DISEASE IN NON-PRODUCTIVE BUSHES

C. H. GADD, D.Sc.

A statement that all diseases of the tea bush ultimately lead to a diminution of its cropping capacity will probably be accepted by all as axiomatic. In some cases the reduction in crop is very evident as when the affected bushes die, say of a root disease, or when part of the frame is destroyed by wood rot or other stem disease. In others the loss in crop is not so evident; it may be difficult to compute, and is often of little economic importance. Amongst diseases of the latter type I would include 'Grey Blight' which normally attacks old leaf only, but does not cause general defoliation.

Although it is true that all disease results in loss of crop, the converse, that all reduction in crop is the result of disease, is obviously not true. We know that some bushes are inherently poor yielders and that no system of agricultural treatment will make them into good yielders. Similarly there are inherently good yielders, but adverse climatic or agricultural conditions may make them appear relatively poor yielders. The apparent poor yield resulting from such adverse conditions is of a temporary nature as improvement in the conditions results in an increase of yield. If that is not true, many are striving in vain.

A shepherd may know each individual of his flock but a tea planter does not and cannot know each tea bush in his fields. One has yet to hear of a tea planter reflecting on the past glories of a particular bush as he might of an old race horse. An old and weary looking dog may have won many prizes in his day but a decrepit looking bush, exhibiting no apparent disease symptom, is apt to be

regarded as having always been a 'dud.' Now if we had conclusive proof that a high-yielding bush did become permanently low yielding, yet exhibited no symptom of a known disease, we should have to regard the failure to put out flushing shoots as being itself a symptom of disease. In case my meaning may be obscure, I will briefly refer to a disease of plantains known as 'bunchy top' with which many of you are possibly acquainted. The bunchy top tree is readily distinguished from the normal healthy one by the fact that the former has its leaves all bunched together forming a rosette at the top of the stem. Such trees are unproductive and their root suckers display the same type of bunched growth. The bunching of the leaves is due to the failure of the leaf stalks to elongate normally. In other words the failure to grow normally is a symptom of bunchy top. The failure of a tea bush to grow normally would also be a disease symptom, though the change from normality would probably differ from that noted in plantains.

We have recently had good cause for suspicion that certain bushes in up-country districts have become, and others are becoming, permanently unproductive and that these conditions are more widely spread than is generally recognised. The staff at St. Coombs is sometimes criticised for refusing to offer opinion in public till such opinions are verified by experiment. Today I am refuting that criticism, or perhaps merely supplying the exception which proves the rule, because I am going to speak of suspicions and opinions not yet verified. Moreover, I prefer to view a new disease, or suspected disease, from its worst possible aspects until I know its capabilities to do damage. So if I make some rather depressing remarks I hope you will bear in mind that they are opinions, not verified fact, put forward for the purpose of discussion and to direct attention to a condition of sundry tea bushes which I consider to be diseased.

My first acquaintance with the disease was in September, 1935 when a tea bush was sent to the laboratories from the Kandapola district for examination. The bush appeared unthrifty, but no lesions, or dead areas, were visible on the surface of the roots, stems of leaves. In short no disease was apparent to account for the condition of the bush. A more thorough examination, however, disclosed the presence of small dead areas in the cortex close to the cambium. The outer cortex appeared quite healthy. Fig. 1 on Plate 2 shows the kind of symptom that was found. The root figured had an area of cortex cut away, and the centre of the cut is very close to, almost touching, the cambium. The dead areas can be seen as dark coloured spots. That specimen is of a rather advanced case. More usually the dead zones are very small and appear as small yellow or yellow-brown specks.

Figure 2 of Plate 2 is a micro-photograph of a root in which the disease was not so advanced as that illustrated in Fig. 1. The rather prominent dark spots are dead cells. Seen under the microscope they appear yellow-brown, not black as in the photograph. The tissue in which they occur is technically termed the *phloem*. It lies just outside the cambium, which is an actively growing tissue forming wood on one side and phloem on the other. The wood, particularly that just within the ring of cambium, forms the channels through which water and the raw food materials dissolved in it are passed from the root to the leaves, whereas the phloem is the tissue through which the elaborated food materials are moved from the leaves to other parts of the plant. A blockage of the wood vessels leads to a wilt, but a breakdown of the phloem leads to starvation. When a tree is 'ring-barked' a complete ring of phloem is removed and that stops all movement of food to the roots. Ultimately, the roots die of starvation and they cease to send water up to the leaves. Then the whole tree dies.

