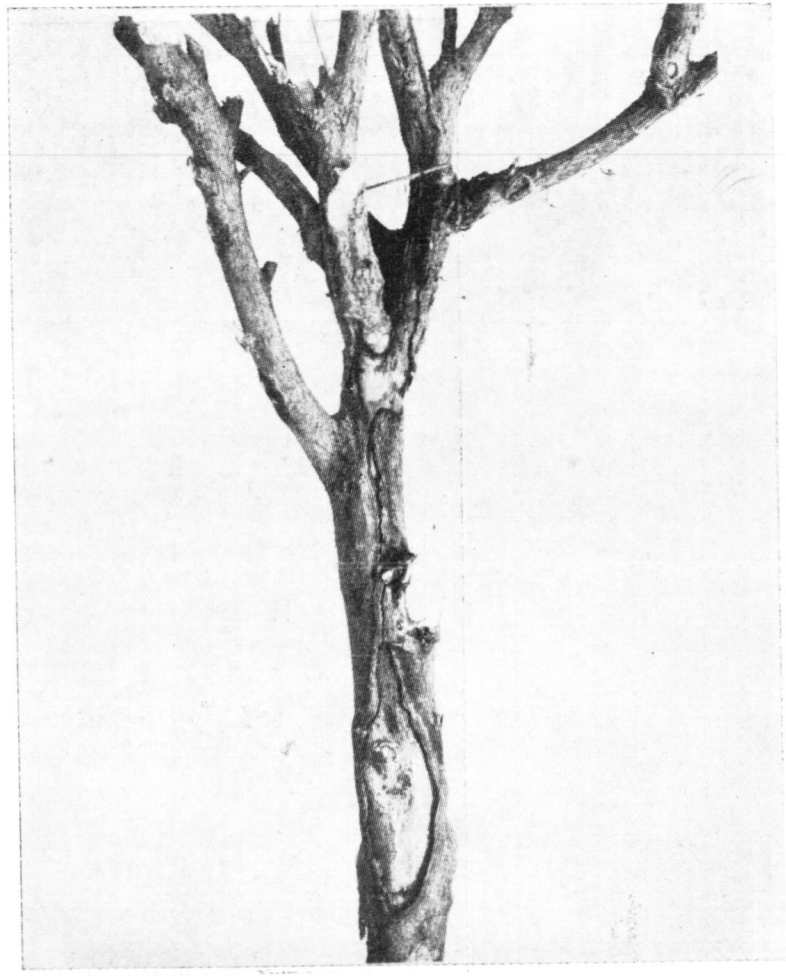

Figure 3.—A 2 year old plant with stem cankers.

