

A VIRUS DISEASE OF TEA.*

C. H. GADD, D.Sc.

Phloem necrosis of tea is a virus disease. Its cause is something quite different from that of other tea diseases with which you are acquainted, so it may be necessary to explain in what respects this differs from other, better known diseases of tea, caused by fungi.

What is a virus? Until recently, the most widely held view was that viruses were minute organisms resembling bacteria, but too small to be seen by ordinary microscopic methods. Recent research, however, tends to show that the chemical composition of a virus is somewhat different from that of an organism. That, however, need not concern us here. The simplest conception of a virus is of something so small as to be invisible under an ordinary microscope, yet resembling a living organism in its power of multiplication.

Apart from its size, a virus differs from most other plant pathogens (disease-causing organisms) by its obligate parasitism. An obligate parasite is one which will grow only in living tissues; growth and multiplication cease when it is removed from its living host. The well-known parasites of tea such as *Poria*, *Rosellinia*, etc. are not obligate parasites. They can live on dead matter, and may be grown quite easily on artificial media in the laboratory. All known viruses are obligate parasites. We may extract a virus from its host plant or animal, but having done that there is no method as yet known, by which that extracted virus can be induced to increase in amount. When, however, it is injected into a suitable living host it increases in amount, very rapidly as a rule. Because of its obligate parasitism a virus rarely kills its host plant. Its own survival depends upon the existence of the species of plants in which it can live. Some viruses kill a few species, but such viruses always have alternative hosts in which they cause little or no disease.

At the Tea Research Institute Conference in February I commented on the fact that *Phloem necrosis* rarely, if ever, killed a tea bush and said that that was one of its worst characteristics. Dead bushes attract far more attention than sickly ones; there is always a hope that the latter will get better after manuring or pruning, but a dead one has to be dug out.

* An address given to the Dimbula, Nuwara Eliya and Dickoya District Planters' Associations on May 29th, June 17th and June 19th, respectively.

Another consequence of obligate parasitism is that when such diseased plants are uprooted and destroyed, no infective material is left in the soil. No advantage is to be gained, therefore, by leaving a site fallow for a period. Supplies may be put in immediately without risk of infection from the soil.

We are accustomed to speak of leaf diseases, stem diseases and root diseases, and those terms are useful in that they indicate the seat of the disease. A leaf disease, as a rule, attacks the leaf only and does not pass into the stem or root. One of nature's ways of dealing with such a disease is to shed the diseased leaves. By so doing the plant may be freed from that particular disease for the time being, at any rate. Its freedom will perhaps be temporary, but if the disease recurs on the new leaves it is usually the result of new infection from outside, not from within the bush. Similarly we may get rid of a stem, or even a root disease, if only we could get at it in time, by removing the infected part. With virus diseases it is more usual for the virus to invade the whole plant with the exception of the seeds, and for that reason the disease is said to be systemic. Plants occasionally localise virus infection but, as a general rule, virus diseases are systemic. Consequently, symptoms become more generally and more uniformly scattered over virus-infected plants than they do with fungal and bacterial infections, which are normally localised in leaf, stem or root.

Phloem necrosis in tea follows the general rule of viruses in that infection is systemic; it affects the whole plant with the probable exception of the seed. Symptoms are found in root, stem and leaf; consequently, it is impossible to get rid of the disease by stripping off the leaves or pruning out the stems. The affected bush would survive such operations in most cases, but the new parts when formed would still be diseased, not as a result of new infection but directly from the parts left after pruning. The only way to get rid of all diseased parts is to dig out the bush entirely.

A natural sequel to the ability of viruses to spread through the vegetative organs of plants is that such diseases can be transmitted by grafting. Transmission in this way has become a test whether a plant disease shall be classified as a virus disease or not when a visible parasite is absent. Fortunately, tea is propagated from seed and not by grafting, so the risk of planting out diseased nursery stock is materially diminished.

Plant viruses do not produce spores to be dispersed by the wind like fungi do. Their distribution is brought about by insects. The particular insect concerned is known for many virus diseases, but not for all. It is probable that many virus diseases which are known

to be transmitted only by grafting or by mechanical inoculation do have insect vectors. The insects concerned are usually those which feed by sucking rather than by biting. As regards *Phloem necrosis*, we know nothing of the way it is transmitted naturally in the field. We know that it has spread extensively through the tea areas above 4,000 feet and that it is still being transmitted. It is highly probable, in fact one might say practically certain, that an insect is concerned, but as yet we have no clue to its identity. It is not necessarily an insect which may always be found in tea, but it is one which can and does feed on tea at times.

As regards the treatment of *Phloem necrosis*, I have on several occasions stated that the disease is incurable, and consequently have come in for a certain amount of criticism. Unfortunately, in some quarters, my opinion has given rise to the view that if the disease is incurable there is no reason to bother about it at all, not even to the extent of ascertaining whether it occurs on particular estates. If anyone interpreted my address at the last Tea Research Institute Conference to mean that this disease is of little or no importance I have been sadly misunderstood. In my opinion, *Phloem necrosis* is the most important disease of tea yet recorded, and judging from the damage done by other virus diseases to other permanent crops, this one should be regarded for the time being at any rate, as potentially as great a menace to tea as the coffee leaf disease proved to be for coffee in Ceylon towards the end of the last century. I should not be sorry to have my opinion proved to be wrong, but I should be extremely sorry if any statement of mine gave rise to indifference amongst even a small part of the planting community regarding the incidence and control of this disease. I do not ask that my opinion be accepted as gospel by anybody, but I hope it will not be disregarded merely because it may sound pessimistic.

