# BROWN BAST*

## SOME CONSIDERATIONS AS TO ITS NATURE

THE origin and nature of this disease are obscure. Many different explanations have been advanced and the literature is at first sight confusing. Later investigators agree that the origin of the disease is physiological, but an explanation covering all the histological phenomena and the generally accepted evidence as to incidence under various conditions seems to be lacking. The present contribution provides some considerations which add weight to the view that the disease is physiological and intimately linked with the phenomenon of "wound healing" in woody stems. Wound healing on all kinds of trees, temperate and tropical, has been investigated by numerous workers and the factors which govern the deposition of "wound gum" at the open wood wound seem to be universal. The reasons for associating Brown Bast with this general tree reaction will be presented and it will be shown that, considered in this light, a very complete explanation of the disease is possible.

### WOUND HEALING IN WOODY STEMS

A recent paper by Swarbrick provides information of exceptional interest in relation to Brown Bast and his findings are set out.

Swarbrick, dealing with the histology of wound healing, examined the effect of making pruning wounds on the branches of temperate zone trees. He finds that, some time after making such a wound, there appears behind the injured surface a block which separates the injured cells from the living ones beneath. The amount and rapidity of appearance of the blocking substance is always greatest during those months of the year when there is active growth and sap movement in the tree, particularly rapid blocking taking place just after wintering. If wounds are made at such a time, starch rapidly disappears from cells below the wound and, coincident with starch disappearance, there appears amongst other substances, a yellow viscous body which actually forms the substance of the block. This body is of an extremely chemically-resistant nature. It is resistant to fat solvents, cellulose solvents and acids except hot concentrated nitric acid which rapidly degrades it. Alkalis cause it to darken in colour but do not dissolve it. When first formed, it gives no reactions for tannins but after some time it develops this property, and with it the property of responding to lignin reactions. After some time, it hardens and contracts and assumes a wrinkled form. This substance, for want of a better name is called "wound gum," and the occurrence of this body has been reported by many other workers notable among whom is Coster whose observations were made on tropical trees. Swarbrick also established the fact that the time when most active blocking takes place is coincident with that of the greatest activity within the tree of the normally occurring enzymes. When enzyme activity is at a minimum, blocking is much less rapid and may be negligible. A wound made at the wrong time of the year may remain unblocked for many weeks until the

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enzymes are again active. Then starch will rapidly disappear and wound gum be laid down. From the work of Swarbrick it is established that the general conditions for wourd gum deposition are :

(1) Living starch bearing tissue abutting upon dead or dying tissue.

(2) Activity of the enzyme system normal to the tree.

## GENERAL HISTOLOGICAL CHARACTERISTICS OF BROWN BAST

The observations made by Rands, pointing as they do to the occurrence in diseased tissue of this wound gum are of great interest. Rands has described the secret on which causes the discolouration typical of brown bast in the rubber tree. He observes that in diseased cases, the discolouration begins at a small point or points at the tapping cut and extends downward and laterally into the undamaged cortex towards the base of the tree. The presence of discoloured areas of this kind below the cut form the chief external characteristic of the disease. The secretion itself is a yellow viscous substance which Rands decides is produced by the living cells surrounding certain of the latex vessels, into which it often makes its way. It is a noteworthy fact that the vessels are always the centres round which the secretion of this substance takes place. Rands has observed that the rate of deposition of the viscous body is variable and is always most profuse in vigorous trees growing rapidly, while in trees spare in growth and lacking in vigour, the amount of material deposited may be negligible. In affected vessels the latex is observed to have coagulated. Areas of cortex, severely discoloured, no longer yield latex. Rands describes the nature of the secretion and finds that, when newly formed, it is plastic in nature but later it hardens and contracts and assumes a reticulated appearance. It is particularly insoluble and resistant. Fat solvents and cellulose solvents are without effect. Acids, except hot concentrated nitric acid which degrades it, are similarly incapable of effecting solution. Alkalis produce a striking darkening in colour but do not dissolve it. When newly formed, it responds to no microchemical reactions for lignin but after some time it develops this property and also gives fairly definite reactions for tannins. Sanderson and Sutcliffe have also noted independently the occurrence of a body giving tannin reactions. Rands came to the conclusion that the secretion occurring in brown bast tissue was of the same nature as the wound gum described by various investigators as occurring at wood wounds in other trees. He made further observations upon the effect of making a single cut in healthy Hevea cortex, and found that there usually appeared within 24 hours, a yellow body secreted uniformly along the wound by the living parenchyma cells beneath. This secretion was localised to a narrow zone quite close to the injured surface. The application of the various microchemical tests described led him to the opinion that the body occurring in small amounts at any such cut, was of the same nature as the wound gum occurring extensively round the vessels in cases of brown bast. He also observed that at such a single wound the appearance of wound gum was followed in a week's time by suberization and the laying down of a cork cambium behind the injured zone. In a similar manner he noted the appearance of the same substance when wounds were made in the wood of Hevea as distinct from the cortex. These observations led Rands to the final conclusion that brown bast was an accentuated type of wound response. It now seems to the writer very unfortunate that Rands was not able to suggest a mechanism to explain the phenomenon.