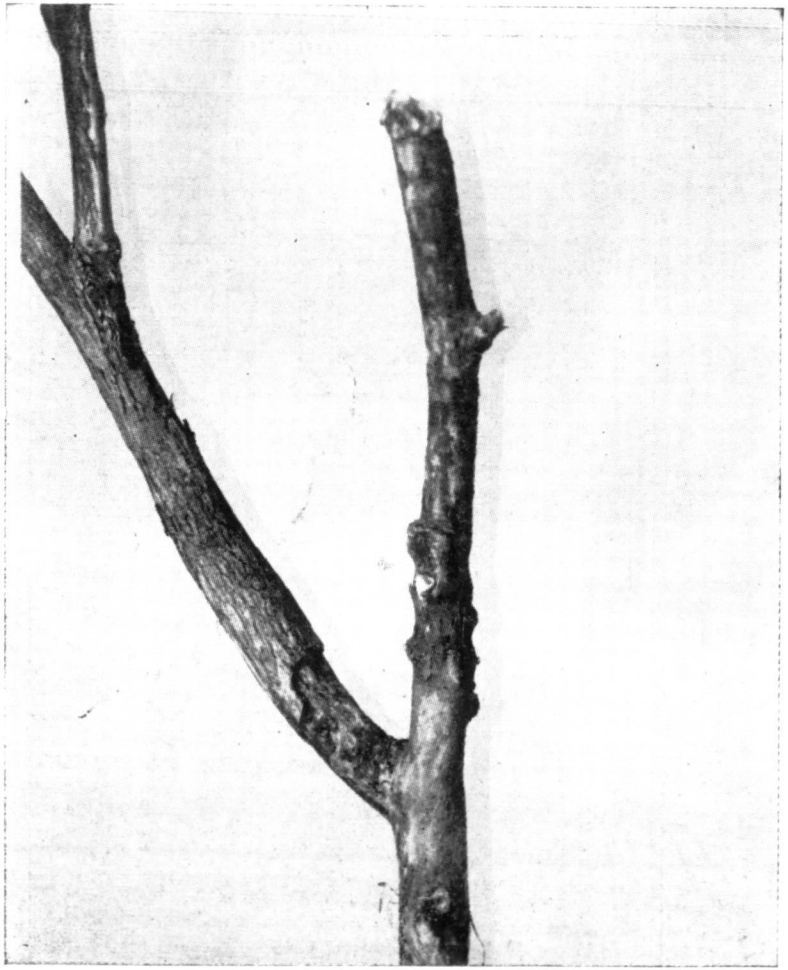

Figure 4.—A branch ringed at the base.

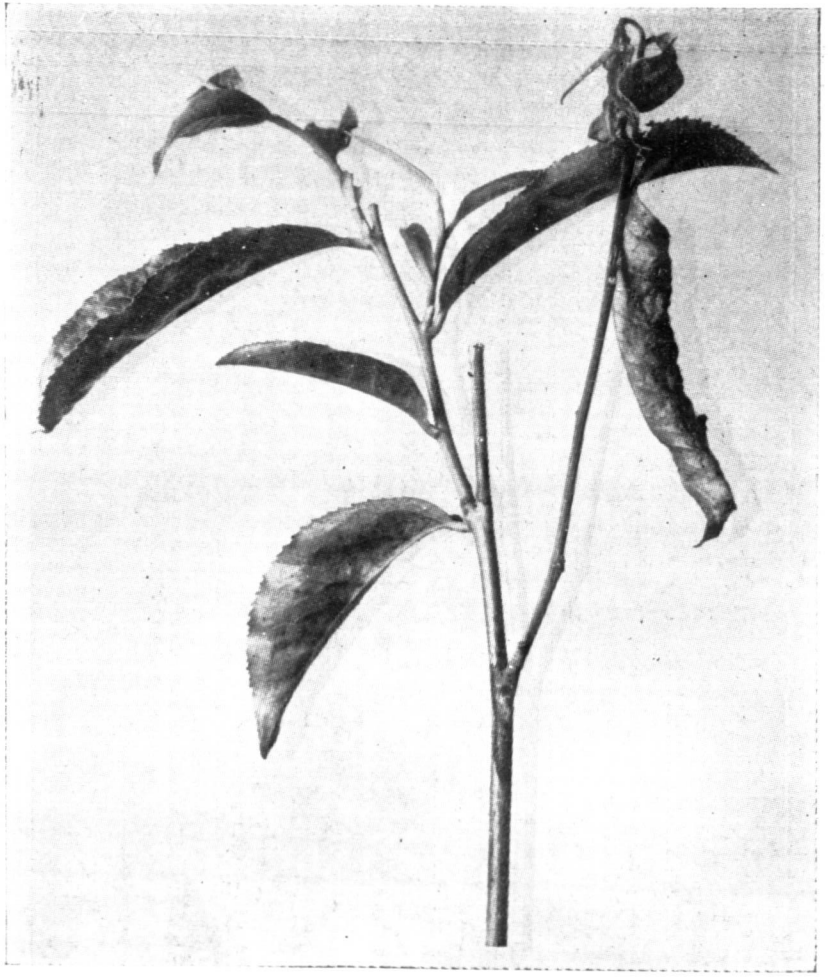

Figure 5.—A young shoot dying as a result of infection arising at the leaf-scar.

