

Varietal name	Received through	Year	Introduction No.	References
Sheung Shu Wai, "President of the Board's Embrace"	G. W. Groff	1920	S.P.I. No. 51469	Groff (3): pp. 96, 101, 143.
T'im Ngam, "Sweet Cliff"	G. W. Groff Coconut Grove	1920 1943	S.P.I. No. 51471 S.P.I. No. 145647	Groff (3): pp. 144.
Tai Tso "Large Crop"	G. W. Groff	1931	S.P.I. No. 94257	Groff (3): pp. 53, 94, 95, 97, 101, 144, 167; Pl. XIX
Wai Chi "Wai River Lychee"	G. W. Groff G. W. Groff C. O. Levine G. W. Groff G. W. Groff	1915 1916 1917 1918 1917	S.P.I. No. 41054 S.P.I. No. 43284 S.P.I. No. 45624 S.P.I. No. 46569 H.A.E.S. No. 3881	Groff (3): pp. 46, 51, 64, 73, 79, 89, 92, 96, 97, 98, 101, 144, 165, Pl. XI, pp. 166; Pl. XII, pp. 167; Pl. XVII, pp. 170, Pl. XXXVI.

SUMMARY

In this paper we present techniques used in description of lychee varieties. The important questions of heritable genetic characters and the nature and development of the aril are briefly mentioned but left for detailed discussion in a future paper. We have compiled an alphabetical list of lychee varieties in order to indicate those that should be of importance in lychee importations, marking with an asterisk the ones growing at present in Florida.

LITERATURE CITED

1. Chen, Wen-Hsun. The Culture of the Lychee. Proc. Fla. State Hort. Soc., 1949.
2. Chandler, Wm. H. Evergreen Orchards. Lea and Febiger, Philadelphia. 1950. (See Chapter 13.)
3. Groff, G. Weidman. The Lychee and Lungan. Orange Judd Co., New York. 1921.

4. Some Ecological Factors Involved in Successful Lychee Culture. Proc. Fla. State Hort. Soc., 1943.

5. Additional Notes on the History of the Brewster Lychee. Proc. Fla. State Hort. Soc., 1948.

6. Hayes, W. B. Fruit Growing in India. Kitabistan, Allahabad, India. 1945 (See Chapter 18.)

7. Li, Lai-Yung and Chu-Ying, Chou. Notes on the Chen-Tze Lychee of Henghwa, Fukien, China. Proc. Fla. State Hort. Soc., 1948.

8. Marloth, Raimund H. The Litchi in South Africa. Dept. Agric., Subtropical Horticultural Research Station, Nelspruit, East Transvaal, South Africa. 1947.

9. Radtkofer, L. Sapindaceae, in Engler, A. and Diels, L., Das Pflanzenreich, 98 (IV), pp. 1-1539. Liepzig, Germany. 1931-34.

10. Bull. Bur. Pl. Ind. U.S. Dept. Agric. Seeds and Plants Imported. Inv. Nos. 15, 16, 19, 28, 31, 32, 36, 40, 43, 44, 48, 52, 53, 55, 56, 59, 60, 66, 79, 87, 88, 89, 91, 92, 95, 100, 105, 106, 107, 108, 109, 120, 121, 122, 124, 125, 139, 146, 148, 150, 151, 1909-41.

11. Vavilov, N. I. The Origin, Variation, Immunity and Breeding of Cultivated Plants. Chronica Botanica 13, No. 1-6. 1951.

12. Wulff, E.V. An Introduction to Historical Plant Geography. The Chronica Botanica Co., Welthan, Mass. 1943.

A STUDY OF THE EDIBILITY OF AKEE (*BLIGHIA SAPIDA*) FRUIT OF FLORIDA¹

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Coral Gables

Akee fruit grows on the tree, *Blighia sapida*, which is found commonly in the dooryards of the West Indies and to a limited extent in South Florida. The whitish, fleshy aril, when sautéed, forms a delicious protein supplement to the diet and in the islands it is often boiled with fish. Although the fruit is commonly used in such places as Jamaica it can not be denied that cases of poisoning have followed the consumption of this article. In Jamaica, a considerable number of deaths each year are at-

tributed to the eating of this fruit. Stories of this poisoning have caused many people, especially in South Florida, to eschew the use of the fruit. Some of the early writers attributed this poisoning to the use of over-ripe or decaying fruit. However, it has since been observed and commented upon that this poisoning can be just as lethal when unripe fruit is eaten. Most of these writers are of the prevailing opinion that mature ripe fruit is harmless and delicious. It would be of value to citizens of South Florida, as well as to the citizens of the Islands south of us, to have some definite data as to when to eat the Akee and when not. It would also be of value to our armed services who have personnel stationed in these areas to learn something more about this fruit and perhaps develop a treatment for its poisonous effects. To that end these investigations have been initiated.