This short description of the function of the phloem indicates how important that tissue is and suggests that the presence of dead cells in that tissue must to some extent affect the transport of food materials within the plant. Because of this symptom (death in the phloem) this diseased condition has been termed *Phloem necrosis*.

In the field the distribution of the affected bushes is suggestive of a root disease. The diseased bushes tend to occur in patches, not so clearly defined as those caused by Poria, as all the bushes within the patch are not apparently affected. The different recognisable stages suggest a slow deterioration but deaths are very infrequent.

Soon after my first field examination of this disease I was requested to visit other estates in the same district for various reasons and was shown similar bushes. Root examinations disclosed the same symptoms — *Phloem necrosis*. After a certain amount of laboratory investigation and a good deal of discussion with the planters in that district, we came to the conclusion that the disease had been present for some considerable time though the condition had not been recognised as a disease. The bushes had been regarded merely as unproductive bushes of rather low jât character. On several estates for some years past, such bushes had been systematically cut out yet that type had not been eliminated. In fact some superintendents were of the opinion that there were as many then as previously, despite the removals. That observation strongly suggests a steady deterioration.

At that stage the main diagnostic character lay below ground — a *Phloem necrosis* in the roots. Above ground we had no clear-cut

PLATE 1

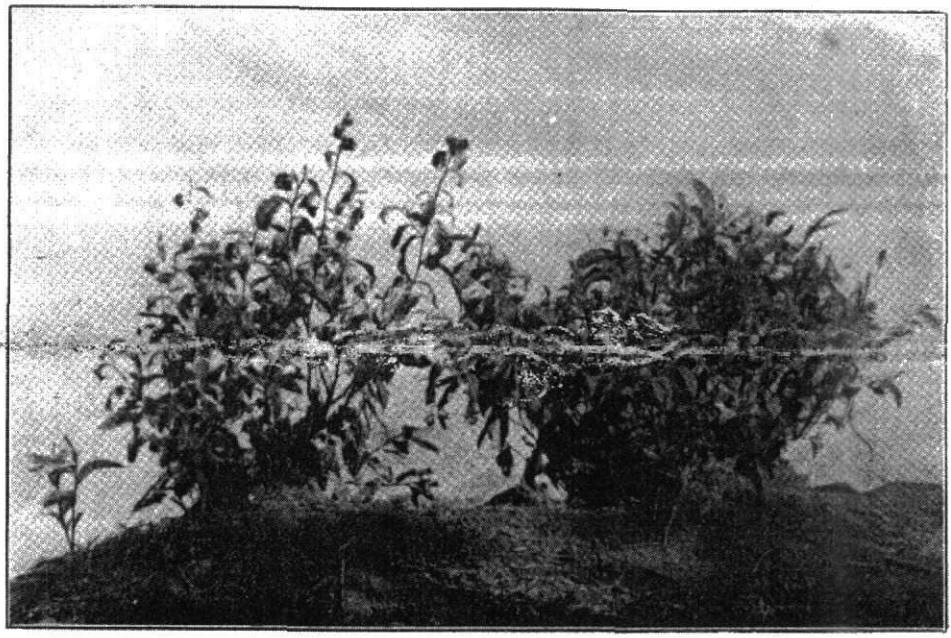

Fig. 1

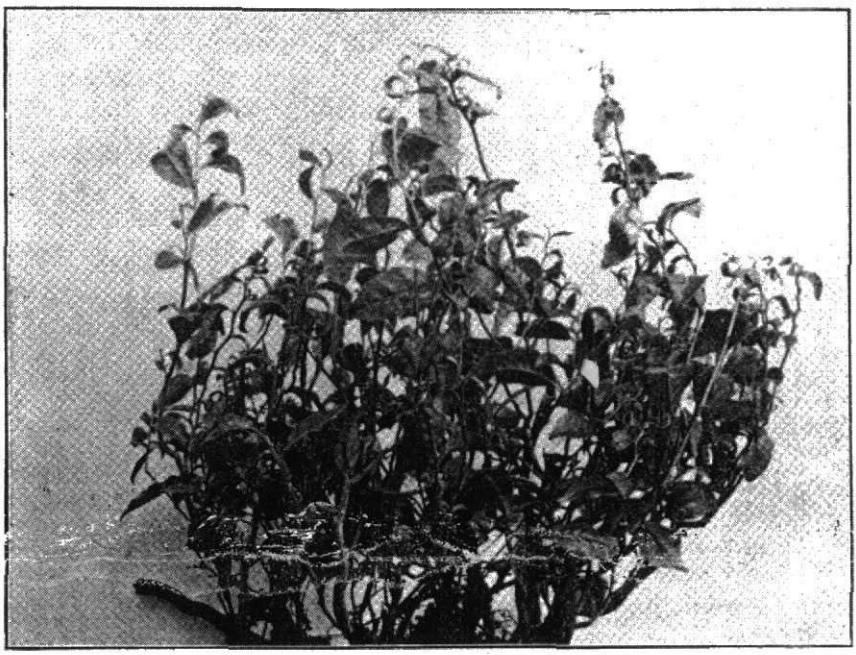

Fig. 2

Fig. 1. The tea bush on the left is severely affected by *Phloem necrosis*; that on the right, slightly. The root sucker on the left is also affected.

Fig. 2. Tea bush affected by *Phloem necrosis*.

PLATE 2

Fig. 1

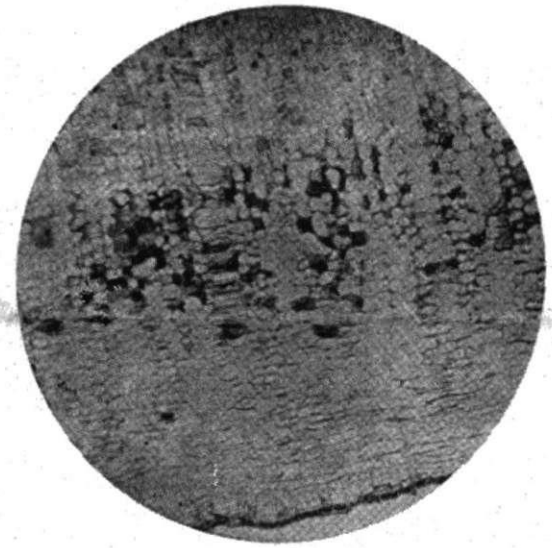

Fig. 2

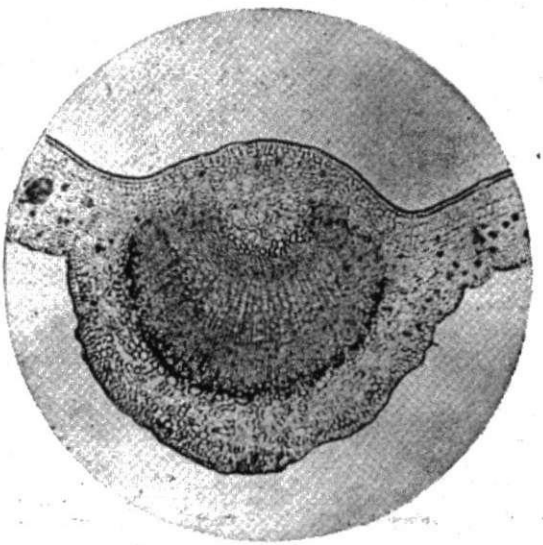

Fig. 3

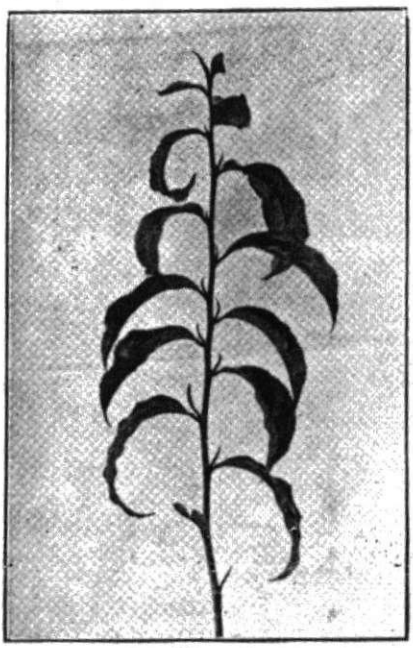

Fig. 4

Fig. 1. Tea root showing necrotic areas near the cambium.

Fig. 2. Transverse section of a necrotic root showing dead cells × 43.

