1980s MARINE TURTLE POISONING FILE
PART 1 OF 3 G.H. BALAZS

Poison. GHB

REFERENCES ON POISONING BY MARINE TURTLES

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A Case Report

TURTLE MEAT POISONING— THE NEW IREAND EPIDEMIC, 1965

BY J. C. H. DEWDNEY.

ALTHOUGH turtles are found all around the coast of New Guinea, and are eaten with relish by the indigenes, reports of turtle meat poisoning are few. Lagi (1932) working as an Assistant Medical Practitioner in Papua heard reports of an incident there in which most of the inhabitants of an off-shore island became ill after eating turtle, and some of these people were thought to have died. Campbell (1960) in questioning many people from different districts in the Territory found only one man able to recall an outbreak of illness following the consumption of turtle meat. In this incident, which occurred near Samarai, seven people were said to have died. Rapson (1966) recalls an epidemic of illness following turtle meat eating on the island of New Hanover (New Ireland District) some ten years ago. The turtle responsible for this illness was dead before being brought ashore. No detailed information is available regarding any of these three episodes.

The reefs around the New Ireland coast provide good hunting grounds, both for turtles and turtle catchers. Late in 1965 a villager caught one of these reptiles and brought it ashore. This was the first act of a tragedy which was to cost five lives.

CHRONICLE.

Saturday, 18th December, 1965.

Villager Soi-Nabien, caught a turtle on the reef fronting the village of Namarodu, a Patpatar-speaking group of beach hamlets in the Namatanai Subdistrict of New Ireland. The turtle was brought ashore to the hamlet of Kanou, where it was turned upon its back and left until the following Tuesday. The turtle was thought to be quite healthy by the villagers.

Tuesday, 21st December, 1965.

In the morning the turtle, apparently still in good condition, was killed and cut up. Portions of meat, and a paste composed of blood, fat and leaves, were wrapped up in banana leaves and tied into parcels. These parcels were then placed among the hot stones of a mumu in the sand close to one of the houses. Although a roof is commonly erected over a mumu, on this occasion no roof was built. The mumu was sealed about noon. Some hours later rain began to fall and continued through the night.

Wednesday, 22nd December, 1965.

Rain fell for most of the day. The amount of rain which fell in the village is not known, but at Namatanai Government Station, a few miles from Namarodu, over 400 points of rain were recorded in the period during which the meat was in the mumu.

At ' lamp-lighting time ', about 6.30 p.m., the mumu was opened. The parcels of meat and paste were distributed, the men and boys taking their portions to the men's house where freshly cooked vegetables were eaten with the turtle meat. The women took their meal elsewhere. Some of the meat was kept for later consumption.

Thursday, 23rd December, 1965 (*First day of illness*).

About 6 a.m. some of those who had eaten turtle became ill with vomiting, and others fell sick as the day wore on. Despite this, however, some villagers ate more of the cooked meat. The illness on this day was markedly vomiting and retching ; one to four bouts per person, little abdominal pain and no diarrhoea. Those who had suffered repeated bouts of vomiting stated that they became very weak and had difficulty in traversing the few yards between their houses and the beach where dejecta are customarily deposited. One elderly man continued vomiting repeatedly and was subsequently admitted to the Namatanai hospital.

Friday, 24th December, 1965 (*Second day of illness*).

Six new cases appeared on Friday. These were more seriously affected than those of the previous day. Five of the new cases had vomiting, abdominal pain (not very severe) and diarrhoea, with weakness of limbs. The sixth case, a baby two months old, commenced vomiting, apparently became comatose, and died in the village a few hours after the onset of illness.

Saturday, 25th December, 1965 (Third day of illness).

The elderly man who had been vomiting intermittently since Thursday was admitted to the Namatanai hospital together with the five survivors of those who fell sick on Friday; one of these, an eleven-year-old boy died about 6 p.m. An infant who began vomiting on Saturday morning became comatose and died in the village. One man, father of the child who died on Friday, commenced vomiting and remained in the village.

Sunday, 26th December, 1965 (Fourth day of illness).

A woman who had been admitted to hospital the previous day died. The man who commenced vomiting on Saturday was brought to hospital dyspnoeic and semi-comatose. Those already in hospital appeared to have improved. No new cases appeared.

Monday, 27th December, 1965 (Fifth and last day of illness).

The man admitted to hospital the previous day died. No new cases appeared on this or subsequent days.

Tuesday, 28th December, 1965.

Mid-morning a message was received in the District Medical Office at Kavieng, 160 miles from Namatanai, reporting the deaths of five villagers following the eating of turtle meat. I arrived at Namatanai that evening and examined the body of the man who had died the previous day. The Namarodu people still in hospital were examined and found to have recovered from their poisoning. Inquiries into the nature and extent of the outbreak were commenced.

Wednesday, 29th December, 1965.

I visited Namarodu village, was shown the site of the mumu, the carapace of the turtle and collected reports of the incident from the villagers. A meatless feast was being busily prepared in honour of the dead. All villagers appeared to be well.

MORBIDITY AND MORTALITY.

The 1965 census population of Namarodu was 144 persons. One-third of these shared the cooked turtle. Of the 41 people (23 males, 18 females) who admitted to eating turtle, 15 became ill—ten males and five females. In addition two infants died in the village at the time of the epidemic. Their mothers denied that these children had been given any part of the turtle, but for reasons mentioned below, the deaths of these infants are regarded as arising from ingestion of the contaminated meat. Seven people were admitted to hospital, and of these, three died. Including the two infants, the sex and ages of those who died were males of two months, six months, 11 and 34 years respectively, and one woman of over 40 years. Morbidity and mortality are summarized in the Table below.