The Akee, *Blighia sapida*, Kon. (*Cupania sapida*, Voight) is a member of the Sapindaceae family. The generic name is in honor of

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Capt. William Bligh, of H.M.S. *Bounty*, who is remembered more in history for the famous mutiny on his ship than for the part he played in bringing the bread-fruit (*Artocarpus incisa*, Linn. f.) to the West Indies (1). The Akee is a native to the Guinea coast of West Africa but is now commonly grown in the West Indies, Tropical America and Florida as a dooryard tree. The tree is small to thirty feet in height, erect in habit with an open crown and stiff branches. The leaves are large, shiny green, stiff, pinnate, with 3 to 4 pairs of short petioled leaflets 4 to 6 inches long (Fig. 1). The

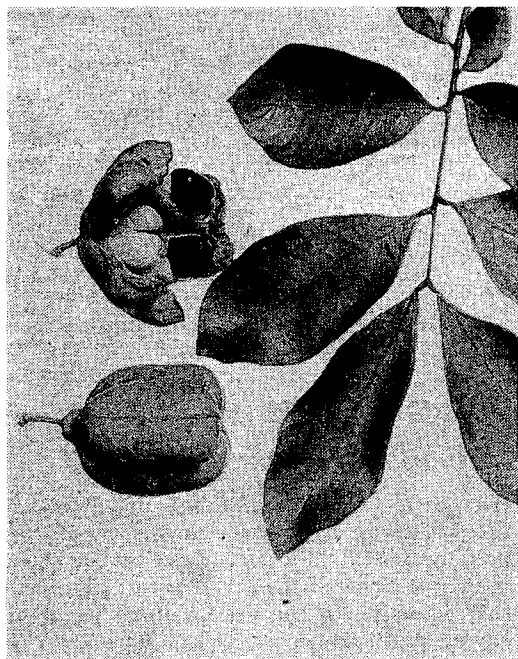


FIG. 1. Mature Akee—Fruit and leaf.

pubescent, polygamous flowers are born on racemes in the axils of the leaves. They have a calyx consisting of five pieces; five white petals bearing a large two-lobed scale near the base on their inside; eight stamens; and a short style bearing three stigmas. The flowers are so fragrant as to deserve distilling. Heaviest blossoming occurs in early spring but usually two or more blooms appear during the year, the additional ones coming in the summer and fall.

The fruit (Fig. 1) is a 3 celled capsule, 3 to 4 inches in length and yellowish to red in color. When mature it splits longitudinally (Fig. 1) exposing the round, shiny, black seed,

one in each cell, seated upon and partly immersed in a white fleshy substance called the aril. The Spanish name (2, 3), "seso vegetal" (vegetable brain) is also significant of its shape and taste for it resembles the brain of a small animal. The aril is firm, oily in texture and has a somewhat nutty flavor. In Tropical America, South Florida, and especially in Jamaica, the aril of the Akee is valued as a richly flavored and wholesome food (4). When tenderized by boiling in salt water and fried in butter it is delicious as a protein supplement to the diet.

In the brief history of akee poisoning which follows, it will be evident there is a considerable amount of information which is correct and some which is of a questionable nature. Due to the diversity of these reports, it was considered essential to institute further investigations on this rather ornamental tree. It is being introduced into the South Florida area and at least some of the nurseries sell the trees with no statement of the possible toxicity of the fruit thereof.

Seidlein (5) in his study of 62 cases of vomiting sickness in Jamaica mentions the venous congestion and hyperemia often observed in the various organs of the body of the people dying from akee poisoning. He also states that the incidence of the disease is largely from November to March and primarily affects the country people. His suggestions for the prevention of this disease are hygienic principles, relief of poverty, etc.

Scott (6) investigated the vomiting sickness of Jamaica which rages during the cooler months of the year. He reports that in this disease, there is usually no fever but the mortality is rather high, 80%. Often there are convulsions which may be followed by coma and then death supervenes. The pathological findings show that the heart presents no abnormality, but the lungs may show a slight catarrhal condition. The peritoneum appears normal and contains no fluid. Microscopic examination of the various organs show that the heart tissue shows fragmentation; the lung tissue shows general congestion and some of the alveoli may be filled with either or both red and white cells. The stomach, duodenum, liver and kidney show hyperemia. This author (Scott) administered arils of unopened akees to kittens which resulted in the death of these animals. The kidneys were congested and some

of the lung tissue showed infarcts. The liver showed general engorgement and the kidneys were hyperemic. The lungs showed intense congestion and the heart muscle showed fragmentation of fibers.

