



## WOUND GUM.

By

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As considerable interest has been aroused during the last year by theories which have been based on the alleged occurrence of a wound-gum barrier in tea stems after pruning, it may serve a useful purpose, and prevent a too hasty acceptance of such theories, if we place before our readers some information concerning wound gum.

In many "broad leaf" trees and shrubs, when they are wounded down to the wood, the exposed wood becomes discoloured, usually brown, and this discoloration extends to varying depths into the wood. On microscopical examination it is found that the walls of the wood cells are impregnated with a brown colouring matter, and that the wood vessels, fibres, etc., are filled with a brown substance. In general, this brown substance is what is known as wound gum.

The name, wound gum, is in some respects an unfortunate one. The substance is not soluble in water and it does not swell when moistened. It is doubtful whether it should be considered a gum.

The term "broad leaf" trees has been used above, as a distinction from "needle-leaf" trees, such as pines and other conifers. In the latter, the substance which similarly fills the wood cells near the surface of a wound is a resin.

The formation of wound gum has been studied by many investigators, and it is now generally accepted that it is formed from the carbohydrates in the medullary rays and wood parenchyma. It is formed in living cells. Hence the cells on the surface of the wound, which have been dried out by exposure, do not take any part in the formation of wound gum. Partly for that reason, a wound gum "barrier," if present, is situated some little distance below the surface of the wound.

The formation of wound gum in plum trees begins a few hours after wounding.

Wound gum, by blocking the vessels of the wood, hinders or prevents loss of water from the exposed surface, and prevents the entrance of air and water into the naked wood. It is, however, incorrect to suppose that wound gum is produced for that purpose. The occurrence of "wound gum" is not restricted to the neighbourhood of wounds. The coloration of the heart wood of many trees, which is found as a normal phenomenon in perfectly sound stems and branches, is due to the deposition of "wound gum." Hence, wound gum is not necessarily associated with wounding nor with exposure of the wood to the air. The precise physiological reasons for the formation of wound gum have not yet been determined.

From the foregoing, it will be understood that even if a plant is forming wound gum, one must not expect to find exudations of gum, such as occur on Acacias or orange trees when injured or attacked by disease. Such exudations, as a rule, are true gums, and their mode of production is different from that of wound gum.

Wound gum cannot be identified by mere naked-eye inspection. Wood may be discoloured by other agencies, notably by fungi, either by the action of the fungus on the wood or by the presence of coloured mycelium in the wood cells. It is necessary to make a microscopical examination, before the presence of wound gum can be definitely determined. The investigations of the last fifty years have led to the introduction of numerous micro-chemical tests and stains for the identification of wound gum, and it is only by means of these that the presence of wound gum in a plant tissue can be established.

Interest in Ceylon in this subject of wound gum has been aroused by the application to tea of the results of investigations carried out in England on diseases of fruit trees, the most important of which, on the Silver-leaf disease of plum trees, have been conducted under the auspices of the Ministry of Agriculture by Mr. F. T. Brooks and his co-workers at Cambridge and elsewhere. When in England in 1925, I had the privilege of seeing Mr. Brooks' experiments at Cambridge, and his microscope preparations showing the effect of wound covers, the penetration of the fungus, etc.

Brooks and Moore found that the formation of wound gum in plum trees is most rapid in June, July, and August. It is not confined to those months, but apparently "gum barriers" are more readily formed in the summer months than at other times of the year. This finding is corroborated by the investigations of Swarbrick (*Jour. Pomology and Horticultural Science*, Vol. V., No. 2).

In the course of infection experiments with the fungus which causes Silver-Leaf of plum trees, Brooks and Moore found that it was almost impossible to obtain successful infections in June and July (and in one year in August), although infection followed inoculation quite readily in other months; and on seeking the reason for that they found that, where infection had failed, the fungus had penetrated to a depth of about half an inch, but further progress had been stopped by a barrier of wound gum. "The wood permeated by the mycelium was brown in colour as usual owing to the presence of gum in the cells, but, on the margin of the wood affected in this way, there was a narrow zone much more intensely discoloured, and, in fact, almost black. The vessels in this deeply discoloured zone contained even more gum than the region permeated by the mycelium, and the gum was darker in colour than in the latter. The mycelium had not entered this delimiting zone, so that the fungus was completely shut off from the healthy wood beyond. It seems certain that this 'gum barrier,' as it

may be called, definitely prevents the fungus from proceeding further."