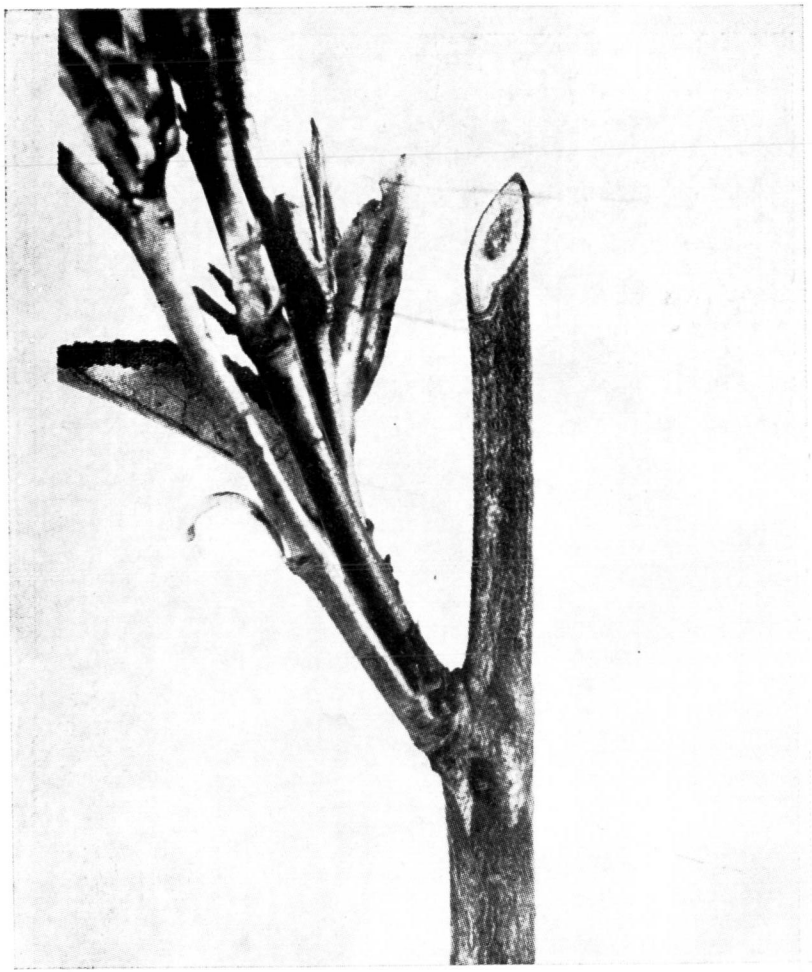

Figure 6.—An example of infection originating at prune cut. Note end of branch dying

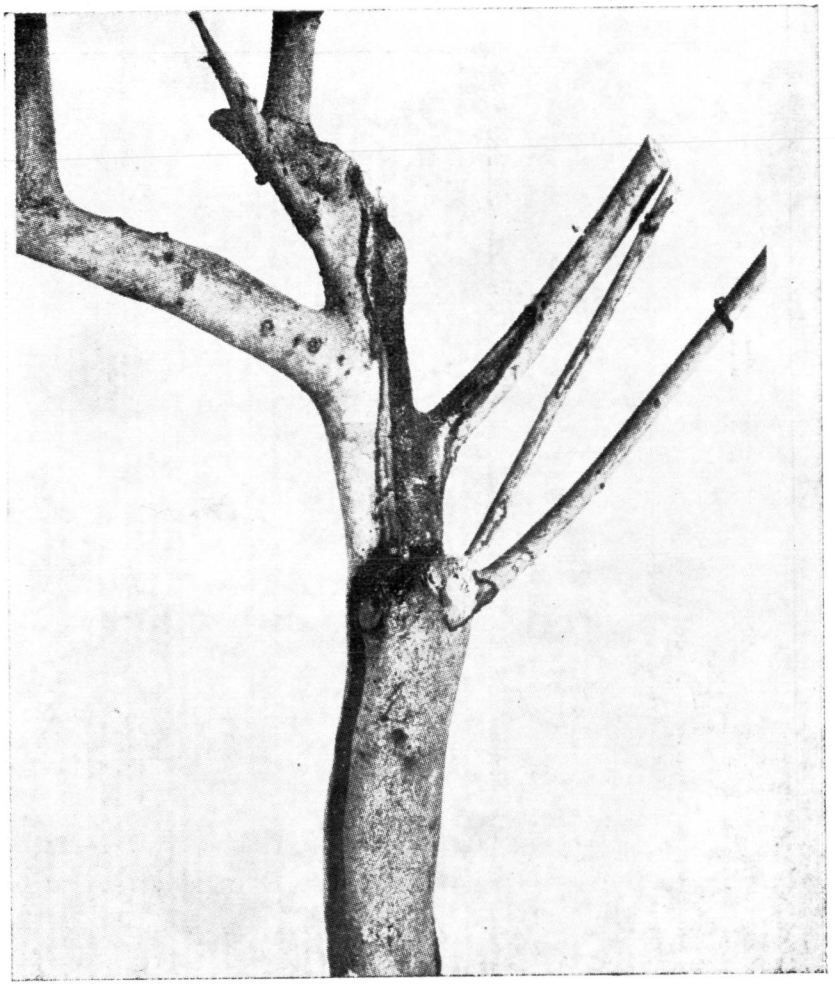

Figure 7.—Branch cankers on frame resulting from sun scorch.

The writer began detailed investigations into the disease in late 1962, and this paper summarizes the results of his experiments and observations.