Where disease is concerned, we are apt to argue from what is known more intimately concerning disease in ourselves and domestic animals, to what may be expected to occur in plants, and thereby often arrive at wrong conclusions. When we acquire an infectious disease we look forward to a normal recovery and restoration to health without the intervention of a surgical operation. A child gets measles. He exhibits all the symptoms; the disease runs its course and in 14 days or so the child recovers his normal health. To achieve that cure, the doctor has directed his skill towards relieving the discomfort, avoiding complications and giving general aid, but the real cure was achieved by the child himself, by what might be termed the anti-microbial properties of his blood. With the cure completed, the child is somewhat different from what he was previously; he has acquired immunity from further attacks of measles, which, by the

way, is a virus disease. Measles is quite typical of so many diseases which run a definite course, and the prime factor in the cure of them is what I have termed the anti-microbial property of the blood. Very few diseases can be cured directly by drugs — malaria is an outstanding example.

A plant has nothing equivalent to the anti-microbial properties of the blood of animals. At best, the plant can limit the invasion of the parasite to a localised area, but as a rule, to get rid of a disease the plant must get rid of the attacked organ. - That is relatively simple when leaves are concerned; the leaves may be dropped prematurely as occurs when rubber is attacked by *Oidium*, *Grevillea* by *Phyllosticta* and when a severe attack of *Cercospora* occurs on tea. New leaves formed later have no immunity against further attack by the same disease. Diseased stems and roots cannot be got rid of by the plant itself, consequently such diseases often prove fatal. Surgical operations may remove the diseased limbs and so save the lives of the infected plants. A parallel in the animal world is the amputation of a limb because of some incurable disease in it, but with animals and humans, surgical operations are last resources. An animal cannot grow a new limb whereas a plant can. So animals have anti-microbial properties of their blood, and plants, an ability to regenerate parts, but there can be little doubt as to which is the more efficient in dealing with disease organisms.

Perhaps this explanation will make it clear, what I mean by the statement that *Phloem necrosis* is incurable. As already stated, the virus is systemic, affecting all parts, so, parts containing the disease cannot be removed without removing the whole bush. The bush has no internal organisation for destroying the invading virus; otherwise, the bushes would recover without our interference. How then may the affected bushes be cured? A little more or less manure cannot help, and no virus disease of plants has yet been cured by spraying or by injecting chemicals. In the light of our present knowledge of virus diseases in general, *Phloem necrosis* of tea is incurable.

But because the disease is incurable, it does not follow that the disease cannot be controlled. The problem of control is clearly one of preventing infection. This could be achieved by eliminating all sources of infection, by exterminating the insect vector, or by preventing it from feeding on tea. As we do not know the identity of the insect concerned, we cannot start upon its extermination, nor can we take any necessary steps towards preventing it from feeding on tea. So we are left with the elimination of all sources of infection and that, I may say, has proved the most efficient method of control of other virus diseases.

The elimination of all sources of infection means the destruction of all bushes immediately they become infected. That may sound simple, but to put it into effect would mean the uprooting of a gigantic number of bushes, the majority of which are still productive. The disease is not restricted to a few districts as we believed to be the case at first. We have records now of the disease on 51 estates. Many of the records obtained since the conference are from estates in one agency, but I do not believe that that agency has anything like a monopoly of the disease. The distribution of the known infected estates suggests that every estate over 4,000 feet is suspect of harbouring the disease to a greater or less extent. When I add to this a statement that some fields have about 50 per cent of the bushes already infected you may form some idea of the number of bushes involved.

The question arises whether it is essential to remove all those bushes immediately. To that I would answer 'No,' which I hope will show that I am not entirely pessimistic. It may become necessary later, but at present I do not think it absolutely essential immediately.

We know of non-productive bushes which are necrotic but for every one such bush we can find many others apparently bearing well, yet exhibiting some symptom or other of the disease. One is apt to assume that the latter have acquired the disease recently and that they all will ultimately become non-productive. No matter how plausible such an assumption may appear, it is nevertheless assumption. Undoubtedly some bushes deteriorate rapidly after infection, but I am not so sure that they all do so. We know that some bushes are severely affected by the disease; we have seen cases where every vein of the leaf is discoloured and swollen. Such bushes are invariably poor ones though sometimes of fairly high jât. Other bushes show only slight symptoms and appear to remain productive. Whether they will continue to remain productive time alone will show, but there is considerable evidence that different bushes react differently to this disease.

That however, is not altogether unexpected. Tea bushes in up-country fields are anything but uniform in type; there is not one variety but many. A crop which is propagated vegetatively, like the potato, is much more uniform. As you know, potato varieties are numerous but an expert can name each accurately on inspection. Moreover, it is usual to grow the varieties separately, and it is possible to cultivate a large area with one variety only. The potato is attacked by many virus diseases, most of them have been studied in considerable detail and it is now known that some varieties are very susceptible to one virus, whereas others when infected by the

same virus show only mild symptoms or no symptom at all. The same variation of reaction by different varieties probably occurs in tea, and if so, that fact would offer the best means of control, in that the effects of the disease could be avoided by growing resistant varieties.

Control measures of that type may be easy for the potato grower, but it is quite a different matter with tea. By a variety of tea I mean something more than jât. The terms, High, Medium, Low, Indigenous, Hybrid, China and other jâts all include numerous varieties. Tea varieties have not been defined and it seems unlikely that they will be in the near future. Moreover, it is unlikely that in the near future we shall see pure seed, *i.e.*, seed which will give rise to anything approximating 100 per cent plants of exactly one type, alike in all characters. Obviously it is almost impossible at the present time to supply vacancies with one variety, and that, resistant to *Phloem necrosis*.