Rands in another paper, dealing mainly with tapping experiments, has shown that with estate trees, a greater frequency of tapping is usually productive of more cases of the disease. He also notes cases where, when

tapping was very drastic—six times a day—trees ceased to yield, the contents of the vessels were coagulated but there was practically no deposition of the gum, and the discolouration which would bring such trees under the arbitrary heading of "brown bast" trees was therefore absent. He says "with other conditions, viz. during dry weather, on defoliated trees or isolated bark areas, the tapping cut may be wholly dry, yet the secretion of gum may be so slight as not to cause noticeable browning of the cut."

The investigations of Sanderson and Sutcliffe direct special attention to another aspect of the disease. They show that very serious disorganization of the latex vessel system is brought about by the establishment of adventitious meristems in the neighbourhood of the vessels in affected parts of the cortex. The adventitious meristem, arising in the cells between the vessels, introduces new actively growing tissue into a zone in which cells are all mature. The zone enlarges as a result and the vessels which bound it are in consequence displaced and often broken. Sometimes the meristem behaves as a secondary vascular cambium cutting off wood elements to the inside and phloem elements to the outside. When this occurs, a hard woody burr is built up within the cortex. The development of such burrs may make the tree completely useless because the tapping surface becomes so irregular that tapping is impossible. These workers also note, that in bad cases of the disease, starch may very largely disappear from the cortex.

Sharples and Lambourne, as a result of extensive tapping experiments, came to the conclusion that the disease is physiological in origin and is caused by loss of vigour of the vessels as a result of tapping. They describe how bursts of the disease may occur at irregular intervals during a year and these are in the nature of a "trigger action" phenomenon. They note that, while the high yielding tree usually develops the disease most readily, the moderate and even the poor yielder may on occasion become affected.

Taylor visualizes the initiation of brown bast as a result of the death of vessels in the functional part of the cortex.

## THE RELATION OF BROWN BAST TO THE UNIVERSAL WOUND HEALING PHENOMENON

These findings of the later workers on brown bast, appear at first sight to have no definite link between them. It occurred to the writer, however, that if the conclusions of Rands could be associated definitely with the more recent work of Swarbrick, it might be possible to obtain a clear mental picture of the mechanism of the disease and one which would rationalize and bring together the views of the various investigators.

It became therefore a matter of some importance to verify the histological data of Rands and definitely to decide that brown bast gum is the same as the wound gum of Swarbrick. The writer, after carefully examining the histological characteristics of diseased Brown Bast cortex could only conclude with Rands that, in so far as it is possible by microchemical tests, to establish identities in such ill-defined, non-reactive secretions, the brown bast gum, the localised secretion at a cut in healthy cortex and the secretion at a wound, are all of the same nature and consist of wound gum. They are clearly of the same nature as the wound gum described by Swarbrick. It should be stated that wound gum is in no sense a gum, nor is it a resin, than both of which it is far more resistant. Its exact chemical nature is unknown and is likely to remain so, until a solvent is found or until some means is discovered of bringing it into solution without degrading it into substances so simple as to afford no clue to its original constitution. The term wound gum, although a misnomer, will be applied to the substance to indicate its probable identity with the body so described by the other investigators.

The writer in some additional microchemical investigations upon diseased cortex, compared in a semi-quantitative manner the soluble substances in

(a) Very severely diseased tissue,

(b) Less severely diseased parts of the same tissue,

(c) Normal tissue.

