mis exhibited no specific response. By the end of 72 hours, however, the cells of the pericycle, endodermis, and inner cortex, in many cases, showed divisions in the radial as well as in the tangential plane (Fig. 2, top). Pericycle cells were especially active, producing a narrow zone of meristematic tissue, which extended well back from the apical region. Active cell division within the pericycle continued until, eventually, the boundary between this tissue and the adjacent tissues became indistinguishable. Because of this active cell division, the pericycle produced a conspicuous mass of dense meristematic tissue, which extended outward toward the periphery (Fig. 2, bottom). The cells of the outer cortex at the basal portion of the root began to collapse during the third day.

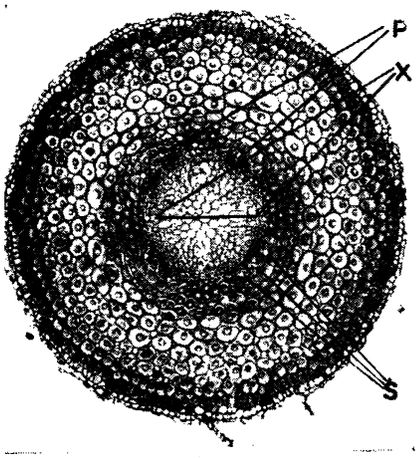

Fig. 1. Transverse section of soybean primary root taken 996 μ behind the stelar initials. *P*, pericycle; *S*, sieve tube; *X*, primary xylem (× 79).

Fig. 2. Transverse sections of soybean primary root, 3 days after treatment with 2,4-D. (Top) Transverse section taken 924 μ behind the stelar initials, showing proliferation of inner cortex, endodermis, and pericycle. (Bottom) Transverse section at the basal region of the primary root showing a conspicuous mass of proliferated tissue produced by the pericycle. The boundaries of pericycle, endodermis, and cortical parenchyma have become indistinguishable. The indented outline suggests the formation of numerous fasciated lateral roots (× 68).

The primary phloem also exhibited a similar response to this treatment. At a distance of about 380 μ from the tip, the nonconducting cells of the primary phloem became meristematic. At a greater distance (about 1640 μ from the tip), even greater meristematic activity was apparent. Here the primary phloem, with the exception of the sieve-tube elements, became indistinguishable from the adjoining areas.

Close to the apex there was no response in the primary xylem. Some dividing cells, however, were observed in the metaxylem in the basal regions.

The striking effect of 2,4-D on the root of soybean is a stimulation of cells in certain tissues to high meristematic activity. The resulting mass of cells is nonpolarized and does not seem to be made up of numerous, closely placed lateral-root primordia as has been suggested by Wilde (*3*). Apparently 2,4-D retards root elongation and disrupts the orderly biochemical and physiological processes of normal development, particularly within the stele and inner cortex, resulting in a change to high meristematic activity within these regions.

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References and Notes

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18 January 1956

Disease in the Giant African Snail Achatina fulica Bowdich

Between March and December 1954, an investigation was conducted to determine the causes for the recent sharp decline in Ceylon populations of the giant African snail (*Achatina fulica* Bowdich) (*1*). One of the most striking aspects of the field research was that in some areas, even after hours of collecting and examining individuals, the oldest live specimens were found to be no more than 1.5 years old.

In many populations, the oldest live specimens were between 2 and 3 years of age, although dead specimens in excess of 5 to 6 years of age were almost countless. In other areas, long hours of diligent hunting failed to bring to light a single live specimen in spite of the fact that 2 to 3 years earlier the place had been overrun with snails.

The last sudden decline in the snail populations in Ceylon took place in 1952, a year during which the large stores of metaldehyde that had been purchased as a result of numerous outbreaks of snails in 1951 were scarcely touched. This reflects a cataclysmic decimation, and perhaps even a near or actual local extermination, of the population. The uniformity of age and the conspicuous absence of older individuals suggest that the present populations are the surviving offspring of those subjected to this catastrophic force. As had earlier been anticipated (*2*), the picture in general was found to be not that of predation but of parasitism or pathogenesis with variable, localized predation.

Of the several predators, only the firefly *Lamprophorus tenebrosus* appeared to offer any possibility as an agent in the biological control of the giant snail. In one area near Pallekelle in the Central Province, however, the fireflies and achatinas had been together for a minimum of 25 years, and the snails could still be classed as "common to abundant" in spite of reasonably common fireflies. Further, it was established both in the laboratory and in the field that the larger the giant snail, the less likely it was to be attacked and killed by the glowworm larva. Therefore, predation by glowworms cannot explain the complete absence of older individuals in some of the erstwhile large, vigorous populations.

In seeking a cause for the manifest sharp decline in the populations of the giant African snail in Ceylon, I found that the greatest amount of evidence pointed toward the existence of a nonspecific, chronic disease of uncertain etiology. In the many different environments examined, the only discernible common factor that was unfavorable to the survival of the snails was a fairly constant syndrome. The most conspicuous symptom of this syndrome was the presence of leukodermic lesions on the fore part of the body (Fig. 1).