Before leaving these generalities, there is another point I would like to mention. The study of certain diseases has shown that there are several varieties of particular viruses. One variety may be very virulent and cause severe symptoms whereas another variety of the same virus may produce mild symptoms only and cause little damage. If different varieties of the *Phloem necrosis* virus occur, their effects in the field would be similar to those I have already ascribed to variation in resistance of different bushes, *i.e.*, some bushes would show mild and others severe symptoms. Since both variation in the virus and variation in resistance of bushes are possible, even probable, it will not be an easy matter to determine whether a bush showing mild symptoms has been infected by a mild variety or whether the bush itself has a high resistance to a virulent variety.

I have spoken about possibilities and probabilities far in advance of what research on this disease itself would warrant. I have briefly outlined them to show that although the disease may be incurable it may yet be controlled.

The recommendations I make at present are that non-productive bushes should be removed early. That recommendation should need no explanation on economic grounds. From the pathologic viewpoint it is apparent that such bushes are not resistant. There is, however, the other possibility that such bushes are infected by a virulent form of the virus. If so, then it is equally obvious that the sooner that infectious material is out of the way the better.

The sites of bushes removed because of this disease may be supplied immediately. I am sometimes asked what is the use of

PHLOEM NECROSIS

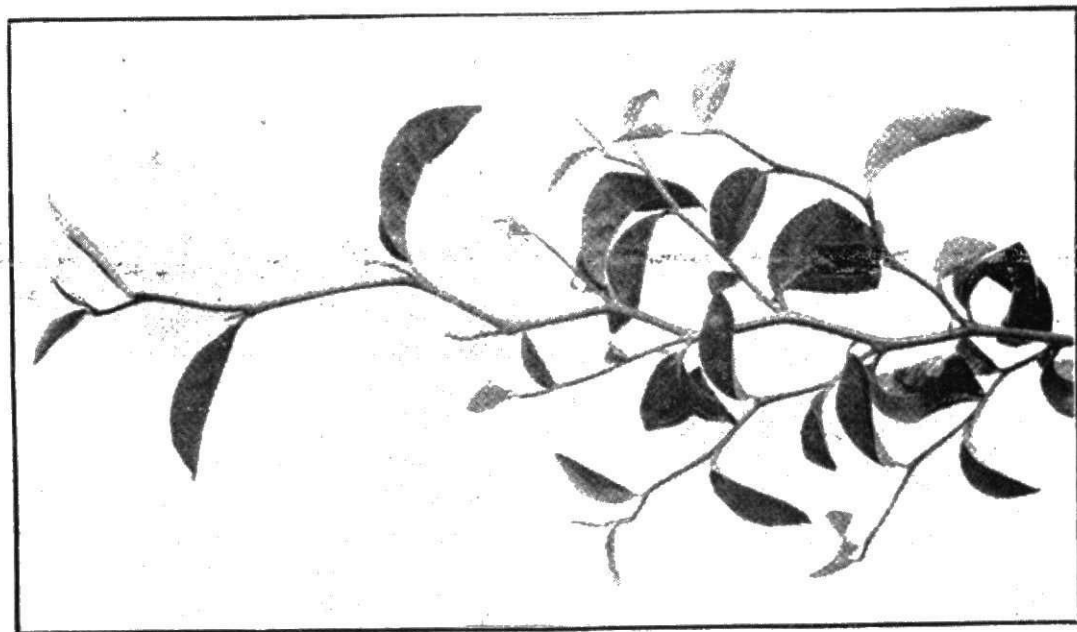


Fig. 2

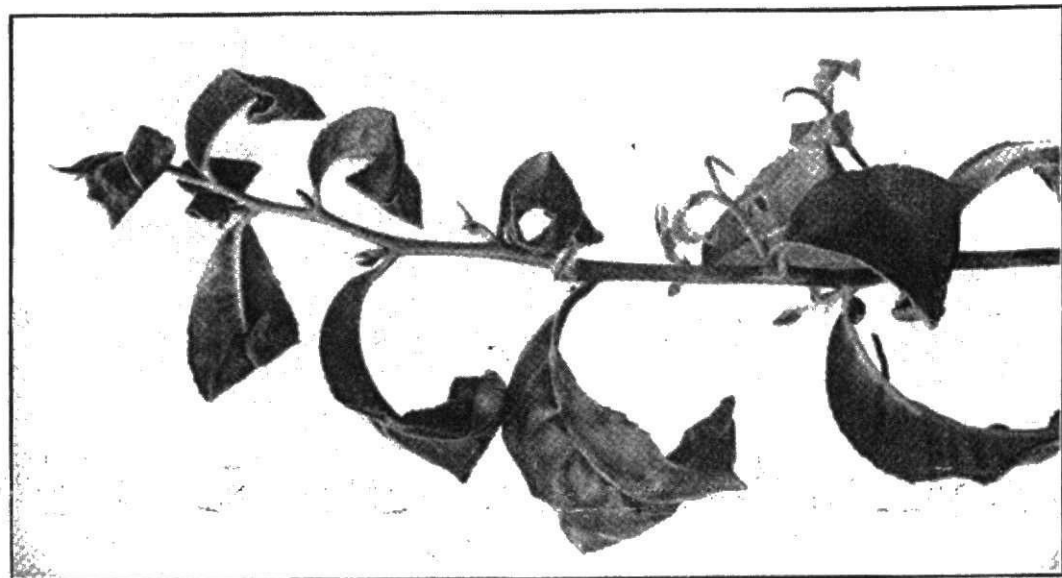


Fig. 1

Fig. 1.—Tipping shoot showing typical curled leaves.

Fig. 2.—A zig-zag branch from a necrotic bush. All infected bushes do not display this type of branching; but when it occurs the bush is usually necrotic.

supplying if the young plants are liable to the disease. To that I would reply that a dog breeder who loses a few pups from distemper does not stop breeding because future litters are liable to distemper, nor does he await the distemper immune breed. If tea plants vary in their resistance to this disease some of the supplies are sure to be more resistant than the bushes which have been removed because of their susceptibility.

