Effect of Injury on the Foliar Sclereid Development in *Fagraea fragrans*

The fact that sclerenchymatous idioblasts provide suitable experimental material to study the influence of certain factors that control plant cell differentiation has been well emphasized[1]. Certain morphogenetic factors have also been identified that would either suppress sclereid formation or alter their number and pattern of distribution[1-3]. While studying the morphogenesis of leaf sclereids in *Fagraea fragrans*, certain variations in the developmental pattern of sclereids were noticed in the injured apices of certain leaves and some of these observations are briefly discussed here.

In this species, the astrosclereids first appear in the apex of 10-day-old leaves and later extend both basipetally and acropetally towards the rest of the lamina. Other details of certain experimental studies on the sequential development of sclereids in the developing leaves are discussed elsewhere[4]. They will be present throughout the lamina in a 40-day-old leaf, and in distribution they are both diffuse and terminal. In order to study the development of sclereids in the growing leaves, the smallest visible leaf primordium was marked and tagged with the date, and the leaves that subsequently enlarged from such primordia were excised at definite intervals for clearing[5], or for microtoming and staining. The leaves in this species are elliptical, decussately opposite, entire with acuminate-cuspidate apices.

While studying the developing leaves, it was found that the apices of certain leaves (50-day-old) were wounded and damaged. 2 opposite leaves on a node, one damaged and the other undamaged, were selected, cleared and further comparatively studied. About 20 injured leaves were studied in detail.

In the apical region of 50-day-old leaves, on an average, about 54 sclereids will be present per 1.54 mm² leaf area (Figure 1), and contrastingly in the wounded leaf apex for the same leaf area only 4 sclereids are present. These are mostly restricted to the midrib region, and lamina portion is almost free from sclereids (Figure 2). A clear zone devoid

[1] D. E. Foard, Nature *184*, 1663 (1959).
[2] D. E. Foard, Pl. Physiol., Lancaster, Suppl. 33 (1958).
[3] K. H. Al-Talib and J. G. Torrey, Am. J. Bot. *118*, 71 (1961).
[4] A. N. Rao and S. J. Vaz, Phytomorphology, in press (1969).
[5] H. J. Arnott, Turtox News *37*, 192 (1959).

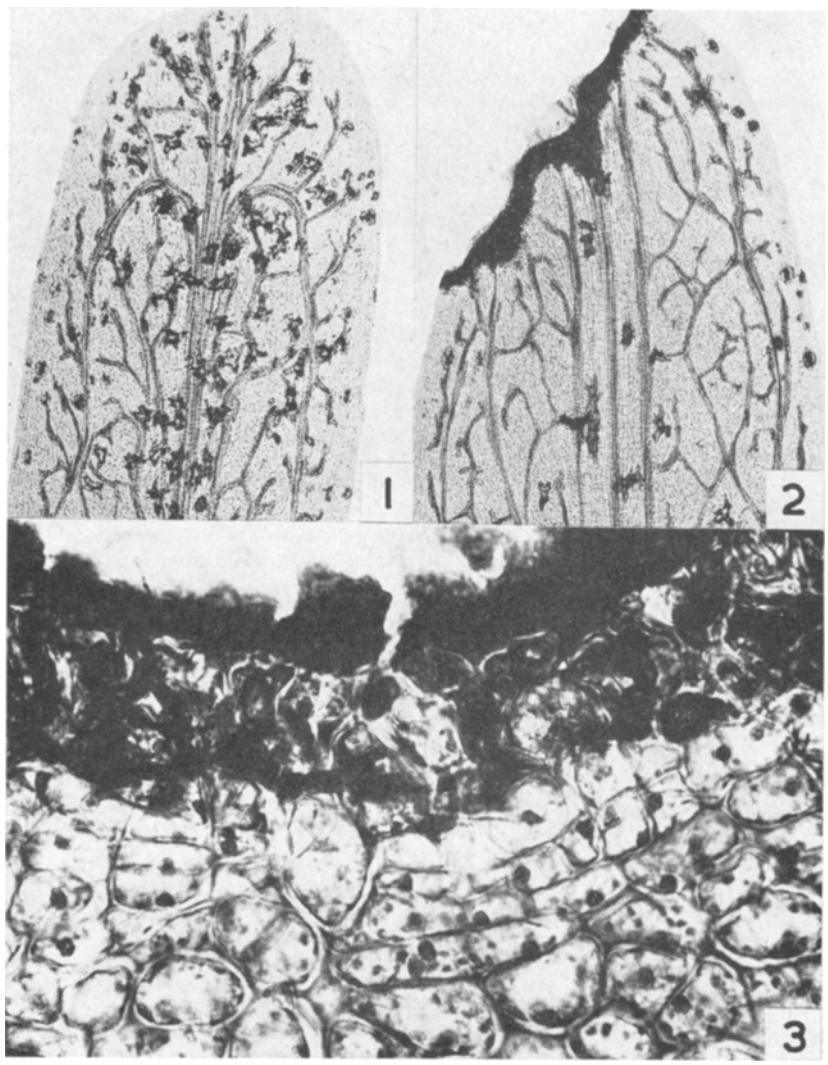

Fig. 1 and 2. Intact and damaged apices of 50-day-old leaves. Note the absence of sclereids in the lamina of injured leaf; the few present are close to the midrib. Both × 65.
Fig. 3. Section of wound region showing wound meristem and suberized cell layers. × 1080.