Fig. 3. Transverse section of mid-rib of a necrotic leaf showing necrotic zone × 32.

Fig. 4. Necrotic tea branch showing slight leaf curl.

symptom other than general unthriftiness and a tendency for some leaves to arch backwards. The bushes had what may be termed low jât characters with small leaf, often V-shaped, the mid-rib being the base of the V. Within the bush, branches with larger leaves could sometimes be found; and if such branches were truly representative of the original type of foliage, degeneration of type must have occurred.

The next advance was the discovery of unproductive bushes with markedly curled leaves. The curl was very similar to that which results from aphid attack on young leaves. See Figs. 1 and 2 of Plate 1. A necrosis was found in the petiole and midrib of these leaves and, in some cases, in all the small veins of the leaf also. This above-ground symptom offered a quicker means of identification and has helped us to recognise bushes in an earlier stage of attack. Plate 2 Fig. 4 shows a branch of a bush not yet in the advanced stage of the disease. The leaves are more or less normal in size but have a tendency to arch or curl backwards. Fig. 3 is a section through the midrib. The black semi-circle in the photo is necrotic tissue. Actually, this area is yellow or slightly brown in the specimen and that colour is largely masked by the green of the leaf when fresh.

The symptoms are not very constant. Some bushes have a marked necrosis in the roots and none in the petioles; others exhibit it markedly in the petioles but not in the roots. Some bushes have merely arched leaves and others exhibit a definite curl. These differences naturally raise the question whether we are dealing with one or more diseases. For the present I regard the disease as one, and the variation in symptoms the results of different reaction by the various varieties of tea.

I am demonstrating the symptoms of this disease tomorrow. I need say no more concerning them now, and will pass on to the cause of the disease.

I may as well tell you at once that we do not know for certain what causes the disease. The main possible causes can be grouped under the following heads:—

1. Visible parasites.
2. Environmental conditions, climate and soil.
3. Old age.
4. A virus, which I will define, rather incorrectly, as an invisible parasite.

1. Our first line of investigation was a search for a visible parasite. Frequent microscopic examinations and many attempts to

culture an organism from the diseased tissue have given negative results. This line of work is now being abandoned as profitless and we can say with a fair amount of confidence that the disease is not caused by a visible parasite.

2. The term 'environmental conditions' covers many factors which may be grouped as climate and soil. The incidence in the field does not suggest that climatic conditions are a main cause. It has been suggested to me that the drought of 1934 was an important factor. That may be so if the disease is transmitted by insects and if drought favours the development of that species. In that event drought takes a secondary position, one favourable to the propagation of the disease but not the cause of it. The symptoms are not, of course, drought symptoms pure and simple.

If soil conditions provide the primary cause, it cannot be a question of soil type, water table or general manuring; the evidence is strongly against those possibilities. A more likely guess is that the cause will be found amongst the minor elements such as manganese, boron, chromium, etc. Such elements in minute quantities are essential to plant growth; in the absence of one or other of them plants fail to make normal growth; if one is too abundant, its effect is apt to be toxic.

At various times we have transplanted necrotic bushes from different estates to St. Coombs. Of these 21 are still alive, and still necrotic. Diseased root suckers, which like the transplanted bushes had their roots freed from adhering soil, were planted in pots on St. Coombs, but are also still necrotic. Those plants are now growing in fresh soil and in a different climate, yet have made no improvement These experiments suggest that neither climate nor soil is the cause, otherwise some of the plants would have thrown off the symptom in their new growth. This evidence forms the basis of a statement I made earlier, viz. that affected bushes are permanently unproductive. I incline to the view that the disease is incurable.

3. Old age in plants is rather a big subject to include in a short paper like this. The hypothesis that this disease is little more than old age is one which appeals to several planters I know, but it is one which does not appeal to me at all. I can see no reason why a tree like tea should ever die If it could be protected against accident and against its many natural enemies, its pests and its diseases. A bush may die of drought, water-logging, lightning, root disease, termites, etc., but never of old age, *i.e.*, of senile decay as that term is applied to animals. I shall be prepared to discuss that subject with anyone interested tomorrow after my demonstration. In the meantime I would ask you to consider definitions (1) of death as

applied to trees, and (2) of a young plant such as tea, dadap grown from a cutting, potato grown from a tuber, and plantain grown from a root sucker. My reason for wanting those definitions is that we have successfully struck 5 single node cuttings from a necrotic bush. Each cutting consisted of one leaf and about an inch of stem. The bush has been dug out and burnt. I hesitate to say that it is dead, because it may be argued that the cuttings are not young plants but are as old as the tea bush from which they were taken; in fact they are the old tea bush. Two of those cuttings have been sacrificed to curiosity and their young roots were found to be severely necrotic. If the cuttings are young plants, necrosis cannot be a symptom of old age. If the cuttings are not young plants, then the original bush cannot be dead although uprooted and burnt. Hence I would like a definition of death as applied to a tree.