Whether such measures will be sufficient to control the disease remains to be seen. They may have to be altered considerably later when more is known about the disease. They may prove sufficient on some estates but not on others, depending upon their elevation, probably.

All that is asked at present is that Estate Superintendents should keep a lookout for this disease; if found, to report that fact to the Institute; and to remove the uneconomic bushes. The only symptom that need be looked for at first is the backward curl or arch of leaves such as is exhibited by the specimens shown. (Figs. 1 and 2). Acquaintance with that symptom usually leads to the recognition of milder cases. The Mycological Division will willingly examine specimens for verification.

The best advice that could be given from a pathological point of view would be to remove every known infected bush, but I fear that on many estates such a recommendation is not economically practicable. Where the number of affected bushes is relatively small and the cost not prohibitive, I think it should be done. In any case, the non-productive bushes should come out early. Records of these removals will, in the course of time, show to what extent the disease is being controlled and what need there may be for more drastic measures. They would show whether infected bushes which are left at one round of removals because they are still economic producers, do in fact become worse and non-productive, and have to be removed at a later round. Information of that type is obviously of value and will assist in getting a better understanding of the disease.

DISCUSSION.

(In order to avoid repetition and to bring related questions together the discussions at the three meetings have been amalgamated).

MR. G. K. NEWTON (Dimbula) said that the Chairman had asked him to open the discussion on *Phloem necrosis* mainly because he had been responsible for first finding the disease in that district.

Dr. Gadd in his address had mentioned that *Phloem necrosis* rarely killed the tea bush and that that was one of the worst characteristics of the disease. In 1937 he (Mr. Newton) had found a dead tea bush on which he could recognise no disease so he sent it to St. Coombs, where the death was attributed to *Phloem necrosis*. Had that bush not died, it would not have attracted his attention and might not have been dug out for examination although it might have looked sickly. After the Institute had given details as to how the disease could be recognised he had found other necrotic bushes. He endorsed Dr. Gadd's opinion that sickly bushes did not always attract attention; and for that reason he thought it safe to assume that *Phloem necrosis* had existed in Ceylon tea for years without their knowledge.

Another characteristic which he considered dangerous was the slowness of its action, relative to crop returns. From a necrotic field of tea in that district the yields over four pruning cycles had been:—

1926/29	—	1,547	lbs.	per	acre
1929/32	—	1,570	„	„	„
1932/35	—	1,634	„	„	„
1935/38	—	1,866	„	„	„

Those figures showed an increase of about 100 lbs. per acre per annum due to liberal cultivation. Those figures suggested that nothing was wrong, but the increase in yield for that period over the whole estate amounted to some 200 lbs. per acre. Thus it could be seen that crop returns gave little indication of the disease during its initial stages.

He had been told of a director who came up from Colombo especially to see a field of necrotic tea, and when informed that the field gave an average of 1,000 lbs. per acre, expressed the opinion that his time was being wasted. It would be difficult to hazard a guess how much longer that field would continue to give 1,000 lbs. per acre but he thought they must now assume that there was little doubt of its falling off slowly but surely.

He complimented Dr. Gadd on his discovery that this disease was caused by a virus and he looked forward to the time when he could congratulate him on the discovery of the identity of the insect transmitter.

His own observations suggested that the disease occurred principally in fields exposed to wind and where soil was not particularly good. He asked whether Dr. Gadd considered such observations to be significant.

He had also noticed that the disease occurred chiefly on bushes of comparatively low jât and not on high jât, light leaf bushes. On that question he would like to hear other opinions.

MR. NEWTON concluded by saying that now Dr. Gadd had strongly recommended the replacement of unproductive bushes by supplies, he foresaw that the many bushes which had been uneconomic units for years would now receive attention which they had escaped in the past. By the substitution of better jât supplies, they might see in the years to come more even and higher yielding areas in tea up-country than was the case that day.

MR. BYDE MARTIN (Dimbula) said that his observations had been confined largely to tea of good hybrid type together with a certain amount of high jât Manipuri type. He had noted the disease to occur in two ways, in single bushes and in groups. Single bushes were situated amidst perfectly healthy, flourishing bushes growing in good soil. Groups occurred where soil conditions were poor, and in places subject to strong winds. Where the disease occurred in groups the affected bushes did not form a compact block as was usual with *Poria* but were more loosely scattered. In a block about the size of that room 25 to 30 per cent of the bushes might be found to be affected.

The age of the tea was mostly between 40 and 55 years; it also occurred in some old seed-bearers about 60 years old which had been cut back about 40 years ago and taken into plucking. He had not found the disease in the high jât tea which was about 30 years old.

The disease occurred most markedly on the top of the estate where the south west winds were felt very strongly, and on exposed ridges. The fields most severely affected by *Phloem necrosis* were quite definitely the worst yielders.

In view of Dr. Gadd's opinion that the seed would probably be free from the virus he asked if any experiments had been carried out to verify that view. He also enquired whether *Phloem necrosis* had been identified in other tea growing countries, the Netherlands East Indies or India, and whether the same disease was known to occur in plants other than tea.

MR. N. M. SANDERS (Nuwara Eliya) said he had listened to Dr. Gadd's most interesting and, from the point of view of the tea

industry, most disturbing address — disturbing perhaps because there were so many unknown factors.

The only cheerful point appeared to be that they might supply a vacancy caused by *Phloem necrosis* without any fear of infection from the soil. Prior to that he had thought it might be necessary to sterilize the soil by allowing it to remain fallow for a considerable period.

He presumed that even if one or two rootlets of the diseased bush, which had been removed, were accidentally left in the soil no possible infection could be transmitted from those to the newly planted supply.

Apart from grafting, the only means of transmitting the disease from one bush to another was by the particular insect vector.