The material was all taken from the same tree at the same time. Ether, alcohol and water solubles were considered and rubber, fats, sugars, glucosides, true tannin, amino-acids and colouring matters were examined. It was found that the only products accumulating in major quantities in diseased cortex were sugars and glucosides. There appeared to be an increase in the amounts of fat, rubber, and true tannin, but the differences obtained were not such as could safely be considered significant. The only major decrease was in the amount of starch. The products which accumulate are those which normally accumulate in the neighbourhood of wounds. Starch too is the substance which Swarbrick has observed to disappear when wound gum deposition is proceeding.

Thus, brown bast tissue contains not only wound gum but also an accumulation of these soluble substances which occur at wounds. A definite connection is thus established between the disease and the phenomenon of wound healing.

## THE MECHANISM OF THE DISEASE

At any tapping cut in a healthy tree, localised secretion of wound gum is laid down. In a brown bast tree, wound gum is deposited not only along the cut, but also in greater quantity along the length of some of the vessels downward from the cut towards the base of the tree, that is, in situations where mechanical injury cannot have occurred.

Now the general conditions governing wound gum deposition, are known as a result of the work of Swarbrick and since the parenchymatous cell near an open wound, if the same conditions be realised along the length of a vessel, which are realised close to a healthy cut, then the parenchyma near a vessel must secrete wound gum as if at an open wound.

From a consideration of these general conditions it will be seen that a very simple happening will produce gumming along the length of a vessel. If a functional latex vessel dies back from the cut, downward along its length into the living tissue below, at a time when the enzyme system is active, the parenchyma surrounding the dying vessel will be in a similar state to that just behind the injured cells at a knife cut. They will secrete wound gum around the vessel and along its length, to the point where die-back has ceased and so lay down wound gum in what may be termed a "brown bast location."

Further, if by any means whatever, a die-back once started can automatically extend down the vessel, then gumming will be able to follow (given correct enzyme conditions) and a travel of discolouration along the vessel system such as is usual in brown bast cases will result. Now a cell wall at death becomes freely permeable to the cell sap of the surrounding tissue and with this in mind, the writer carried out experiments which are of direct interest. Pieces of bark, freshly removed from the tree, were subjected to the vapour of chloroform for half an hour. They were then centrifuged and the sap, so removed, tested for acidity with a capillator apparatus. It was found that this sap was frequently as acid as pH 5·2 and since it is known that rubber latex floculates at pH 5·0-5·2, the permeability of the vessel wall at a die-back at once assumes importance as providing a means by which a spread of discolouration may take place.

The entry of such a cell sap at a small die-back would bring about the coagulation of the latex and probably the cell protoplasm to a point a little further down the vessel, thus extending the dying region. It is difficult to visualize the vessel continuing to pursue its normal functions in that part of its length in which so drastic a phase reversal has taken place and in its contents have become literally a plug of solid rubber. The extension of the dying zone would allow of the entry of more cell sap, causing the pathological affection to travel still further along the vessel and this process, automatically repeated, could create a steadily lengthening path for wound gum deposition which is attendant upon the pathological or semi-pathological condition of the vessel.

Gumming once started under favourable conditions in this way, will be able to spread along the vessel system both downward and laterally, since the vessels in each ring frequently anastomose.

To somewhat unusual nature of the cortex itself thus provides a means by which a spread of discolouration is possible under favourable conditions.

Conversely and as a test of the correctness of the present view, if a die-back were induced in some vessels of a tree of little or no vigour, or in a vigorous tree temporarily reduced to an inactive condition, (as during defoliation) or in an exhausted area of cortex such as results from heavy experimental tapping of isolated panels, then with enzyme activity reduced to a minimum, the bark should on occasion become dry, with the contents of its vessels coagulated, but discolouration should be absent. There should be coagulation without gumming. The cases recorded by Rands in which this result is shown to have been obtained under just such conditions, lend support to this suggested mechanism.

A discoloured area of cortex having been established as a result of a die-back, then just as a cork cambium is laid down behind the localised secretion at an open wound, meristematic activity may also be initiated around gummed areas within the cortex, in the manner described by Sanderson and Sutcliffe. This activity, by causing the breakage and death of vessels perhaps in another row and perhaps previously functional will produce new sites for gumming and, apart altogether from the ultimate burr formation and disorganization of the cortical tissues, will act as a powerful instrument in the spread of discolouration, not only in the vessels of the same row, but as between row and row. The introduction of this latter factor completes the picture of the mechanism involved in a severe case of brown bast.