4. A virus is possibly not an organism, but it is so small that it cannot be seen even with a microscope and it is capable of increasing and multiplying like an organism, when situated in a suitable environment. There are many known virus diseases of plants but up to the present none has been described on tea. I am of the opinion, however, that this disease which we have called *Phloem necrosis* is caused by a virus. As yet I have no proof. If the disease is caused by a virus then it is probably transmitted by an insect about which we know nothing, not even its identity.

In the tropics we are well acquainted with insect transmitted diseases like Malaria. Virus diseases are transmitted similarly but the insects concerned are not mosquitoes as a rule. They are sucking insects like aphids, scales and bugs which suck the juice out of a plant much as a mosquito sucks blood. The malaria germ is, however, visible under a microscope but a virus which occurs in the juice of an infected plant is not.

I am basing my opinion that the cause is a virus on the symptoms, the necrosis and the mode of occurrence in the fields. Proof that the cause of the disease is a virus depends largely upon an ability to transmit the disease in certain ways to a healthy plant. One way, probably the surest, is by grafting diseased tissue to healthy. Tea is not one of the easiest of plants to graft even when dealing with healthy tissues, so the great majority of our attempts to affect union between the diseased and healthy tissues have failed. We have now, however, a few grafted plants but so far we have not observed any symptom in the healthy stock, but we cannot yet say that the result is negative *

In this connection there is a question which cannot be answered, namely how long must elapse before the first identifiable symptom becomes evident, *i.e.*, what is the incubation period? The develop-

* The disease has later been transmitted by grafting.—*Ed.*

ment of the disease in the field is very slow, a year seems to make little difference. Judging by that measure it is likely that considerable time must elapse between infection and the development of a visible symptom. That, however, is a guess, not an ascertained fact, so the incubation period remains obscure.

I regard the slow development of the disease to be one of its worst characteristics. At the last conference I pointed out the impossibility of eradicating a disease like Poria so long as infectious material was left in the ground. Poria may take 3 years to kill a bush and during the greater part of that time no above-ground symptom is apparent. By the time the bush is dead the fungus has passed on to the adjacent bushes. But that is an old story. *Phloem necrosis* takes longer to make a bush unproductive than Poria takes to kill it. Moreover if the disease is carried by a flying insect the spread of the disease will be much greater than what we know to be the case with Poria, which travels underground.

To illustrate my point I should like to recount a little phytopathological history. There is a disease of peach in the United States which goes by the name of Phony peach. The cause is now known to be a virus, but the way in which it is transmitted in nature is still unknown. Transmission can be carried out artificially by root grafts only and 18 months elapse between infection and development of the first identifiable symptom. The word "phony' is defined as "bogus, counterfeit, false." The affected trees are dwarfed. The disease does not kill the attacked tree, but roots from such trees have never failed to communicate the disease to normal trees in root grafting experiments. The longevity of phony trees is recognised to constitute a serious problem in the eradication of the disease.

The phony condition was first recorded in Georgia before 1890. Phony trees began to be more prevalent about 1900, but not until 1915 was the situation regarded as serious and the U. S. Department of Agriculture's aid requested. At that time few of the growers believed it to be a disease, but considered it in the category of a sport, a degeneration of stock, (*i.e.*, old age) or an overdose of nitrogenous fertiliser. The great havoc it was destined to work was not foreseen. About 1928 the disease was proved to be contagious and control measures became a federal activity in 1929. By 1936 the disease was known in 15 States located in the S. E. quarter of the United States.

One of the problems of effective control is the reinfection of orchards from nearby diseased, abandoned and escaped peach trees, the removal of which was outside the scope of regular federal activities and beyond the means of available regular funds. From the Emergency Relief Appropriation was allotted a sum of 840,000

dollars for the removal of such trees by a force of 2,000 men. In four months 2 million abandoned and 3½ million escaped peach trees were removed. These figures which refer only to the expenditure outside the commercial orchards, will give you some idea of what this disease has cost the United States and the peach industry.