Symptoms

The disease shows plainly from a distance, the first obvious symptoms of attack being the yellow or brown leaves of a killed branch or an entire bush standing out in contrast to the green foliage (Fig 1). A close examination of such a bush will show a canker or lesion located at the collar of a dead bush or at the base of a killed branch. Cankers are developed by gradual killing of the bark at or around the site of infection. In the early stages they are not so apparent, but old cankers can be easily recognised by the raised margin due to development of callus. The diseased area may be regular or very irregular in shape, often sunken and grey to black in colour. On scraping with a knife, the underlying dead tissues can be seen easily. In cases where the collar is girdled completely, a thick ridge of callus forms at the upper margin of the canker (Fig 2).

In young tea cankers can arise on any part of the bush and have been seen frequently on the stem, branches, at the crotch, base of twigs, prune cuts and leaf-scars, but prove fatal only when the collar is ringed (Fig 3-7). Sometimes small twigs and branches are killed without a definite canker being formed; this is most common in old tea recovering from pruning.

Field observations indicate that cankers are invariably associated with:

(*a*) wounds and mechanical damage, mainly in young plants;

(*b*) at nodes on young branches and twigs in both young and old plants; and

(*c*) sun scorch and purne cuts especially in old tea.

The implications of these observations will be discussed later.

Damage

This is difficult to evaluate accurately. The disease is primarily one of young plantations and damage consists of death of plants between 2 and 8 years old. In older plantations damage due to branch die-back is quite frequent, but seldom are bushes killed. Branch cankers are not serious unless so many branches are cankered that the frame is much reduced. So far the disease has not been observed in the nursery.

The fungus kills the bark up to the cambium and the sapwood is also invaded. If a stem is girdled, it dies, but if not, the formation of callus tissue begins and further development of canker is arrested. If the canker is small, it may be entirely grown over by callus.

The disease is sporadic in appearance and varies in intensity from year to year and between localities. In young clearings the death of a few to a dozen or more bushes is not uncommon, but much more serious are instances where entire clearings had to be abandoned as replacement of losses was considered uneconomic. The following examples illustrate the above observations clearly.

In a young clearing on St Coombs, 31 clones and a batch of seedlings were planted in 1961 in a randomized block design. There are about 5000 plants in this

experiment. The clearing is now in its fourth-year, and so far only 6 bushes have been lost, and only 2 clones affected. There are no obvious signs of the disease on the remaining plants, even on those of the effected clones. This is an example of a minor attack. Several recent reports from estates are of this kind.

The disease was more serious on Nayabedde Estate. There in 1961 in a 6-year-old clearing of clone 2024 (1½ acres; elevation 6,100 ft) only two bushes died of Collar Canker after exceptionally hot and dry weather. The next year, however, when the tea was pruned about 50 plants were found to be badly attacked and 10 did not recover from pruning. The remaining bushes continued to grow though the frames were severely cankered. A similar clearing on a lower elevation (4,600 ft) showed no signs of the disease.

A case similar to Nayabedde was Dambatenne, where, as mentioned earlier, about 100 bushes died in a clearing of 22 acres.

A more disturbing case was seen recently on an estate in the Maturata District. On this estate a 4-year-old 2024 clearing of 2 acres had to be abandoned in July this year due to ravages by Collar and Branch Canker. The writer visited the clearing in April, when he found that 1½ acres of it had already been uprooted and planted with Guatemala grass and marigold on the assumption that the damage was due to nematodes. The remaining half acre had about 60 per cent vacancies, and 100 bushes were selected at random from this area and examined for cankers. Results showed that 77 bushes had branch or stem cankers, 43 Collar Canker, and 40 both Collar and Branch Cankers. A subsequent examination in July showed that many of the bushes with Collar Canker were dead. It thus became clear that the large number of casualties in this field were probably the result of Collar Canker. This is by far the worst attack that has come to our notice.

There are indications from field observations that certain clones are more susceptible than others to the disease. The clones that have been found susceptible so far are given below for the information of estates:

> TRI 2024 and 2025, Kenilworth 16/3, Neluwa 3/1, Brunswick EM9, Liddesdale 23, 275 and 502, Eskdale 9/5 and 2/12, Tangakelle 2, Downside 686 and 740, Tillicoultry 10, UD 30, Dambatenne 434, Nayabedde 3, Drayton 1, Chapelton 171 and Craighead 13.