If, as he understood, the disease was continually spreading from diseased to healthy bushes by means of the insect vectors, it would seem that replanting areas, bordered on all sides by old tea, some, perhaps all, of which was infected by *Phloem necrosis* could not be immune from attack for very long. Yet, taking the case of young supplies which, looked at in relative proportion, were exactly similar to replanted areas, it seemed extraordinary that such supplies, even up to ten years of age or more, appeared to exhibit no symptoms of the disease.

That led him to ask whether it was not better policy to replant, say, five acres a year rather than to supply individually the whole estate. In his own case, the cost would be very little extra and the coming into bearing of the replanted area would probably be very much quicker.

He wished to know whether Dr. Gadd could give an explanation of that fact. Could it mean that those insect vectors transmitted the virus at very irregular intervals, governed possibly by climatic conditions? Or was it that young tea was naturally more resistant to such attack?

The statement that *Phloem necrosis* was systemic (except possibly the seed) was, he presumed, an assumption drawn from the fact that it was rare to find a badly infected bush in the reproductive stage. He had with him, however, a few samples which, to a certain extent, belied that, and in consequence he was led to think that in the case of bushes less highly infected, fruiting would continue. It would therefore seem necessary that all recognised sources of tea seed should be subjected to inspection as a precaution against planting out from infected stock.

He wished to know whether *Phloem necrosis* had been discovered on tea areas which had been out of production for a number of years. If that might not be the case, might there be some connection between such tea and supplies, the inference being that tea in plucking was less resistant to the virus as well as indicating the possible method of approach of the disease — *via* the shoots broken by plucking?

With regard to the elimination of the source he wished to ask what steps were being taken to identify the insect vector. Could estate superintendents be of any assistance in that direction? If such insect was identified was it likely that a parasite could be discovered to combat it?

DR. GADD said that he thought Mr. Newton's memory to be at fault when he had stated that he (Dr. Gadd) has diagnosed *Phloem necrosis* on a dead bush in 1937, because that disease could not normally be recognised in a dead bush. He too had a Ceylon memory, so he could not recall what the top of the bush looked like, but he was certain that the roots were alive. Otherwise the presence of dead cells in the phloem would have been meaningless. So Mr. Newton evidently took more interest in sickly bushes than he pretended. He thought, however, there would be general agreement on the fact that dead bushes attracted more attention than did a larger number of sickly ones, particularly if the sick bushes were not together in a compact block.

He agreed that crop returns gave little indication of this disease in its initial stages, and judging from some figures that were given at the Tea Research Institute Conference in February, it appeared that in some instances, advanced stages of the disease might not be reflected markedly in the crop. In contradistinction, he recalled that before much was known of *Phloem necrosis* he was asked to examine a field to ascertain, if possible, the cause of its steady but marked fall in yield. After investigation he had no doubt that the real cause of that fall in yield was the disease *Phloem necrosis*.

It was a fairly common experience on estates where badly necrotic bushes had been taken out in large numbers, that the immediate effect on yield was negligible. It was to be expected that as more bushes became unproductive and were removed for that cause, there would be a diminution of crop harvested. He understood that such falls were occurring in some fields.

To illustrate the fact that the removal of non-productive bushes would not depress the yield he stated that he had been told by an estate manager of a field from which 5 per cent of the bushes

had been removed, not because they were necrotic but because they were considered uneconomic. During the next cycle the yield from that field had increased.

It would be apparent that the effect of that disease could be masked, at least for the time being, by liberal cultivation. How long the effects could be so masked he did not know, but he thought it would be inadvisable to accept the view of the director Mr. Newton had referred to, and to think all was well so long as the crop yield was satisfactory.

DR. GADD said that he had not personally noticed that the disease occurred principally in wind-swept fields nor where the soil was not particularly good. The significance of the observations would depend upon the extent of their applicability to all places where the disease occurred. The observations might be correct when limited to a small area but their value would decrease if they were not equally correct when applied to the larger area. He felt disinclined to generalise the soil observation, because the disease occurred very widely in the Kandapola area and he was not prepared to agree that the soil in that district was not particularly good, especially when compared with soils from other districts where the disease was not known.

MR. E. G. GROVES (Chairman, Dickoya Planters' Association) enquired whether Dr. Gadd had arrived at any conclusions regarding the effect of wind on the distribution of the disease.

DR. GADD replied that he personally had not been able to determine any connection between wind and the distribution of *Phloem necrosis*. If the insect vector were carried mainly by the wind the disease would probably follow a course defined by the direction of the prevalent winds. But so far he had no evidence to warrant any such conclusion.

Concerning the mode of occurrence, DR. GADD agreed with Mr. Byde Martin that although the disease was best known in patches such as he had described, it also occurred more or less as single bushes surrounded by healthy tea. Recently he had attempted to survey a rather lightly affected field by examining every bush in certain rows and noting the number healthy and diseased. There might be 20 or 30 healthy bushes in a row, the next one or two might show symptoms of the disease, and the row then continue as healthy bushes. Where diseased bushes were found in those circumstances it was often possible to find other bushes nearby in adjacent rows. He feared that the apparent single bush cases were really the beginnings of patches, but prolonged observation was necessary to be sure of that.

DR. GADD said that he had found no record of *Phloem necrosis* of tea in places outside Ceylon but intended making further enquiries on that point. Also, he knew of no plant other than tea which carried the specific virus concerned in that disease. Such plants might exist, but if so, he did not know what they were.

MR. HUGH GORDON (Dickoya) asked if Dr. Gadd could say how long *Phloem necrosis* had existed in the Island.

DR. GADD replied that he had no idea. He had first become acquainted with the condition about 1935 and the disease was well established then. He knew of one affected field which the Superintendent said had been in the same condition for the last 25 years, but when pressed, could not remember whether the bushes exhibited curled leaf or whether the field had so many unproductive bushes when he first knew it. In the absence of written record it would be extremely difficult to estimate how long this disease had occurred in Ceylon.