The first sign of pathogenesis is the presence of vague, patchy, granular areas on the tentacles, face, and neck. Microscopic examination reveals that the melanophores in the dermis of these areas are undergoing complete distintegration and disappearance. Concomitantly, there is a proliferation of dermal connective tissue cells. The epidermis, however, remains intact. Hence, there is no appearance of frank ulceration. But the epidermis reflects the dermal disturbances by

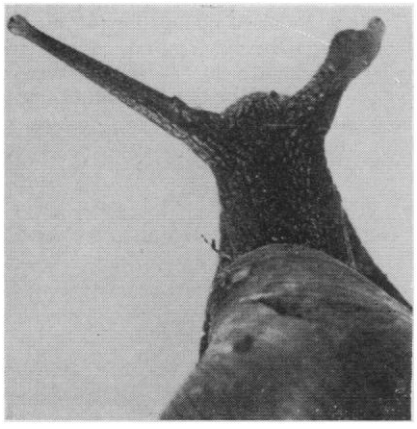

Fig. 1. *Achatina fulica.* The right tentacle has become distorted and shortened as a result of pathologic tissue alterations in the dermis. Small, initial, melanotic tubercles appear basally on the left tentacle and on the face.

elevating into verrucous ridges, "horns," and tuberculations, eventually allowing a serious distortion of the tentacle. As the leukodermic lesions enlarge and coalesce, the shortening tentacle remains partially invaginated and ultimately completely invaginated. In some cases, the primary lesion is initially melanotic, but it becomes melanoleukodermic or leukodermic as melanolysis continues. Evidence shows that there is some slow regeneration of damaged tissue; but the infection spreads to other areas of the body.

Histopathologic examination of the hepatopancreas ("liver") and kidney disclosed marked tissue changes that are the result of either toxemia or the direct action of the pathogen. Other organs doubtless will be found also to be involved in the visceral phase of the disease. Any pathology in the reproductive tract naturally would be significant both in its possible effect upon fecundity (and therefore the size of the population) and in a possible transovarian mode of transmission.

The incidence of the disease in the populations of the giant snail in Ceylon is high, varying approximately from 35 percent to 60 percent. Experiments suggest that the incubation period is reasonably short; that the disease is highly contagious; that, at least initially, there is relatively little natural immunity to the disease; and that contact is one of the modes of transmission. This latter point has implicated the ubiquitous phorid flies (*Spinophora* sp.) as possible mechanical or even cyclical vectors and has implied a direct correlation between population density and incidence of the disease.

The epizootiologic picture is that of a chronic, endemic disease with low gastropod host specificity. Typical symptoms were found in all endemic snails examined in any quantity. Since the disease was found in populations of native snails far from the frontier of invasion of the giant snail, the inference is that the disease was in Ceylon before the arrival of *A. fulica.* But until the nature of transmission is completely understood, an antithetic hypothesis cannot be ruled out as a possibility.

Without exception, every population of the giant snail in Ceylon which was examined not only had diseased individuals, but had many of them. The high incidence of the disease apparently has assured infectivity in specimens introduced into uninfested areas. It therefore was not surprising to find the disease present in achatinas in Singapore (66 percent of 33 specimens) and Hong Kong (40 percent of 63 specimens), nor is it difficult to predict that it will be found in Indonesia, Formosa, and the Trust Territory of the Pacific Islands. It is significant that a decline in the population has been reported from all of these areas.

The incidence of the disease in the Hawaiian specimens is appreciably lower (10 to 23 percent), and this quite possibly reflects the fact that the population is still in the early stage of invasion. The slow development of the population undoubtedly is due entirely to the limiting effects of rigorous control measures that have been carried out by the Hawaiian government since 1938. But the recent extension of the range of *A. fulica* in Hawaii indicates that there is a transition in progress which may result in a full blossoming of the snail pest within the next few years, despite efforts to prevent it. Any prognostication might well be based on the history of the giant snail population in Hong Kong. This population grew to prodigious size despite control measures and the presence of the disease, but it is now in a definite decline, and there are only occasional reports of localized buildup and damage.

Because of the lack of control specimens in Ceylon, the causative agent of the disease could not be identified with certainty. However, culture plates and tissue smears and slides indicated that it is unlikely that the disease is caused by a spirochete, protozoan, yeast, or fungus. This leaves essentially the bacteria and viruses. A coccus form was frequently isolated, but it may have been a contaminant. Inoculation experiments are currently being planned to determine positively the etiologic agent. Both field and laboratory work have eliminated the possibility that the symptoms are simply physical or physiologic reactions to unfavorable environmental factors (*3*).

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References and Notes

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3. A paper concerned with the details of the field and laboratory investigations and especially with the implications in biological control is in preparation.

13 December 1955

Ivory-Nut Palm from Late Tertiary of Ecuador

During his geologic explorations in South America, A. A. Olsson, of Coral Gables, Fla., has, on several occasions, had the good fortune to find interesting and significant fossil plants. In 1945, for example, he collected well-preserved, carbonized walnuts from marine outcrops near Esmeraldas, Ecuador, which I described as *Juglans linki* (*1*). In 1955, about 10 mi east of the walnut locality but at about the same stratigraphic level, he found a fossil tagua or ivory-nut, (*Phytelephas olssoni* Brown, n. sp.).

This fossil nut (Fig. 1) is similar to those produced by species of the palm genus *Phytelephas,* rounded-angular, 4.5 cm long, 4 cm in diameter, showing two flattened faces whose narrowed ends converge toward an apical depression, the raphe. The surface is lined by more or less irregular, shallow, veinlike, interconnecting furrows.

The fossil is a sandstone cast, most probably the filling of the thin and brittle endocarp covering the nut. It was taken from the Punta Gorda formation of late Miocene or early Pliocene age that crops out at Quebrada Camarones. Apparently the fleshy part of the original nut either decayed or was eaten out by marine organisms, permitting sand to fill the resulting cavity. The granules of the

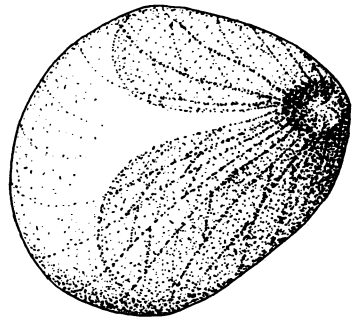

Fig. 1. Sandstone cast of a late Tertiary ivory-nut, *Phytelephas olssoni* Brown, n. sp. (natural size.)