Occurrence and Distribution

The disease is now known to occur in at least 7 major planting districts of the island, *viz*, Hewaheta, Udapussellawa, Welimada, Haputale, Badulla, Madulsima and Dimbula. The last-named is an exception in that it is the only district outside the North East Monsoon Zone. The author has identified the disease with certainty on 20 estates and 18 of these fall within the N. E. Monsoon Zone; the two remaining estates, namely St Coombs and Uda Radella, are in the Dimbula District.

Elevation also appears to be an important factor. Eighteen of the 20 estates are at an elevation of 4000 ft or above; the other two are at 3000 and 3500 ft respectively.

The Casual Agency

Isolations.—Samples of the various types of infection were collected from different estates and isolations were made to determine the causal agency. The bark was shaved off from cankers and pieces of infected wood taken from the advancing edge were plated on potato-dextrose-agar (PDA). Plates were incubated for 2-3 weeks at 25°C.

Phomopsis theae was invariably isolated. The cultures were often pure, but even when other fungi grew from the infected tissue, *P. theae* was also present. Frequent contaminants were *Pestalozzia theae*, the fungus which causes Grey Blight of tea, and *Botryodiplodia theobromae*, a very common tropical fungus that attacks only weak or moribund tissues.

Leptothyrium theae or *Macrophoma theicola*, the two fungi known to cause stem cankers in tea, were never isolated. Identity of *P. theae* was confirmed by Sutton (1963) of the Commonwealth Mycological Institute.

Preliminary Inoculation Experiments

1. *Greenhouse.*—These were carried out in the greenhouse using 2-year-old potted plants of clone TRI 2024. The plants were placed in a humid chamber for 48 hours, and then inoculated with 10-day-old pure culture on PDA. Inoculations were made through Λ — shaped cuts on the stem, and cuts were bound immediately with polythene tape. The plants were again held at 100% humidity for 48 hours before transferring to greenhouse. Four isolates were inoculated separately in December 1963. The resulting cankers were measured the following May, when callussing began at the edges of lesions and cankers ceased growth (Table 1).

TABLE I.—*Results of inoculations with isolates of Phomopsis theae on tea — (Clone TRI* 2024)

Isolate	No of successful inoculations	Size of lesions (cm)
LT 1	2/5	1.5–2.0
LT 3	3/5	1.0–4.0
LT 4	3/5	1.5–2.0
LT 5	3/5	1.5–7.0
Control	0/12	

All control inoculations remained uninfected and all 4 strains were re-isolated from cankers, thus fulfilling Koch's postulates.

2. *Field.*—Inoculations were made on the collar of 2-year-old 2024 plants on a clearing at Nayabedde Estate. Two isolates were used and 7 plants were inoculated; 3 plants were only wounded and served as controls. Examination 2 months later showed that cankers had formed on all inoculated plants, the largest measuring 14.5 cm (Fig 8). None of the cankers, however, girdled the stem completely and no plants were, therefore, killed. Both strains were recovered by re-isolation.

Further inoculation experiments carried out in the greenhouse to elicit information on the mode of entry of the fungus into the stem showed that *P. theae* is essentially a wound parasite. No infection occurred when plants were inoculated without wounding, but cankers developed invariably on young bushes that were inoculated via prune cuts, centering cuts and leaf-scars.

Comparative Pathogenicity Tests

Further proof of pathogenicity and virulence of *P. theae* was obtained from comparative pathogenicity tests conducted in the field using *P. theae*, *L. theae*, *M. theicola* and *B. theobromae*. Cultures of *L. theae* and *M. theicola* were supplied by Dr Venkata Ram of the Tea Research Station of South India. Two experiments were carried out, one at St Coombs and the other at Maturata.

St Coombs Experiment—This was conducted on a clearing of clone 2024 in its 2nd year. Inoculations were made at the collar, on branches, and on green shoots ; those at the collar were performed by removing a disk of bark using a cork-borer and placing on the exposed wood a disk of agar containing the fungus. Plants were inoculated in March 1964 and resulting cankers measured in June (Table 2).

TABLE 2.—*Results of field inoculations with various fungi on tea—(Clone TRI 2024—St Coombs). Number of successful inoculations and mean length of cankers (cm).*

Fungus	Site of inoculation					
	Collar		Branches		Green Shoots	
	No	Length	No	Length	No	Length
Phomopsis theae	8/10	1.9	20/20	1.8	30/30	1.9
Leptothyrium theae	0/10	—	4/20	1.1	0/30	—
Macrophoma theicola	0/10	—	1/20	1.3	0/30	—
Botryodiplodia theobromae	0/10	—	0/20	1.5	0/30	—
Control	0/10	—	0/20	—	0/30	—

Maturata Experiment—In this experiment only *P. theae* and *L. theae* were compared. Inoculations were carried out on a young clearing (clone 2024 ; 4-year-old) that showed severe Collar and Branch Canker. By careful examination plants free of cankers were selected and inoculated as before. Each plant was inoculated once on the collar and thrice on the branches, and 10 plants were used for each fungus. Six plants served as controls. Inoculations were made in May 1964 and cankers were recorded in July (Table 3).