The foregoing is quoted from Brooks and Moore's paper, Silver-leaf disease—V., Journal of Pomology and Horticultural Science, Vol. V., No. 2, March, 1926, as is also the following extract.

"The reasons for the rapid formation of a 'gum-barrier' in June, July and August, thereby preventing infection, are by no means clear. 'Gum barriers' are certainly not limited in time of formation to these months, because whenever there is failure to proceed with invasion after initial penetration, and in all cases of recovery from the disease, a 'gum-barrier' is responsible for the defeat of the fungus. There is reason to suppose, however, that 'gum barriers' are more readily formed in the summer months than at other times of the year. This is probably due to some special physiological condition of the twigs during these months. Alternatively, these barriers may be the more readily formed then because the fungus perhaps attacks most vigorously during that period. With the enhanced temperatures of the summer the fungus grows faster, although it may be argued that with higher temperatures the protective gum is also more easily formed. If it be true that the protective gum barriers are brought into being by the vigor of attack of the fungus, the ultimate escape from infection can be compared with the well-known case of resistance of certain varieties of wheat to *Puccinia glumarum*, where the ultimate immunity is due to a too vigorous initial attack by the fungus. The almost constant lack of infection during the summer months is, however, probably related to some peculiar feature in the initiation of attack of the fungus during the period, in association with the wound response of the host; otherwise one would expect that invasions by this fungus begun in April would stop in June: that is certainly not the case. It is the early stoppage of invasion in the summer months which is of peculiar interest in these experiments, but whether this is due primarily to the host or to the fungus, or whether the 'honours' are even between the two, for the present remains somewhat obscure, although the available evidence points to the physiological condition of the host as being most important."

The practical deduction drawn by Brooks and Moore from the foregoing results is that, to escape infection by *Stereum purpureum*, the fungus of Silver-leaf disease, cutting out of dead wood and living branches of fruit trees should be done in the early summer.

But as regards wound covers, Brooks and Moore state "*Wounds should, of course, be protected either with soft grafting wax or with one of the paints described in the earlier part of this paper*" (italics mine,—T.P.).

It must be particularly borne in mind that these results refer to plum trees, in which the existence of a protective gum barrier (under certain conditions) has been proved, and to a particular parasitic

fungus, the cause of Silver-leaf disease. They cannot be transferred, without investigation, to tea, in which the existence of a gum barrier has not been demonstrated, and to the numerous saprophytic fungi which cause wood rot in tea.

Before these results can be applied to tea, it must be shown (1) that tea can form a similar gum barrier, and (2) that this barrier is effective in stopping the progress of all the saprophytic fungi which grow on tea wood.

Moreover, although Brooks and Moore were aware of the existence of the gum barrier in plum trees, and that it stopped the progress of the fungus which causes Silver-leaf, they nevertheless recommended that wounds on plum trees should be covered with grafting wax or a protective paint. But in Ceylon, the practical application of Brooks and Moore's results is diametrically opposed to what those investigators recommended, for in this country the advice which has been based on those results is that no protectives need be applied to wounds on tea:

In Ceylon there is a tendency to confuse wound gum formation with callus growth. These two are different processes, callus growth being the growth of new tissue from the cambium at the margin of the wound, while gum formation is the decomposition of reserve materials in the wood. Naturally, both depend, in the ultimate, on the presence of reserve food in the plant, but otherwise there is no relation between them. The presence of callus at the margin of the wound cannot be taken as evidence of the existence of a wound gum barrier below the exposed wood.

Nor can a marginal callus exert any protective function over the centre of the wound which is not covered by it. In the average tree, the external part of the woody cylinder, the sap wood, is less resistant than the central heart wood. Consequently, when a stem is cut across and the whole of its cross section exposed, the outer layers of the wood may be more readily attacked by fungi than the central part. (That is not universally true, as some fungi prefer the heart wood). The callus growth, which, as a rule, starts at the margin, covers first the less resistant sap wood and protects it from the direct attack of fungi, leaving exposed for a longer time (often permanently) the more resistant heart wood. The heart wood decays more slowly (in general) because it is harder and, in some cases, impregnated with gums, etc., not because it is surrounded by a ring of callus.

As regards tea, this idea of an immunity from decay conferred on the exposed wood by a marginal callus is even less applicable, as there is not the marked difference between heart wood and sap wood which occurs in deciduous trees.

As may readily be seen, when a cut stem dies back, the callus grows from the living part of the plant over the dead tissues, surrounding the dead stem with a socket of living wood and bark. The callus cannot grow through the dead stem along the surface of a gum barrier.