I have mentioned the phony disease of peach to show what extensive damage a disease which does not kill rapidly may do. I am not suggesting that *Phloem necrosis* of tea will follow a similar course, but it is up to us to see that it doesn't. To this end I ask your active co-operation. I am of the opinion that the disease is spreading. We now know it in the Kandapola, Bandarawela, Nuwara Eliya, Hewaheta, Dimbula and the Agras districts, and it possibly occurs on many estates not yet recorded. It is quite impossible for an officer of the Institute to visit every estate to search for the disease If, however, superintendents when on their rounds would keep a lookout for this disease and send specimens of suspected cases to the laboratory for examination, we should be able in a relatively short time to acquire a fairly accurate knowledge of the present distribution of the disease. With that information acquired, it would be possible to formulate reliable conclusions concerning its rate of spread and the conditions which favour it.

Since becoming acquainted with the disease a watch has been kept for its occurrence on St. Coombs. We found it for the first time last year and early stages are now known to occur in several fields. Our neighbours, however, have more bushes exhibiting advanced stages and I have no doubt that the disease has been present in Dimbula for some years, but unrecognised. The disease has probably been present on St. Coombs for some time yet remained unnoticed, but I think the fact that the majority of the affected bushes show only the early symptoms of the disease may be interpreted as indicating that the disease has arrived fairly recently. We know nothing about the way the disease is transmitted and very little about its rate of spread.

In the course of an experiment we examined 639 bushes and diagnosed 266 of them, *i.e.*, 42 per cent, to be affected by *Phloem necrosis*. The affected bushes were by no means all non-productive; in fact many of them would be passed by a planter as healthy. Eight months later the same bushes were again examined when the number diagnosed as diseased had risen to 334 or 52 per cent. This represents an increase of 10 per cent in 8 months. The plots as a whole and the bushes individually did not look worse at the second examination. I do not suggest that these figures represent the general rate of spread. They are the only figures we have and they relate only to a small area of tea in a heavily infected area.

There remains the question what should be done about it. My recommendation at present is that unproductive bushes should be removed. That is a sound economic proposition independent of any question of disease. I realise that by making the criterion unproductivity for the time being, the Institute is open to criticism that it is avoiding its responsibility for dealing with the disease as a disease. To that criticism I woud reply that if unproductive bushes are removed they will include amongst them the worst cases of *Phloem necrosis*. I admit that that treatment alone would never stamp out the disease if the opinions I have expressed are founded on fact. Let us assume for the moment that my opinions have been proved to be true by experiment and observation, and then consider what steps should be taken to eradicate the disease. The most important would be the removal of every bush as soon as it showed the first symptom of the disease no matter whether the bush cropped well or not. It would be of little use for one estate to remove its infected bushes if its neighbours did not do the same. Treatment may, therefore, have to be compulsory, and it would have to be very thorough. Before the Industry would consider taking such a step it would have to be convinced that such measures are essential. It would require facts and proofs, not opinion such as I have expressed today. So perhaps you will agree that the recommendation to remove unproductive bushes is as far as we can go at the moment. Later action will depend upon the results of further investigation.

I therefore ask your active co-operation in an effort to acquire a more intimate knowledge of this disease. I have viewed it from some of its worst possible aspects. Possibly, some of my views are wrong and investigations may prove them so. Until that is done I consider that *Phloem necrosis* should be regarded as a disease capable of causing very great damage.

In conclusion, I wish to thank the many planters, particularly those of the Kandapola district, for their active co-operation and the ready help they have given me to acquire what little knowledge we have of this disease. I would also express my appreciation of the work put in by my assistant, Mr. C. A. Loos. In one respect, at least, he has proved a great time saver in that he can detect the presence of necrotic zones in the petioles of diseased leaves without the aid of a microscope — a thing I cannot do myself.

DISCUSSION

Mr. F. R. Francillon first expressed appreciation of the interesting address Dr. Gadd had given. He agreed that the spread of the disease was very slow as he had known of that condition of

tea, now called *Phloem necrosis*, for the last 20 years. During that time there had not apparently been any great increase of it, and the field in question had in 1938 yielded 1,080 lbs. of made tea per acre which of course is a very good yield. Was it not possible Dr. Gadd was being over pessimistic?

