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.

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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

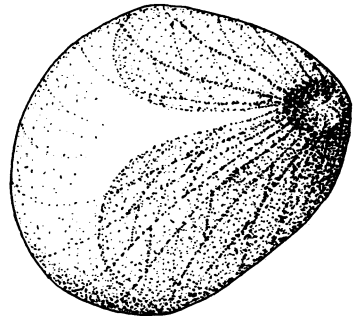

Fig. 1. Sandstone cast of a late Tertiary ivory-nut, *Phytelephas olssoni* Brown, n. sp. (natural size.)

cast include quartz, glauconite, and numerous foraminifers, with a clay binder. The living nut very likely came from a plant that grew on or near the coast and was washed into fairly deep water, as the kinds of globigerinid foraminifers seem to indicate.

The nuts of *Phytelephas* palms are at first relatively soft and edible but on ripening become very hard, with an ivorylike endosperm, and are enclosed in a thin skin or endocarp. Four or more of these covered nuts, closely pressed together (hence the angular faces) make a burlike or warty cluster. They supply much of the "vegetable ivory" of commerce from which buttons and other articles are turned.

The species of *Phytelephas,* variously estimated from 3 to 15 in number, include low, erect or prostrate plants with graceful, arching, pinnate leaves that are sometimes 15 to 20 ft long. The commonest species is *P. macrocarpa* Ruiz and Pavon, which is widely esteemed and cultivated. The genus inhabits tropical America, and the species grow along streams and swamps, generally in coastal situations.

The fossil record of the palms dates back certainly to the early Jurassic, as exemplified by impressions of fan-shaped leaves from Normandy, and perhaps to the Triassic, if the palmlike leaves I have described (*2*) from southwestern Colorado are indeed palms and not some other monocotyledon. The only other record of fossil *Phytelephas,* however, is part of a stem from the Miocene of Antigua, described as *P. sewardi* Kaul (*3*), and now in the Natural History Museum, South Kensington, London.

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Effect of Gibberellin on Elongation, Water Uptake, and Respiration of Pea-Stem Sections

Gibberellin, which induces hyperelongation in many kinds of green plants (*1*), has been shown (*2*) to be a type of substance that has growth effects different from those of auxin. It reduces the growth inhibition brought about by concentrated solutions of auxin and by some other growth inhibitors, such as maleic hydrazide (*3*). It exerts no effect on root growth but inhibits root formation (*3*).

Preliminary to a study of the mechanism of action of gibberellin, its effects on elongation, on water uptake, and on respiration of pea-stem sections were examined (*4*).

Stem sections 5 mm in length were prepared from the third internodes, which were about 15 to 20 mm in length, of etiolated pea seedlings that had been grown for 7 days in a darkroom at 25°C. The sections were set afloat in 0.03*M* phosphate buffer solutions at *p*H 7.0, with and without addition of crystalline gibberellin A (*5*), and were kept in the same darkroom until the experiments were finished.

As is shown in Table 1, pea-stem sections elongated in acid solution, either with or without gibberellin. If the elongation of gibberellin-treated sections is compared with that of the controls at the same *p*H, it may be seen that the effect of gibberellin is most pronounced at *p*H 7.0.

Water uptake was determined by the increase in 24 hours in fresh weight of ten stem sections. Just as in the case of elongation, water uptake was higher in acid solution, and the effect of gibberellin was the greatest at *p*H 7.0 (Table 2).

The oxygen uptake of freshly prepared pea-stem sections was measured, using the Warburg respirometer, which was shaken at 80 rev/min at 30°C in the dark. Each flask contained ten sections, bathed in 2 ml of 0.03*M* phosphate buffer solution, with or without 10 mg/lit of gibberellin. Experiments ran for 4.5 hr; they were preceded by a ½-hour equilibration period. The results, presented in Table 3, show that the oxygen uptake of stem sections stimulated by gibberellin was about 15 percent greater than that of the controls. When the sections were pretreated by 10 mg/lit of gibberellin for 8 hours, their oxygen uptake was about 20 percent higher than that of controls, other conditions being the same.

Table 1. Effect of gibberellin (10 mg/lit) and *p*H on the elongation of pea-stem sections in 24 hours. Initial length, 5 mm. Each value is the average of ten sections.

*p*H	Increase in length of section (mm)		Increase over control (%)
	Control	Gibberellin	
5.3	1.11	1.71	54
6.0	1.02	1.63	60
7.0	0.74	1.30	76
8.0	0.66	0.97	47

Table 2. Effect of gibberellin (10 mg/lit) and *p*H on the water uptake of pea-stem sections. Each value is the average of values obtained in three experiments.

*p*H	Water uptake (% of initial fresh wt.)		Increase over control (%)
	Control	Gibberellin	
5.3	42	57	36
6.0	49	69	41
7.0	25	40	60
8.3	31	36	16

Table 3. Effect of gibberellin (10 mg/lit) on the respiration of pea-stem sections.

Expt. No.	O_2 uptake [μlit/10 mg (dry wt) hr]		Increase over control (%)
	Control	Gibberellin	
1	52.0	59.6	15
2	51.3	59.2	16
3	45.3	52.2	15
Average			15

The effect of gibberellin in stimulating respiration was not observed with stem sections from the first and second internodes.

Although gibberellin does not cause the inward curvature of split pea stem as do auxins (*2*), it does increase the elongation and the water absorption of the unsplit material. The essential difference in the physiological effects of gibberellin and auxin is not yet known. Some clue to this difference may be found when the effects of enzyme inhibitors on the gibberellin-stimulated respiration are studied and compared with the effects described for auxin-induced respiration.

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They know enough who know how to learn.—HENRY ADAMS.