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Rapid communication

Fatal intoxication due to ackee (*Blighia sapida*) in Suriname and French Guyana. GC–MS detection and quantification of hypoglycin-A

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ABSTRACT

Between 1998 and 2001 the deaths of 16 Surinamese children were recorded along the Maroni River, which forms the border between Suriname and French Guyana. After a metabolic origin was eliminated, ethnobotanical research in the field led to a hypothesis of intoxication through the ingestion of ackee. Ackee (*Blighia sapida*) is a large green leafy tree of West African origin. Its unripe fruit contains large quantities of two toxic molecules: hypoglycin-A and hypoglycin-B, the former being the more toxic. We have developed a GC–MS procedure allowing us to demonstrate the presence of hypoglycin-A in the gastric fluid of one of the deceased children, and to compare the content of hypoglycin-A in fruit collected on the road to Paramaribo in Suriname (5.1 mg/g) with samples from Burkina Faso (8.1 mg/g) and Jamaica (9.2 mg/g).

Field research showed the misuse of this little-known plant by Maroon witch doctors. The *Bushinengue* witch doctors were informed about the dangers of ackee, and no new cases have been reported to date.

1. Introduction

1.1. Background

Between 1998 and 2001 the deaths of 16 Surinamese children were recorded along the River Maroni, which forms the border between Suriname and French Guyana. Some of them were brought by their families to French Guyana and admitted to the pediatric department of the Franck Joly Hospital in Saint Laurent du Maroni. These children had similar symptomatic characteristics (hypoglycemia, increased hepatic enzymes) and antecedents (malnutrition, hypovitaminosis) to several reported cases, notably in Burkina Faso, Benin and Haiti. After a metabolic origin was eliminated, the pediatrician considered the possibility of poisoning, and contacted the Cayenne Institute of Research and Development (IRD). Ethnobotanical research carried out by Mr Christian Moretti confirmed the presence of ackee on the west bank of the Maroni River in Suriname. Its presence was also confirmed by the LAT LUMTOX team in the Paramaribo region, where it is grown in plantations established by importing young plants from Jamaica.

1.2. Objectives

The first aim was to compare the content of hypoglycin-A in unripe fruit collected by the LAT LUMTOX team on the road to Paramaribo in Suriname with samples from Burkina Faso and Jamaica which have been supplied by Dr Barennes for Burkina Faso and Dr Jackson for Jamaica. The purpose of this fruit comparison was made to verify that the specie introduced in Suriname was homogeneous on its toxins' content with those present in Africa and Jamaica, the toxicity of which was already proved.

Five fruits were collected from five different trees at a stage of maturity of the order of 2–3 weeks before the fruit splits into three parts. The flesh of the five arils was removed, desiccated at +25 °C during 30 days then pooled and crushed in a ball mill until the obtaining of a fine light brown homogeneous powder.

Secondly, the gastric juice of a child who had died in Albina, a Surinamese border town was analyzed. In order to rule out the

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hypothesis of poisoning by any toxin other than hypoglycin-A, a complete toxicological analysis has been conducted with a specific attention to toxic components that were the possible cause of Reye's syndrome and/or major hypoglycemia. In a second step, a specific technique for identifying hypoglycin-A has been developed. Due to the extremely poor local infrastructure (especially in Suriname) there was no police inquiry, nor was there any autopsy, and no ante-mortem or post-mortem blood or urine samples were taken. Gastric juice collected after a vomiting episode 12–14 h before death was the sole fluid available and subjected to analysis.

2. Ackee

2.1. Botanical and dietary information

Ackee (*Blighia sapida*) is a large green leafy tree of West African origin. The fruit is about the size of a small pear and opens spontaneously into three parts when ripe. The edible part is the yellow flesh around the three large shiny black seeds (Fig. 1).

Ackee is the national fruit of Jamaica, where it is usually boiled in milk or water and served on its own or in meat or fish dishes such as 'ackee and salt fish'. In some African countries it is also eaten raw. It tastes a little like hazelnut or avocado [1].

It is an important dietary element, especially in poor populations suffering from undernutrition, sometimes exacerbated by natural disasters and their consequences on local agriculture.