TABLE 3.—*Results of inoculations with P. theae and L. theae on tea (Clone 2024—Maturata). Number of successful inoculations and mean length of cankers (cm).*

Fungus	Site of inoculations			
	Collar		Branches	
	No	Length	No	Length
Phomopsis theae	7/10	17.4	29/30	19.5
Leptothyrium theae	0/10	—	0/30	—
Control	0/6	—	0/18	—

Canker development was rapid and extensive in this experiment. Unfortunately observations could not be continued for more than 2 months as the estate concerned was in haste to uproot the tea.

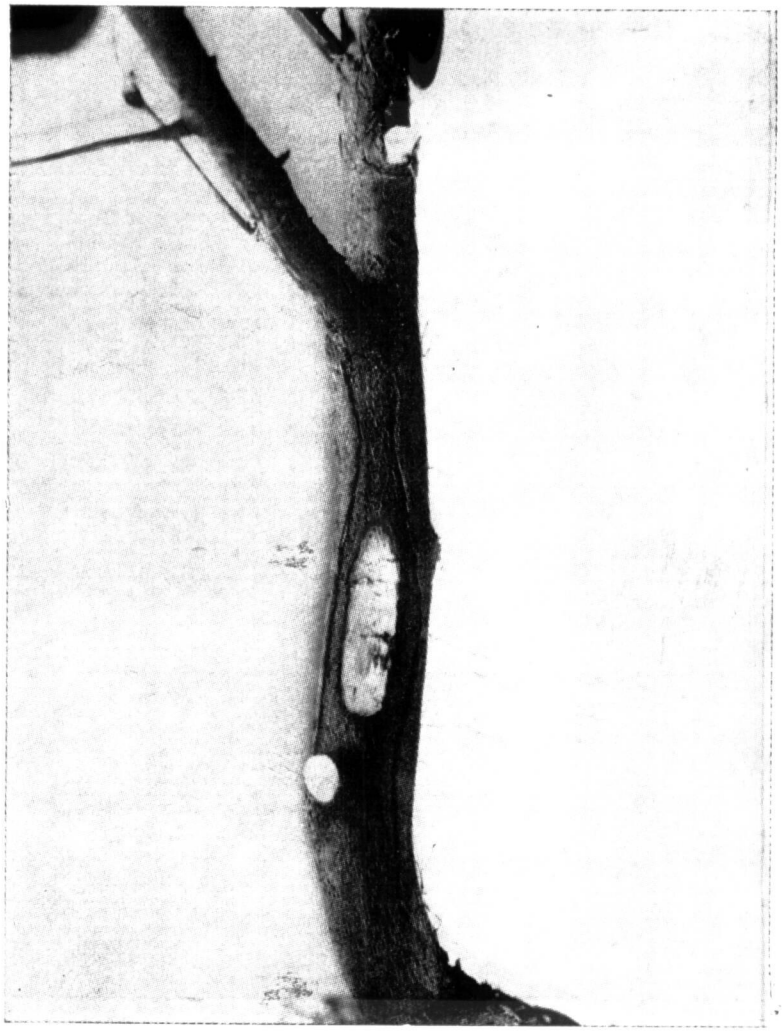

Figure 8.—Piece of an infected branch showing numerous pycnidia and an extruded 'spore horn' or 'spore tendril'.