MR. C. M. DURBIN said that coming from the Kandapola area he had considerable practical experience of the trouble which Dr. Gadd has described, and that experience suggested that the disease was a very serious menace to the Industry. The disease had probably been present in the Kandapola area for the last 10 or 15 years and they (the planters) had attributed it to a deterioration of jât as a consequence of high elevation. Now the disease had reached a much worse stage and it was not only killing individual bushes but was affecting the yield of estates.

He had been very much impressed by the remarks of Sir John Russell that morning concerning minor elements and the part they played in causing disease. They were accustomed to applying nitrogen, phosphoric acid and potash in manure mixtures but they had no information concerning the requirements of tea as regards minor elements. While he was in London recently he saw a report on the analyses of healthy and necrotic tea specimens sent home by the Tea Research Institute and that report seemed to indicate the possibility of soil conditions being the cause of the disease. So far as he remembered, that report suggested two possibilities, (1) a deficiency of manganese or (2) an excess of chromium. In view of these suggestions he hoped that Dr. Gadd would not reject soil conditions as a possible cause until the effects of minor elements have been determined.

Dr. Gadd had been rather severe on the suggestion that the disease was a result of old age, and in view of what had been said it seemed that the hypothesis would have to be abandoned. Still he thought there was the possibility that bushes 50 or 60 years of age, maltreated as they were, particularly at pruning time, might be more susceptible to disease than young plants would be. At present it seemed that only old bushes were attacked. That appeared to him to be a very important point because young supplies were being put out in the necrotic areas. The planting of supplies had been going on for the last 10 years and so far no necrosis had been found in the young plants. If necrosis should be found later in those young plants, the problem would become much more serious and he hoped in that case that it would not be long before he could retire. Although he admitted the possibility that the disease might attack young tea he sincerely hoped that it would not.

Finally, he said that he would like to tell Dr. Gadd that the planters in the Kandapola district were well aware of and much appreciated his great interest in this problem, which they regarded as of serious importance, and wished to thank him for all he had done to help them. He also expressed thanks to Mr. Loos who had worked extremely hard upon that problem.

DR. GADD in reply said that he felt sure that Mr. Francillon did not regard the high yield of the field he had mentioned to be a result of *Phloem necrosis*. He had been told by superintendents of surprisingly large numbers of diseased bushes which had been removed from fields without depressing the yields. Such statements illustrated to what small extent necrotic bushes contributed to the yield of the fields. Consequently he thought that Mr. Francillon would find that his excellent yield would not be materially reduced if he (Mr. Francillon) removed the severely diseased bushes.

With regard to Mr. Durbin's remarks concerning minor elements he stated that the spectrographic analysis, which had been very kindly and expertly done for the Institute at Home, had limited the field of exploration very considerably. He assured Mr. Durbin that that aspect of the problem would not be lost sight of. In fact, while Mr Durbin was on leave the Institute used a part of his estate for that very purpose. The minor element experimented with was boron but the boron treated plots showed after 8 months the same increase of the disease as did the control plots.

Mr. Durbin had referred to chromium as a possible cause of the disease. Dr. Gadd stated that chromium in excess was well-known to be toxic to plants so he had enquired of the chemist who had made the spectrographic analysis what quantities were involved in the tea samples. At considerable trouble the chemist had determined that the healthy tea contained about 1 part per million while the diseased samples contained about 2 parts per million. He was rather puzzled at the moment as to how he could increase the chromium content of healthy tea from 1 part to 2 parts per million in order to ascertain whether thereby the disease symptoms could be induced.

Concerning the age of affected tea plants, he said that he felt so confident that old age was not an important factor that he was working mainly with seedlings and young plants. Only last week he had examined two branches taken from a supply not more than 4 years old which had been sent to him suspected to be affected. The branches showed unmistakable external symptoms of the disease but as he had failed to find any definite internal necrotic areas he regarded his examination to be negative for the time being. He felt

sure that in a few weeks' time he would have little difficulty in finding necrotic areas in that plant. In that event it would be evident that young plants are not immune. He had dealt rather harshly with the old age hypothesis because he did not want them (the planters) to believe that, old age being the cause, the trouble would be avoided merely by pulling out the old tea bushes. His fear was that young tea would acquire the disease as readily as old bushes. He regarded that as a very serious character of the disease.