strains on shrubs, two on furze flowers and one on myrtle leaves. Van Uden *et al.* did not consider that plant materials were a natural habitat for *C. albicans*, but thought that in this situation the pathogen was a contaminant with prolonged powers of survival. All evidence in my past and present findings suggests that *C. albicans* is not a true member of the phyllosphere flora. Yeasts from this habitat are almost invariably non-fermenting and are either mucoid in consistency or pigmented. Sources of contamination are common in New Zealand, and Parle[3] found *C. albicans* in the alimentary tracts of sheep, dogs, hedgehogs and Australian opossums, all of which had access to the area from which the grass sample was taken.

It now seems probable that *Candida albicans* occurs commonly on vegetation, for surveys of yeasts from this habitat are made infrequently. Even if this is so, it is unlikely that contaminated grass is a hazard to public health. Up to 50 per cent of normal humans carry the yeast in their alimentary tracts[4] and exposure to it, in small numbers, must be a daily experience. Clinical and experimental studies suggest that host factors, such as disturbance of the normal flora or weakening of body defences, may play a part in causing *C. albicans* to behave as a pathogen rather than as a saprophyte.

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M. E. di Menna

Soil Bureau,
Department of Scientific and
Industrial Research,
Wellington, New Zealand. March 6.

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Transmission of Juvenile Rooting Ability from Seedlings to Adults of *Hevea brasiliensis*

The Para rubber tree, *Hevea brasiliensis* (Willd. ex A. Juss.), is notoriously difficult to propagate from cuttings. Previous reports[1] have shown that cuttings from the bases of young trees root readily, whereas cuttings from the branches of mature trees do not. The formation of adventitious shoots in prone trees and the rooting of cuttings from these shoots has been described[2].

As Robbins[3] has pointed out, grafting of adult stems on seedlings of many different kinds of plants has induced a juvenile type of growth in the adult scion. Attempts were made to induce juvenility and thereby enhance rooting ability in *Hevea* by grafting buds from a mature *Hevea* clone (Tjirandji 1) on seedling plants.

Buds were taken from branches of 8–10-year-old trees and grafted at the base of stems of 12-month-old seedlings. The method of budding has been previously described[4]. When the bud had sprouted and grown 3–5 ft., cuttings were made and planted under a mist spray. At the same time, buds were taken from the sprouts and grafted to other seedlings. This sequence of budding and planting cuttings was repeated four times. 30 per cent of the cuttings from scions of the fourth and fifth graftings formed roots in about eight weeks after planting under the spray, but cuttings from the original clone and from the first, second and third graftings failed to root.

Fresh basal sections from seedling stems were ground and extracted with water, alcohol or ether. These extracts were used to treat other cuttings and air-layers but failed to improve the rooting ability of the adult plant.

These results are further evidence that the juvenile form may transmit a substance to the adult which induces the adult to assume juvenile characteristics. They suggest that if a substance is actually transmitted from stock to scion that this material is not readily soluble in ordinary solvents, but that it may be slowly absorbed and accumulated by the scion in sufficient amounts to induce a change in rooting ability.

T. J. Muzik*
H. J. Cruzado

Federal Experimental Station,
Mayaguez, Puerto Rico. Feb. 25.

* Present address: Department of Agronomy, Washington State College, Pullman, Washington.

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Ion Permeability of the Plasmalemma of the Plant Cell

In an earlier communication[1], I advanced evidence for the existence, in *Nitella*, of a plasmalemma with limited ion permeability. This evidence has been questioned by Prof. G. E. Briggs[2].

He suggests a model *Nitella* cell in which the tonoplast is the only membrane, and says that this model is consistent with the results of my d.c. resistance measurements. He argues for his model that "if the tonoplast is practically impermeable to cations, then the conductivity of the cytoplasm . . . will depend on the concentration and mobility of anions in (this phase)". This is clearly incorrect. In such a model the cytoplasm would have a very high conductivity (it would contain 0·8 *M* cations if univalent). This high conductivity would not be modified by the presence of a tonoplast impermeable to cations. The flow of current would, on Prof. Briggs's model, produce a large potential gradient across the tonoplast and a small potential gradient across the cytoplasm. His model is therefore inconsistent with the results, as is any model containing a tonoplast alone. A structure responsible for the greater part of the d.c. resistance of the cells must lie between the bathing medium and the inner cytoplasm. It is difficult to doubt that this is the plasmalemma.

With reference to the determinations of calcium activity, the pond water used was filtered. Similar results were also obtained on cells in calcium chloride solutions. The method (including the use of flowing external solution) was as previously reported[3]. The potential difference between the cytoplasm and the pathing medium (E_{co}) was measured, and the calcium chloride concentration ($[Ca^{++}]_o$) in the latter was known.

E_{co} measured	−140 mV.	−144 mV.	−137 mV.
a_oCa^{++} known (≒ $[Ca^{++}]_o$)	0·5 m*M*	0·5 m*M*	0·05 m*M*
a_cCa^{++} calculated if there is Ca^{++} equilibrium	34 *M*	46 *M*	2·7 *M*
Time allowed for equilibrium	20 min.	22 hr.	2 hr.

Here, as previously[1], a_cCa^{++} is calculated from the equation (wrongly given in my earlier communication):