Viewed therefore as a wound healing phenomenon, the mechanism of the disease is simple. As a result of tapping, a random and occasional die-back or drying out may take place in a vessel or vessels of the tree. Subsequent events are governed in such a case by the universal conditions for wound gum formation. One of these conditions is activity of the enzyme system normal to the tree. Thus, gumming can be very rapid or very slow. This factor is closely linked with the vigour of the individual tree and is one which previous explanations of the disease have not been able to take into definite account.

It will however be seen that the mechanism agrees with and cover the findings of the various recent investigators. Sharples and Lambourne have come to the conclusion that the disease is initiated by a loss of vigour in the vessels as a result of tapping and the present explanation, in terms of the wound healing phenomenon, demands what is essentially the same thing, namely a random die-back as a result of tapping, producing a pathological condition in the vessels. Taylor also predicts the death of a vessel previous to its becoming a seat of brown bast. The subsequent laying down of wound gum covers all the observations of Rands and the findings of Sanderson and Sutcliffe form another very essential part of the

mechanism. Viewed in this way, recent investigations lose their isolation. They fall nicely together to give a much clearer picture of the disease, and the whole is definitely linked with a universal tree reaction.

## BROWN BAST AS A MANIFESTATION OF THE WOUND HEALING PHENOMENON

It is now possible to consider the incidence of Brown Bast under various conditions and such a consideration shows that some rather obscure observations, permit of a ready explanation.

When trees are in tapping, cell contents, proteins, carbohydrates and mineral salts as well as caoutchouc and water are being regularly removed from the vessels. This will either have no effect on the vigour of the vessels, or it will tend to produce a premature pathological condition. One can safely assume that, very occasionally, a vessel or two in a few of the trees will tend to die-back a little way below the injured zone at the cut into the living and undamaged parenchyma. This random and occasional occurrence produces the initial seats for brown bast discolouration near the cut. Now the regular operation of tapping, also tends to oppose the establishment of discolouration, because the excision of cortex tends to carry away small gummed areas. Tapping thus introduces opposing tendencies. Trees which develop brown bast are those in which the deposition of gum at an embryo seat of disease has been able to outpace removal by the knife. The greater the tendency of rapid gumming the greater the likelihood of the development of disease. It has however emerged that gum deposition is governed by enzyme activity so that the greater the enzyme activity within the tree the greater the predisposition to brown bast.

(*a*) *The Vigorous Tree and brown bast.*—The well-grown vigorous tree is normally in a state of greater enzyme activity than the ill-grown feeble specimen and an average estate population contains trees of all degrees of vigour. We are thus provided with a reason why the well-grown tree usually develops brown bast more readily than its more feeble fellows. In the vigorous tree, gumming at an embryo seat will always be more profuse and more liable to outpace removal by the tapping knife.

(*b*) *Bursts of Disease*.—The activity of the enzyme system of a normal tree varies in the course of a year over a considerable range. It is low when the tree is about to winter and during defoliation. It is high when the tree is refoliating after wintering and growth is actively proceeding. In the tropics there are subsidiary bursts of growth, which occur at irregular intervals. These are largely governed by climatic conditions and each burst involves a burst of enzyme activity. The reason for occurrence of sudden disease bursts after wintering and at other irregular intervals is thus immediately apparent.

(*c*) *Halts in the spread of discolouration*:—It is often noticed that a discoloured area on a tree may cease to enlarge and remain stationary for a long time after which it may quite suddenly begin to enlarge again.

When and if conditions in a tree become adverse to growth and enzyme activity, gum can no longer be laid down. The necessary pathological condition may be present in many vessels, but gum deposition cannot proceed, until improved growth conditions are re-established.

(*d*) *Frequency of tapping and incidence of disease*:—Under estate conditions, the change to a heavier tapping system, say from alternate daily to daily, is usually productive of a greater brown bast incidence.