MR. WATKINS BAKER (Dickoya) said that he understood Dr. Gadd to say that the lowest elevation at which the disease was found was 4,000 feet. Was it likely, and would it surprise him to find it at sea level?

DR. GADD said that that was a difficult question to answer. The lowest elevation at which the disease had been recognised was about 3,400 feet but with that exception the estates known to have this disease were situated above 4,000 feet. It seemed to him that the nearer he worked to the 4,000 feet mark the more difficult it was to recognise the disease. Was it that the symptoms developed better at higher elevations and so enabled the disease to be more easily recognised? That, to his mind, raised the important question whether temperature had an important bearing on this disease. The bush might have the virus within it, yet because the temperature was such, the symptoms did not develop. It was known that plants could be infected by a virus and yet show no symptom. In that respect they were like enteric carriers, persons who enjoy normal health, yet have the misfortune to infect their neighbours with enteric.

He referred to a virus disease of passion fruit in Queensland. Symptoms of that disease were most readily detected in spring. Owing to the increased temperature of summer the symptoms then became masked, but the plants produced a worthless crop of woody fruit in the winter. He had no evidence that temperature had a similar masking effect with the virus of tea, but if that possibility were taken into account the virus may occur at lower elevations than was then suspected.

Another possibility was that the insect vector occurred only in up-country districts. In that case the range of the disease would coincide with the distribution of the insect. In the absence of conclusive evidence and until such time as the facts could be determined, those arguments were not worth pursuing. The experience of the Institute was that the symptoms of the disease did not appear much below 4,000 feet.

MR. M. L. WILKINS (Dickoya) asked whether the examination of specimens with a microscope would help superintendents to identify the disease more easily.

DR. GADD replied that he did not wish to trouble superintendents with methods of identification of that sort. The only symptom he would ask superintendents to look for first was the backward curl or arch of the leaves. There were of course other symptoms, some of which were difficult to put into words, but if superintendents would, in the first instance, look for the symptom mentioned they would also become acquainted with other characters of affected bushes. In cases of doubt the Institute was always willing to examine and report on specimens.

MR. C. OWEN (Dickoya) suggested that it would be useful if cheap reproductions of photographs depicting the symptoms of the disease could be distributed among the more intelligent of the labour force. They would then be in a position to judge of the existence and extent of infection on estates.

MR. E. M. WINDUS (Dickoya) thought they should not be cheap ones. They should have perfect reproductions for the purpose of identifications.

DR. R. V. NORRIS promised to go into the question. He thought the cost would not be prohibitive as the principal item was the preparation of blocks and a number of blocks prepared in connection with the recent conference were already in existence. So the actual cost of producing copies would be only that of printing.

MR. MARSH SMITH (Dimbula) enquired whether a badly infected bush could have some leaves not showing the symptoms of *Phloem necrosis*.

DR. GADD replied that young, recently formed leaf rarely exhibited the symptoms of the disease though they might develop later as the leaves aged. The leaves produced on a shoot during one rush of growth might exhibit the symptoms markedly in those leaves near the fish leaf, yet the symptoms would not be evident in the younger leaves near the growing point. For that reason it was preferable to send older leaves for examination when there was doubt

regarding infection. Some bushes showed the symptoms clearly on part of the bush only but as a rule badly infected bushes were easy to recognise.

MR. H. L. CLEGHORN (Dimbula) asked whether a necrotic bush became more normal as it became older from pruning.

DR. GADD replied that the disease was most easily recognised in the fields coming back from pruning, about tipping time. In the second and third years from pruning, fields as a whole looked better, but in his experience bushes which were found to be diseased at tipping could be recognised as diseased throughout the cycle although their general appearance was better in the later part of the cycle. Really bad cases looked as bad in their third year as in their first.

MR. C. HUNTLEY-WILKINSON (Dimbula) asked if Dr. Gadd would tell them where the phloem tissue occurred in the tea bush and what was its function.

DR. GADD then explained the position and function of the phloem. He stated that he had omitted such explanations from his address as he had previously described them in his paper at the Tea Research Institute Conference, published in *The Tea Quarterly*, Vol. XII, Part II, p. 77.

MR. M. L. WILKINS (Dickoya) asked if there was any truth in the theory that *Phloem necrosis* was peculiar to bad jâts of tea, and conversely, if there was any evidence to support the idea that a good jât was immune.

DR. GADD said that the disease occurred most frequently in fields of the hybrid type. He had seen the disease on bushes of good jât also, so he could not say that good jât bushes were immune and poor jât susceptible. It had to be borne in mind that a dwarfing of the leaves frequently accompanied this disease. As high jât was distinguished from low jât principally by the size of the leaf, it followed that necrotic bushes tended to appear lower in jât than they really were. That tended to make the disease mainly one of low jât bushes but undoubtedly the disease did occur on bushes of good jât.

MESSRS. A. W. CANTLAY (Dimbula) and G. E. D. FOSTER (Nuwara Eliya) enquired whether it was possible for infection to occur through the use of a pruning knife.

DR. GADD replied that it might be possible to transmit the disease with a pruning knife or even by pluckers' fingers as suggested by Mr. Sanders (Nuwara Eliya) because it was the sap of plants which contained the virus, but he did not think it probable. He described an experiment which he had carried out to test that

point. Freshly made pruning cuts were covered with fresh, pulped, necrotic tissues in order to ensure that the cuts were well wetted with juices from infected plants, but so far the treated bushes showed no sign of the disease.

MR. GORDON WINDUS (Nuwara Eliya) asked whether infection was conveyed through the leaves or bark, and MR. STANLEY HOWARD (Nuwara Eliya) whether Tea aphis, which was common in that district, was a probable vector.