Jordan and Burrows (7) believe that the akee poison is a glucoside and that it is present in the seeds and pods of both ripe and unripe akees. The arils and placenta do not contain this poisonous substance. Evans and Arnold (8) report that arils of the unopened akees are lethal to guinea pigs in a dosage of 3.5 grams per 100 grams of body weight given either subcutaneously or intravenously. They also state that the arils of the open akee with embedded seed is toxic, but that the arils of the open akee with the normally exposed seed is non-toxic. According to these investigators, the akee contains, in the early stages of the development of the fruit, a saponin which is hemolytic. They believe that the phytosterol fixes the saponin and renders it non-toxic.

Arnold (9, 10 & 11) reported 273 cases of vomiting sickness of which 107 died. Arnold believes the toxin present is a saponin the amount of which varies inversely with the development of the fruit. He believes that it is present in larger quantities in the arils with small seeds and that cholesterol or phytosterol is an antidote for the poison. Arnold also states that the arils with immature seeds are more toxic than those with mature seeds and that the arils from unopened akees are toxic but that exposure to the air either reduces or causes a complete loss of toxicity. He believes that akees picked unopened and left in sunlight are not toxic if allowed to "yawn" and that treatment of individual poisoning by the akee should be correction of the dehydration and the induced acidosis. Arnold summarizes his findings in the various parishes of Jamaica by concluding that I. Akee is the principal cause of vomiting sickness, II. Only the fully opened large seeded and good akees should be eaten, III. Akees with small seeds are the most dangerous and should not be eaten and IV. The akee pot water should not be utilized.

In our experimental studies of akee poisoning, we have used 82 rats, 7 dogs, 5 monkeys and 65 rabbits. These animals have received varying amounts of the arils, the whole seed or the red membrane from both ripe or unripe akees. We have also used the arils from the fruit which has been tree ripened, i.e.,

"yawned" in situ as well as the arils from fruit which has yawned after picking. We have also used the arils which had aborted seeds.

In the preparation of these various portions of the akee, we have carefully separated the seed or arils, then cooked the material in boiling distilled water for about 15 minutes, homogenized the material in the cooked water and added sufficient water to make it a definite volume (5%, 10% or 20%) before administration. In some few cases, we have discarded the cooking water and fried the arils in a fresh commercial cooking fat. All of our material has been administered to the animals either intragastrically by means of a stomach tube or in the case of the monkey, the material was given on either bread or banana.

In our preliminary experiments our findings seem to show that: The arils of the yawning akee are non-toxic when administered to rabbits, dogs or rats. The seed of either the ripe or unripe akee is definitely toxic. The aril of non-yawning akee is decidedly toxic to the rabbit. The rat is more resistant to the poisonous principle or principles of the unripe arils than the rabbit. The dogs and monkeys also show some toxicity from the unripe arils.

The rabbit shows the following symptoms from the administration of the unripe akee. The animal in about 24 hours seems to be depressed and the respiration becomes rather rapid and shallow. Within an hour or so of the rapid shallow respiration, the animal collapses. The respirations continue to be shallow and rapid and then in an hour or so the respiration ceases and the animal dies.

The following tables probably will give a clearer resumé of our results: Table 1 shows

TABLE 1. AKEE ADMINISTRATION TO RATS AND RABBITS			
RATS			
Material administered	No. animals	Deaths	Survivals
Arils, "non-yawners"	24	5	19
Arils "yawners"	13	0	13
Cotyledon	19	4	15
RABBITS			
Material administered	No. animals	Deaths	Survivals
Arils, "non-yawners"	19	11	8
Arils, "yawners"	7	0	7
Cotyledon	6	6	0

the results using rats and rabbits as the test animals. The results demonstrate that in these animals the non-yawning arils and also the cotyledons have some toxic effects. Though akee poisoning is sometimes referred to as vomiting sickness, no mention is made of this effect in this table because rodents cannot

TABLE 2.
AKEE ADMINISTRATION TO MONKEYS AND DOGS

MONKEYS			
Material administered	No. animals	Emesis	Deaths
Arils, "non-yawners"	7	2	4
Arils, "yawners"	2	0	0
Seed, whole "non-yawners"	1	0	1
DOGS			
Material administered	No. animals	Emesis	Deaths
Arils, "non-yawners"	5	4	0
Arils, "yawners"	4	0	0
Seed, whole "non-yawners"	---	---	---

vomit. The results produced in the monkeys and dogs, Table 2, are indicative of similar effects to those produced in rodents. We realize that we have not tested a sufficient number of either monkeys or dogs to report any conclusive results, but they do show that the akee has some deleterious action in both.