It is apparent that the more frequently the tree is tapped and its vessels are denuded of contents the greater will be the chance of a small random die-back occurring in a few vessels of a few trees. Thus the greater the frequency of tapping the greater the chance of producing an

embryo seat of disease. But the greater the frequency of tapping, the greater the excision of cortex and the greater the chance of removing such embryo seats of disease. There are two opposing tendencies. Now it has been recorded by Rands and the record is typical, that under good conditions, discolouration has been observed to spread down a panel at the rate of one metre in less than a month. This rate is more than thirty times greater than the rate at which cortex is excised by daily tapping. It follows then that the factor of excess cortex removal will only be of significance when gumming is proceeding at low speeds. When enzymic activity is such that gumming is able to proceed even at one-tenth of this optimum speed, the excess bark removal will be powerless to prevent the establishment of gummed areas of cortex. Thus, when and where enzyme activity is reasonably high the chief factor in disease incidence will be the greater tendency to produce more embryo seats. Now on the average estate the ill-nourished tree-type usually forms only a small proportion of the whole, the majority being reasonably well grown and vigorous. In such an area, over an average year, the tendency to produce more embryo seats will in general be the more important factor. A greater tapping frequency is therefore predicted as likely to produce more cases of brown bast in the average estate population, but exact proportionality between frequency and number of cases can obviously not be expected, nor is it observed.

(*e*) *Number of cuts per inch and incidence of disease.*—A reduction in the thickness of the shaving removed at each tapping usually results in a greater incidence of brown bast.

The gumming begins from points at the cut. The rate of deposition is variable as between tree and tree and from time to time, dependent upon the vigour of the individual. Thus in trees of lesser vigour and activity, and in times of lesser activity in the more vigorous trees, that is when the thickness of a shaving can exercise any measure of control, the thicker shaving by tending to eliminate more completely any small seats of disease will in general be expected to produce fewer well established cases of disease.

(*f*) *The anomaly of the small holding.*—It is a remarkable fact that there are usually far fewer well developed cases of brown bast on the small holding than on the average estate. It is known that tapping is usually very drastic and a great number of cases would at first sight be expected, yet the actual incidence is very low. Estate managers rightly observe that if such drastic tapping systems were applied to estate trees the percentage of brown bast cases would be enormous. The apparent anomaly of the small holding is usually dismissed by saying that bark consumption is so high that it constantly removes diseased cortex. Now this explanation is not of itself sufficient, for assuming the small holder to indulge in twice daily tapping and to make as few as fifteen cuts per inch, he would then be removing cortex at the rate of not more than five inches per month on any one panel, which is less than one-seventh the rate at which discolourations have been observed to spread. While this high bark consumption cannot fail to have a great effect in removing diseased cortex, if it were the only factor operating against the disease, there would still be an appreciable brown bast incidence. A correct conception of the disease must provide a ready explanation of this apparently anomalous state of affairs.

Now it is also the fact that the average small holding contains infinitely more permanently "dry trees" than the average estate. Trees of this type, in which practically all the vessels have ceased to be functional and which must be tapped very close to the cambium in order to obtain even a small flow of latex, abound in the small holding. They show no discolouration and do not therefore come under the heading of "brown bast" trees. This fact is rarely mentioned, nor is it usual to consider it as having any relation to the brown bast problem.

The average small holding is closely planted and the individual trees are usually ill-nourished, thin, hard-barked specimens in comparison with estate trees of similar age. They are tapped heavily and the result is that vessels die-back exactly as in an estate tree. The pathological condition necessary for wound gum deposition is produced in their vessels as it would be on the estate tree but wound gum deposition cannot in the majority of cases follow. The enzymic machinery is so feeble as to be unable to produce discolouration. Their case is exactly analogous to that described by Rands for estate trees heavily tapped during defoliation, or on isolated bark panels. The cortex is dry, the contents of the vessels are coagulated but there is no discolouration. The small holding presents fewer brown bast cases, because it has so few even reasonably vigorous trees. For the same reason it presents more cases of trees which are "dry" but which might otherwise be "brown bast" trees.

## SUMMARY

Brown bast disease is a phenomenon closely related to the tree reaction of "wound healing" and obeying the same general laws.

When the disease is considered in this light, the chief findings of the various recent investigators are brought together into a harmonious whole.

A pathological condition, occasionally induced in a few vessels by the operation of tapping, is followed by the deposition of wound gum and the formation of burrs.

The deposition of wound gum is essentially an enzymatic process carried out by enzymes normal to the tree and the disease being thus enzymatic in its operation, is closely linked with the state of vigour of the individual concerned.

The linking of the disease with enzyme activity and tree vigour enables a simple explanation to be made of many obscure but fundamental points connected with its incidence.

A new light is thrown upon the relation between "brown bast trees" and "dry trees" in small holdings.