DR. GADD said that as he did not know what insect carried the disease he did not know whether it was transmitted through the leaf, stem or root. Tea aphis was the first insect to be suspected and he had tried to transmit the disease by means of it. He had been unable to transmit the disease under the conditions he then used.

Replying to MR. SANDERS (Nuwara Eliya), Dr. Gadd said that sucking insects usually became very common during times of drought, so the disease might possibly be transmitted naturally only at such times. He was not prepared to state that was actually the case.

Up to the present very little had been done towards determining the insect vector. He personally regarded that as a job more for the Entomologist than for himself. He realised that the Entomologist might require a good deal of ground cleared for him. For instance, he would want some symptom which developed fairly quickly after infection had occurred. At present, considerable time, a year or more, had to elapse before they could be sure that an experimental plant had acquired the disease.

When the insect had been found he advised them not to bank too much on the possibility of eradicating it. Very few insects, if any, had ever been eradicated by spraying and such like methods, and one could not possibly envisage the spraying of tea at frequent intervals for obvious reasons. They had been very fortunate in finding an effective parasite for tea tortrix, but he warned them not to be too optimistic about finding other parasites to control all their other pests.

MR. GORDON WINDUS (Nuwara Eliya) enquired whether it was advisable to burn an infected bush immediately it was uprooted.

DR. GADD replied that he thought it unnecessary to do so unless the bush was infected by insects, which was extremely rare. When removed from the ground the bush was not a very favourable food supply for a sucking insect. It was very unlikely that such insects would visit dried up bushes so there was no great objection to such bushes being carried to the factory as fuel. In that respect *Phloem necrosis* was not on par with *Poria* or *Rosellinia*.

MR. E. G. GROVES (Chairman, Dickoya Planters' Association) asked whether it was advisable to continue propagation by cuttings in view of *Phloem necrosis*.

DR. GADD said that he was asked at Dimbula whether they should stop propagation from cuttings and had answered 'No.' He still thought that answer correct. Estates working on propagation from cuttings first selected their highest yielding bushes. So if the selection had been made in fields where *Phloem necrosis* occurred the chosen bushes would probably be either immune or had escaped infection. It seemed unlikely that diseased bushes would be selected as such bushes would not be high yielders. If truly immune bushes were discovered, vegetative propagation might become the normal method of supplying.

Concerning seed, Dr. Gadd said that Mr. Sanders (Nuwara Eliya) had apparently misunderstood him. When he had stated that it was unlikely that the *Phloem necrosis* virus would be carried in the seed he had not intended to imply that seed production on infected plants was a rarity. He had found very few seeds on necrotic bushes, but then many healthy bushes did not produce seed.

Answering Mr. Byde Martin (Dimbula) on the same subject DR. GADD admitted that his statement was a generalisation from what was known of other virus diseases. He had collected and germinated a few seed from necrotic bushes but it was too early yet to decide whether the plants were really free from necrosis.

At Nuwara Eliya, DR. GADD also stated that if it were proved later that tea seed did, in fact, carry the virus, it would be very unlikely that infected seed would be recognised by mere inspection, as had been suggested.

MR. E. M. WINDUS (Dickoya) asked whether there had been any research regarding the relationship between age of the bush and its susceptibility to the virus. He thought that might have some bearing as to whether it was worth while supplying. In other words was a young plant less immune to the virus than an old one?

DR. GADD said that the disease was very rarely found in supplies which had been put out into the field. In one district a large number of necrotic bushes had been removed and so far they had not found the disease in any supply put in their places. But that did not necessarily mean that young plants were immune.

For certain reasons he worked with young plants and had succeeded in definitely transmitting the disease to a two-year-old plant. He believed it possible to transmit the disease even to young seedlings.

He said that immunity to a particular disease was usually an inherent character, so if a plant was immune during the early years of its existence it was likely to prove immune for the rest of its life. Immunity however could not be recognised by mere examination. If a man lived for a time in a malarious district without taking any necessary precautions and yet did not acquire the disease, was that man immune to malaria or had he merely been fortunate in escaping infection? As they knew, it was not every mosquito bite that led to infection. The same would hold good of the transmission of *Phloem-necrosis* by an insect vector. The insect would first have to feed on a diseased plant and then on a healthy one before the disease was transmitted, and possibly the virus would have to be left in a particular tissue before infection occurred. Thus it was not possible to distinguish easily between immunity and escape from infection. A plant might grow for some years in a virus area and escape infection but the longer it lived there the greater its risk of acquiring the disease, unless it was really immune.

It would be difficult to determine immunity and its relationship with age until the technique of transmitting this disease was improved and some symptom by which early stages of infection could be recognised. His opinion was that a young plant was as susceptible as an old one.

Replying to Mr. Sanders (Nuwara Eliya), DR. GADD said that whether blocks of tea should be uprooted for replanting or whether individual bush sites should be supplied depended upon the circumstances of the case. If only a few necrotic bushes had to be removed it seemed unnecessary to uproot a whole block.

He thought that the policy of supplying should be continued. If, as he had suggested, mature bushes exhibited various degrees of resistance, the same would probably hold good for supplies. As the supplies replaced bushes which had proved to be susceptible, the number of resistant plants in the field would be increased.

He could not offer a very satisfactory explanation of the observation that all supplies up to ten years of age appeared free from this disease although growing in a heavily infected area. It was fairly common to find two bushes next to each other, occasionally growing in the same hole, and one was necrotic and the other healthy. Because of the time an old bush had run risk of infection and yet remained healthy, he had assumed that the healthy bush remained healthy because it was immune or had a high degree of resistance. But he could not assume that all supplies were immune otherwise he would not have been able to transmit the disease to a two-year-old plant, as he had done.