of sclereids develops near the wound. The sections of such damaged portions reveal the organization of a wound periderm in the subsurface layers of the damaged portions. The cells at the cut surface assume a wavy outline with broken cell walls and the plastids in them disappear; cell walls are suberized. The 3rd or 4th layer of epidermal cells situated inwards from the damaged layer of cells enlarges and divides periclinally, organizing a wound meristem (Figure 3). Some of the cells above the wound meristem and closest to the damaged surface develop into brachysclereids with extremely thick (10–16 μ) lignified walls. The brachysclereids are very much smaller in size when compared with the other epidermal or mesophyll cells. Hence it is obvious that certain cells, derived as a result of wound meristem activity develop into brachysclereids. Normally brachysclereids are not found in the lamina portion of this species, but are abundant in the petiole region. It appears that, under the influence of wound hormones, certain epidermal cells can redifferentiate into brachysclereids. Another interesting change is in the thickness of cuticle on the damaged surface. In a normal uninjured leaf, the cuticle is 6.5 μ thick, and the same on the damaged portion is 84 μ thick, and thus the newly formed cuticle as a result of wound response is very much thicker (Figure 3). Other studies on wound healing in leaves and the associated histological changes are summarized by BLOCH[6].

In *Camellia japonica* most of the sclereids are densely arranged near the leaf margin, and in the rest of the lamina they are very sparsely present[1]. When incisions are made parallel or perpendicular to the long axis of the lamina in very young leaves, the parenchyma cells near the new margins which normally would have developed into mesophyll cells differentiate into sclereids, showing a dense arrangement. In *F. fragrans*, some of the mesophyll cells which would have normally developed into sclereids remain as such without undergoing any change when they are present near the wound region. The response of mesophyll cells to wounds, when natural or artificial in these 2 cases, are fundamentally different. The wound or the wound hormone either promotes or retards the development of mesophyll cells into sclereids as in *C. japonica* and *F. fragrans* respectively. It is thus evident that the formation and developmental pattern of sclereids are under the control of certain other factors surrounding the sclereid initials, perhaps including the wound itself, activity of the wound meristem or the wound hormone as is obvious from the present studies. Other experiments conducted concomitantly with this investigation reveal the probability of hormonal control in the morphogenesis of foliar sclereids in *F. fragrans*[7,8].

Résumé. L'effet d'une blessure naturelle sur le développement du tissu sclérifié et la distribution des feuilles est étudié sur la *Fagraea fragrans*. Un méristème blessé est préparé et son histologie décrite. L'influence inhibitrice de la blessure et de l'hormone de blessure sur le développement du tissu sclérifié est très prononcée. Certains points importants de l'étude présente sont discutés, et comparés à des observations antérieures.

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[6] R. BLOCH, Bot. Rev. *7*, 110 (1941).
[7] A. N. RAO and M. SINGARAYAR, Experientia *24*, 298 (1968).
[8] I am thankful to Dr. C. R. METCALFE, Royal Botanic Gardens, Kew, England, for some helpful suggestions.

Histological Changes in the Skeletal System of the Developing Quail Embryo Treated with Sodium Salicylate

Skeletal abnormalities are frequently seen in teratological studies but only on rare occasions have histological studies on their early manifestation been carried out with a view to elucidating the mechanisms of their induction. Administration of high doses of sodium salicylate[1–10], acetyl salicylate[2,4,11–15] and phenyl salicylate[15] to pregnant rats, mice, hamsters, rabbits and guinea-pigs has been shown to induce skeletal abnormalities but no effects have been demonstrated in avian embryos. However, acetyl salicylate is known to enhance egg production in the domestic fowl[16]. In this study, the effect of sodium salicylate on the developing quail embryo was investigated both by injecting the laying hen and by treating eggs. The abnormal embryos were examined histologically and a possible mechanism of teratogenesis suggested.

20 adult hen quail were injected i.p. with a single dose of 2 ml of 0.5% aqueous sodium salicylate (buffered to pH 7.0 with dilute acetic acid), and 20 control birds were injected with 2 ml of dilute acetate buffer (pH 7.0). Approximately 100 eggs were collected from each group of birds from 1–20 days after treatment, and incubated

[1] K. S. LARSSON, H. BÖSTROM and B. ERICSON, Acta paediat., Stockh. *52*, 36 (1963).
[2] K. S. LARSSON and H. BÖSTROM, Acta paediat., Stockh. *54*, 43 (1965).
[3] K. S. LARSSON, B. ERICSON and H. BÖSTROM, Acta morph. neerl. scand. *6*, 35 (1963).
[4] J. WARKANY and E. TAKACS, Am. J. Path. *35*, 315 (1959).
[5] E. TAKACS and J. WARKANY, Teratology *1*, 109 (1968).
[6] A. S. GOLDMAN and W. C. YAKOVAC, Proc. Soc. exp. Biol. Med. *118*, 857 (1965).
[7] A. S. GOLDMAN and W. C. YAKOVAC, Archs envir. Hlth *8*, 648 (1964).
[8] K. S. LARSSON, Acta paediat., Stockh. *55*, 569 (1966).
[9] K. S. LARSSON, Acta path. microbiol. scand. *66*, 560 (1966).
[10] K. S. LARSSON, Br. med. J. *105*, 352 (1963).
[11] H. J. K. OBBINK and L. M. DALDERUP, Lancet *565* (1964).
[12] D. G. TRANSLER, Lancet *606* (1965).
[13] A. V. JACKSON, J. path. Bact. *60*, 587 (1948).
[14] J. D. MCCOLL, M. GLOBUS and S. ROBINSON, Toxic. appl. Pharmac. *7*, 409 (1965).
[15] T. BABA, Osaka Cy med. J. *12*, 23 (1966).
[16] B. L. REID, A. A. KURNICK, J. M. THOMAS and B. J. HULETT, Poult. Sci. *43*, 880 (1963).