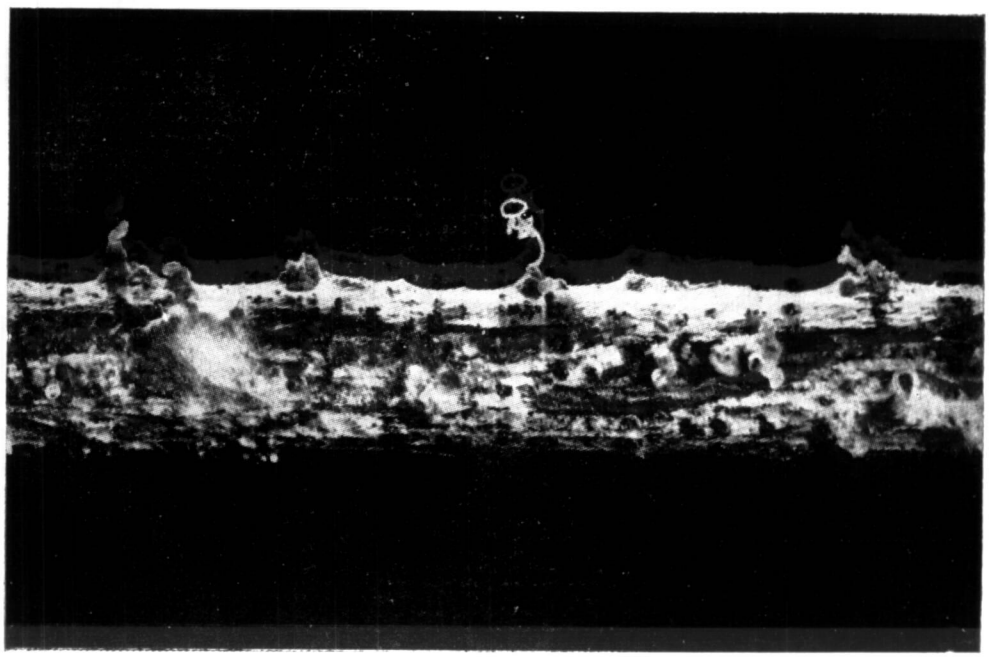

Figure 9.—A 2-year old clonal plant showing a canker resulting from artificial inoculation.

It is clear from the two experiments that of the four fungi tested *P. theae* is easily the most pathogenic, as indicated by the percentage of successful inoculations and the size of the lesions obtained.

The Fungus

P. theae belongs to the group *Fungi Imperfecti* and is closely allied to *L. theae.* It differs from the latter in that it produces two kinds of spores or conidia, namely the 'A' spores and the 'B' spores. The A-spores are spindle-shaped and resemble closely the spores of *L. theae.* The B-spores are needle-shaped and slightly curved.

Fructifications develop abundantly on the cankered bark and break through as small raised pustules somewhat pimple-like and about the size of a pin-head. From these pycnidia sticky pycnidiospores ooze out during moist weather in yellowish or amber-coloured, long, coiled spore horns or tendrils (Fig 9). When there is excess moisture, as in culture flasks, the spore masses are globular or spread out. Pycnidia apparently can develop at any time of the year and usually appear a month or so after infection, both on naturally infected wood and in culture.

Infection can occur on stems of any size by means of spores, which being sticky are most probably dispersed by rain. When dry spores can also be dispersed by wind. Since the fungus cannot penetrate undamaged bark entry is invariably through wounds. So far no host other than tea is known. The fungus probably grows as a saprophyte on all kinds of dead wood.

Discussion

The experiments and observations described above have shown that Collar and Branch Canker now prevalent on estates in the high-country is caused by *Phomopsis theae* and not by *Leptothyrium theae.* The writer has never been able to isolate *L. theae* or even *Macrophoma theicola* from diseased specimens. *M. theicola* is essentially a fungus of the low-country (Petch, 1923.) and it is, therefore, not surprising that it did not appear in our isolations. *L. theae,* on the other hand, has so far been considered as a fungus mainly of the high-country, so that its absence indicates that it is less prevalent now or that its earlier identification was incorrect. As *L. theae* is still common in India, it unlikely that it did not occur in Ceylon earlier.

The evidence presented also shows that of the three fungi in question, *P. theae* is the most virulent under up-country conditions. Inoculations with *P. theae* were on the whole very successful in that they produced cankers similar to those found in nature. However, the failure to kill bushes completely indicates that suitable environmental conditions were not obtained in the experiments, except perhaps at Maturata where large cankers developed rapidly.

Inoculations have demonstrated that cankers can arise only through injuries. Wounds can be caused by normal horticultural operations like pruning, weeding and cultivation; by agents like wind, sun and insects; and by falling rocks etc. The initial entry points of cankers can be traced clearly only in young infections, especially those originating from leaf-scars, and centering and prune cuts. However, it is very likely that at least some collar cankers originate from wounds caused by weeding scrapers and falling rocks, and most cankers on branches from prune cuts, natural cracks, breakages due to wind and man, and sun scorch. Many of the cankers on green shoots and twigs appear to arise from leaf-scars. It is not known whether leaf-scars resulting from normal leaf fall are vulnerable. In young tea, further avenues for infection are provided by centering cuts and other wounds created during operations carried out to induce bush development.