He thought that the fact that supplies escaped infection might, in some way be connected with the insect vector. Obviously an older bush formed a larger target for the insect and possibly, the insect preferred to feed on older bushes because of their greater shade or other character.

MR. P. A. ARMSTRONG (Nuwara Eliya) said that he thought where the diseased area was small relative to the rest of the field the chances of infection would be small if the area was replanted. Was it so when a large number of necrotic bushes were removed while others were left? He thought that supplies put in the places of bushes removed for *Poria* would have a much better chance of escape than supplies put into a definitely necrotic area because of infected bushes in the vicinity.

DR. GADD said that *Poria* was a fungus which could grow from an infected root for only a short distance through soil in search of another root. Bushes in the immediate vicinity of an infected bush ran the risk of infection. By vicinity, there, was meant within a very few feet of the infected bush. The chances of survival of a supply put into a *Poria* patch depended upon the distance it was away from the nearest infected root. A few feet usually meant safety. But when they spoke of vicinity of a necrotic bush they did not necessarily mean within a few feet. Much depended upon the flight range of the insect vector. For instance, if the insect normally flew about 50 yards all bushes within a range of 50 yards from an infected bush would be liable to infection. He did not suggest that 50 yards was the actual flight range of the insect concerned, because as he did not know the insect he knew nothing of its habits or movements. Also he could not assess the risks of infection from outside a replanted block as compared with the risks when individual sites were supplied.

MR. C. M. DURBIN (Nuwara Eliya) said it was of vital importance to find out what was the position with regard to the disease as it affected young supplies. He felt that Dr. Gadd has not drawn a convincing picture in his analogy to dog-breeders and distemper. If distemper was absolutely incurable, dog-breeders would soon go out of business. Here was a disease which was incurable. He failed to see what chance they had for their young supplies if they put them out with just a vague chance of their being an immune variety.

He had a field, which Dr. Gadd knew, was heavily infected. It was full of supplies over ten years. Some of them were very big bushes and therefore were more likely to catch the vector's eye than ordinary bushes in plucking. Not one of these supplies over ten years had been infected as far as he was aware. The point

required investigation to find out the reason for their apparent immunity. Unless they could be sure of the economic life of supplies over 30 or 40 years it was a very poor outlook indeed. He thought there was some other reason than those suggested so far as to why supplies over ten years, growing up into big bushes, were still apparently untouched by the disease.

The other point Dr. Gadd had stressed was the importance of eliminating the sources of infection. It appeared to him a counsel of despair. Seeing how very heavily their tea could be affected and they did not even know the early symptoms, it seemed to him, although it might be well to stress the importance of eliminating those bushes, that it was a dark outlook. He wished to see the Tea Research Institute, while emphasising this method of attacking the problem, bringing in the Entomologist to see if something could not be done to find out what the insect was in the case. He also wished to know whether any efforts had been made to get further assistance in investigating the problem.

DR. GADD said that he hoped they would understand that he had not come before them with any pretence of knowing all the answers to questions they might put to him. The question regarding supplies was certainly one to which he did not know the correct answer.

He had given a good deal of thought and time to the study of that disease. He had all along painted it as a black picture because he regarded that disease as the worst they knew in tea. Much of the information given by him had been derived from various sources, from research work carried out with other crops and with other viruses. But he had tried as far as was possible to ascertain how far that information held good as regards tea.

As regards the efficacy of supplying, DR. GADD said that if he was to be asked whether it was any good going on trying to supply areas, he would ask in return what they would suggest to be done in place of it.

He realised that his answers concerning the apparent freedom of supplies from this disease might not be very satisfactory but he thought that Mr. Durbin's remarks provided another answer. If the early symptoms were unknown it was probable, as Mr. Durbin realised, that many old bushes had acquired the disease and yet showed no recognisable symptom. If he would admit that as regards old bushes would he not allow that the same thing might occur as regards the supplies.

DR. GADD admitted that he could not recognise the earliest stages of infection. The disease was recognised from certain gross characters and from the presence of dead cells in certain tissues. He could not envisage those cells being born dead. He knew for a fact that they were not, because they were alive in the youngest leaves of affected bushes. Evidently a period of time elapsed between infection and recognition that infection had occurred.

To illustrate that point he said that a healthy plant two years old growing in a pot had been grafted to a necrotic bush. The necrotic branch was later severed from the parent below the graft; the pot plant was brought back to St. Coombs, removed from the pot and planted out. Twelve months elapsed before he was absolutely certain that the disease had been transmitted.

The amount of virus which had passed through the organic union of the graft from the diseased scion was immeasurably greater than that which would be injected by an insect during natural transmission. As 12 months passed before the disease was recognised after grafting might not a much longer period have to pass before the disease could be recognised when only a very small quantity of virus was injected by an insect?

That experiment illustrated one of the many difficulties encountered in the study of this disease, namely the recognition of it in the early stages.

DR. NORRIS said that it would have been gathered from Dr. Gadd's remarks that one of the most important pieces of work yet to be done in connection with *Phloem necrosis* was to find some means of recognising the earliest possible symptoms of the disease. They felt that the symptoms described were not early symptoms, so the disease might be affecting bushes some time before it could be recognised. That was one of the greatest dangers because it meant that the disease was well in advance and measures to combat it were trailing behind. Early recognition of the disease was obviously desirable. That meant considerable laboratory investigation.

It would interest them to know that the Institute was endeavouring to obtain additional assistance for Dr. Gadd. Enquiries were being made at Cambridge, Rothamsted and the Colonial Office to ascertain if a temporary man — they could not afford a permanent appointment — experienced in that type of work could take up that side of the investigations. There must be some difficulty about it because people with experience of virus diseases were not plentiful, but they were endeavouring to do the best possible.