For example, many cases of ackee poisoning (over 50 of them fatal) were recorded in Haiti in November 2000 after flooding had affected 25,000 families and destroyed maize and sugar cane crops [2].

2.2. Toxic compounds

Unripe ackee contains large amounts of two toxic molecules: hypoglycin-A (in the seeds and flesh) and hypoglycin-B (in the seeds alone), the former being the more toxic (Fig. 2).

The concentration of hypoglycin-A in the flesh of the unripe fruit is 10–100 times greater than that in the ripe fruit, depending on the season. In ripe fruit, the concentration in the flesh reduces rapidly once exposed to sunlight, but the seeds remain poisonous [3].

2.3. Clinical analysis of intoxication by hypoglycin-A

Ackee poisoning is due to hypoglycin-A, a natural hepatic toxin found in large amounts in unripe ackee fruit. Hypoglycin-A is a powerful hypoglycemic. Its method of action does not stimulate insulin secretion; it induces hypoglycemia by inhibiting secondary gluconeogenesis, limiting the cofactors (CoA and carnitine) essential to the oxidation of long-chain fatty acids [4].

Fig. 1. Photograph of a ripe ackee fruit held in the hand.

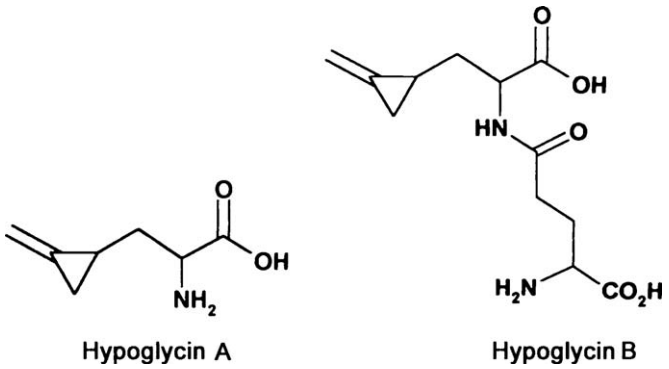

Fig. 2. Molecular structure of hypoglycin-A and hypoglycin-B.

The pathology induced by the consumption of the unripe fruit is known in Jamaica as "Jamaican vomiting sickness" or "ackee poisoning". The symptoms of ackee intoxication are similar to those of Reye's syndrome.

Clinical features of the illness include nausea, severe vomiting leading to loss of consciousness, disturbed mental states, and paresthesia. Convulsions can occur, and a coma followed by rapid death (within 12–24 h) in cases where large amounts have been ingested.

From a biochemical point of view, there is major hypoglycemia (<0.2 g/l), increased hepatic transaminases and hepatic histology reveals microvesicular steatosis of the liver.

In contrast with ackee poisoning, Reye's syndrome is characterized by the release of large amounts of fatty acid as a result of lipid metabolism anomaly. The causes of the syndrome remain unknown, although there seems to be a link between the development of the syndrome and the use of aspirin to treat viral infections in children (e.g. chickenpox, flu, gastroenteritis, and upper respiratory tract infection) [5].

2.4. Cases reported in the literature

Table 1 is a non-exhaustive list of different cases of ackee poisoning reported around the world.

3. Analysis of hypoglycin-A

3.1. Reagents

Acetonitrile, methanol, standard D-phenylalanine, N,O-bis-(trimethylsilyl)trifluoroacetamide containing 1% trimethyl chlorosilane (BSTFA) and N-methyl-N-(trimethylsilyl)trifluoroacetamide (MSFTA) were supplied from Sigma–Aldrich (Saint Quentin Fallavier, France).

Table 1
List of different cases of ackee poisoning reported around the world.

Country of origin	Number of deaths	Year	References
Jamaica	271	1980–1991	[10]
Ivory Coast	>100	1984	[8,11]
Togo	132	1988–1989	[1]
Jamaica	6	1989–1991	[7]
Haiti	>10	1991	[2]
Jamaica	28	1992	[12]
USA	1	1994	[9]
Benin	24, 40	1996, 1997	[8]
Burkina Faso	29	1998	[7,8]
Haiti	>50	2008	[2]