Because we noticed that animals poisoned by the akee were cold to the touch we decided to determine the effect of repeated administration on a group of six rats. These animals received 5cc. of a 10% suspension of the arils of unopened fruit per kgm. of body weight at three successive times, the first immediately after the normal temperature, the second after the P.M. temperature on the first day and the third after the A.M. temperature on the second day. The body temperatures are recorded in Table 3. Five of these 6 rats died. From ei-

friable. The kidneys are usually rather pale. The large intestine, spleen, pancreas, and the voluntary muscles appear to be normal.

A few studies have been made on the ratio of the solids of the blood to the plasma, on the blood chloride content and on changes in the red and white cell count of the blood of animals receiving various portions of the akee. We do not have enough data to warrant a report on these phases of the problem at the present time.

CONCLUSIONS

1. The arils of the "yawning" akee are non-toxic. (This is not a surprise to us as we have found in casual conversations that a large number of our associates and acquaintances consider the arils of the ripe akee a delicacy).

2. The arils of the "non-yawning" akee are quite toxic to rabbits.

3. Some of the symptoms induced in akee poisoning are those of secondary shock.

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TABLE 3.
TEMPERATURE OF RATS IN °C FED ARILS FROM "NON-YAWNERS"

Animal number	Normal	Day 1 P.M.	Day 2 A.M. P.M.	Day 3 A.M. P.M.	Day 4 P.M.	Day 5 A.M. P.M.	Day 6 A.M.	Day 7 A.M.
7-D	38.4	37.9	37.7	38.1	37.8	36.7	35.4	*
8-D	39.1	37.8	37.8	37.2	37.9	36.2	33.5	32.0
9-D	38.6	37.8	37.7	37.6	38.0	36.8	38.1	37.6
10-D	38.2	37.6	38.0	37.8	37.8	37.2	38.8	30.0
11-D	38.8	37.6	38.4	38.4	37.8	35.8	*	
12-D	38.4	37.3	38.4	38.0	38.2	35.6	*	
Aver.	38.6	37.7	38.0	37.8	37.9	36.4	36.4	33.3

* Death

ther the individual records or the averages, it is obvious that the body temperature falls.

At the present time we have some data on the microscopic changes induced in the various organs of a few animals dying from akee poisoning. Preliminary microscopic studies of the tissues and the gross pathological changes induced in these animals (chiefly rabbits) lead us to believe that the poison or poisons in the arils of unopened akee or cotyledons cause some of the symptoms of secondary shock. (12, 13) At autopsy the auricles of the heart are usually distended with dark venous blood. The lungs of most of the animals show evidence of congestion. The peritoneal cavity is usually very moist and in some cases there is some fluid present. The stomach and small intestine of most of the animals show some hyperemia and in some the mucosa is rather

thanks are extended to the owner for his fine cooperation.

LITERATURE CITED

1. Lindley, John and Thomas Moore, *The Treasury of Botany*, London, 1889.
2. Bakeland, L. H., *The Akee*, Florida State Hort. Soc. Proc. 48, 1935.
3. Popenoe, Wilson, *Manual of Tropical and Subtropical Fruits*, MacMillan, N.Y., 1920.
4. Cook, O. F. & D. N. Collins, *Economic Plants of Porto Rico*, U.S. Nat. Herb., Vol. 8, part 2, 1903.
5. Seidlein, H., *Annals of Tropical Medicine and Parasitology* 7, 377-479, 1913.
6. Scott, H.H., *Annals of Tropical Medicine and Parasitology* 10, 1-79, 1916.
7. Jordan, E. O. and W. Burrows, *American Journal of Hygiene* 25, 520-545, 1937.
8. Evans, K.L. and L. E. Arnold, *Transactions Royal Society of Tropical Medicine and Hygiene* 32, 355-362, 1938.
9. Arnold, L. E., *Jamaica Medical Review* 1, 26-55, 1947.
10. Arnold, L. E., Private communication, 1950.
11. Arnold, L. E., *Brochure, Public Health Dept. Jamaica, Vomiting Sickness (Or Akee Poisoning)*, pages 1 to 7, 1944.
12. Moon, V. H., Private Communication, 1950.
13. "Am. J. Pathol." 24, 235, 1948.