The extent to which each of these various types of wounds is important is still not known. It has been very difficult to study this problem at St Coombs because of the relatively low incidence of the disease there.

The disease appears to be mainly confined to high elevations and the NE Monsoon Zone in particular. The climate of the NE Monsoon Zone varies between extreme droughts to heavy rainfall and this seems to favour the disease in some manner. A careful examination of field records show that some of the most serious attacks by *P. theae* seem to have occurred after exceptionally dry weather. This has happened at Dambatenne (Power, 1959) and Nayabedde (Burnet, 1961) in recent years. Another significant observation is the association of the disease with poor soil conditions. It was most evident at Maturata, where the disease caused an entire clearing to be abandoned. Here the soil was very sandy and gravelly. Similarly, the soil on some of the badly affected fields at Nayabedde is very quartzy (Burnet, 1961). These observations clearly suggest a relationship between the disease, drought and poor soil. This is also supported by the results of the inoculation experiments where large cankers resulted only from inoculations made during severe drought. A similar relationship has also been observed in the case of *M. theicola*. According to Hainsworth (1952) some of the worst attacks by *M. theicola* occur on tea growing in sandy soil and under drought conditions.

Field observations clearly indicate a marked variation in clonal susceptibility. The disease has been recorded most frequently on clone TRI 2024 on several estates, but this may be due to the fact that it is used very widely in the Tea Replanting Subsidy Scheme. At St Coombs in a 3-year-old clearing only 2 out of 32 clones have so far been found to be affected by the disease. Curiously enough, clone 2024 is free of the disease in that field. Similar instances have also been noted on other estates.

There is some indication that clones (eg. 2024 and NB 3) which tend to become unproductive during drought are very susceptible to the disease. It is possible that during very dry weather, when plants of these clones are almost dormant, the tissues become inactive and more vulnerable to infection. Moreover, wounds formed at this time would be slow to heal and remain open to infection for a relatively longer period. It is significant to remember here that many canker diseases of temperate plants (eg. Bacterial Canker of cherry and plum) also occur during the dormant season.

A further possibility is that during very hot weather damage to the collar can occur as a result of overheating of the soil at this point. Some of the collar cankers observed in the field probably arise in this way.

With regard to control of the disease, only certain tentative measures can be suggested at this stage. Every effort should be made to minimize injuries on young tea, and after such operations like centering, bending and pruning, it may be advisable to protect the resulting wounds from infection by spraying. Such treatments should also be carried out at a time when the tea is in a vigorous condition and climate ideal for growth in order to promote rapid healing of the wounds. During drought steps should be taken to conserve soil moisutre by thatching or other measures.

Without further information on the periods most critical for infection it is not possible to formulate any long-term spray schedule. Pending evolution of satisfactory chemical control, the use of resistant clones seems most desirable and may in the end prove the best means of controlling the disease. Clones well-known to be susceptible to this disease should not be planted on estates where the disease is prevalent, particularly if the soil is sandy or gravelly and the area subject to severe droughts. It is suggested that estates should carry out their own selection in view

of the sporadic nature of the disease and the variation in susceptibility of certain clones in different localities. Susceptible clones can be easily eliminated by careful examination of plants in multiplication plots, especially after pruning. Clones showing cankers should be discarded immediately.

Finally, it may be worth mentioning that diseases caused by *Phomopsis* species are by no means rare. Several *Phomopsis* spp. attack forest trees causing cankers and die-back of both conifers and hardwoods like pine, fir, elm and chestnut (Boyce, 1938)

Summary

Collar and Branch Canker of tea earlier attributed to *Leptothyrium theae* is now shown to be caused by *Phomopsis theae*. The disease occurs at high elevations and mainly in the North East Monsoon Zone. Canker attacks are sporadic, and vary in intensity from year to year and between localities.

Field observations also indicate that the disease is serious only in young plantations up to 8 years in age. There is marked variation in clonal susceptibility. Drought and poor soil conditions appear to be predisposing factors.

Canker infections were found to be associated with wounds on the collar and frame, and with the nodes on young branches. Inoculation experiments showed that of the fungi commonly associated with stem canker in tea, namely *P. theae*, *L. theae*, *Macrophoma theicola* and *Botryodiplodia theabromae*, *P. theae* is the most pathogenic in the high-country.

The above observations are discussed in relation to the control of the disease.

Acknowledgements

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