DISCUSSION.

Cause of the epidemic.

The occurrence of the symptoms of food-poisoning shortly after the ingestion of an unusual item of diet arouses a suspicion that this food, in some way, may have caused the illness. When, further, one learns that the food was meat which, after a comparatively short time in a hot-stone cooker, was left in a rain doused pit for many hours, then eaten without re-cooking, that suspicion becomes extremely strong.

Table showing number of Namarodu villagers who ate turtle meat, became ill, were admitted to hospital, and died, December, 1965.
(Total population of village at 1965 Census = 144 persons.)

Age group (Years).	Number eating turtle-meat. M.	F.	Number becoming ill. M.	F.	Number admitted to hospital. M.	F.	Number of deaths. M.	F.
0-11/12	2*	-	2	-	-	-	2	-
1-14	5	9	1	1	1	-	1	-
15-44	11	5	5	2	1	2	1	1
45-	7	4	4	2	2	1	-	-
Totals by sex	25	18	12	5	4	3	4	1
GRAND TOTALS	43		17		7		5	

* Unproven.

The environment within the banana-leaf wrapped parcels left in a cooling mumu would appear to be an ideal culture medium for micro-organisms.

Incidence of the illness.

Of the 41 people who admitted eating the turtle meat, only 15 became ill. It appears probable that some parcels of the food did not contain a sufficient load of the pathogenic agent to cause sickness. Possibly because of their position among the hot stones such parcels received a more thorough heating before rain cooled the mumu ; their contents may have provided a less favourable environment for microbial growth ; or perhaps their contents did not become contaminated prior to the tying up of the parcels. An attempt was made to ascertain whether those who became sick had eaten from parcels other than those eaten by their more fortunate neighbours. Due to informants contradicting one another and themselves, this enquiry was not pressed.

Mothers of the dead infants averred that their children had not been given any of the turtle meat, and other villagers agreed that such food would not be given to babies prior to the eruption of teeth. Asked to explain the children's deaths, villagers replied that the turtle meat had poisoned the mother's milk and this was the reason for the illness and death of the infants. It seems more probable that the infants were, in fact, given some of the cooked food, possibly the blood and fat paste, but the mothers' fear of admitting breach of a taboo, and perhaps their fear of police action against them, prevented their complete frankness.

The man, father of one of the dead infants, who himself died in hospital on the fifth and last day of the incident, had almost certainly eaten turtle at least 36 hours after the meat had been removed from the mumu, so it is highly likely that some meat was kept in his house after the Wednesday night feast, and was given to his son on the Thursday.

Clinical features of the epidemic.

Villagers who ate portions of the cooked turtle within a short time of its removal from the mumu, either experienced no ill effects or suffered a relatively minor illness in which vomiting was the main complaint ; abdominal pain and diarrhoea were either minimal or absent. Those who ate meat on the day following its removal from the mumu, or later, presented a more serious picture, including repeated vomiting, abdominal pain, diarrhoea, muscular weakness and inco-ordination of movement, dysarthria, changes in or loss of conciousness, respiratory depression and finally death. Post-mortem examination of the man who died on the final day of the incident showed no extensive lesions of the alimentary tract and findings were consistent with death from respiratory failure. None of those who had eaten the meat complained of mouth and throat symptoms described by Romeyn and Haneveld (1956).

The toxic agent.

The increasing severity of the illness with the lengthening of the interval between the meat's removal from the mumu and its being eaten, together with the clinical features reported and the post-mortem findings suggest a microbial toxin as the agent responsible for this epidemic. However, the picture does not unequivocally typify either staphylococcal or clostridial contami-

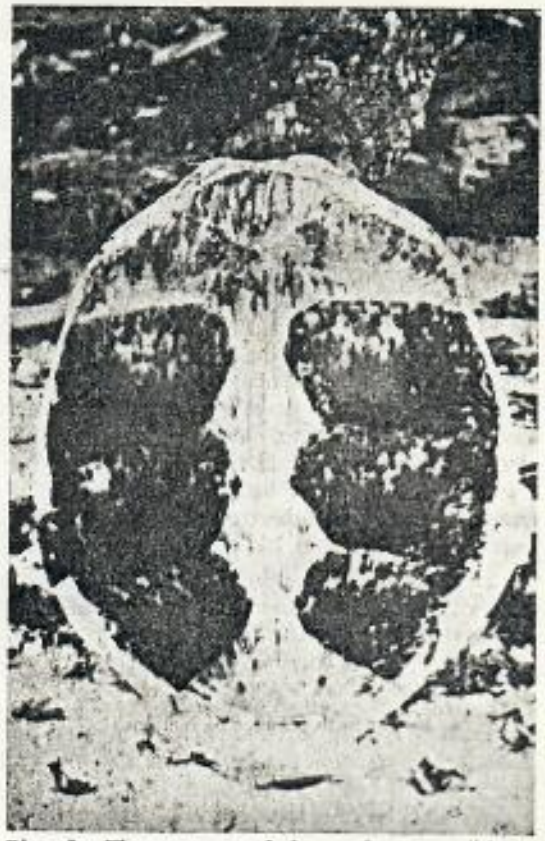

Plate I.—The carapace of the turtle responsible to this epidemic.

nation. The absence of mouth and throat symptoms points to an agent other than that responsible for the epidemic in Netherlands New Guinea (West Irian) reported by Romcyn and Haneveld (1956).

The carapace of the turtle responsible for the Namarodu tragedy was photographed (*Plate* I). Unfortunately some of the scutes had been removed before this photograph was taken and this has made positive indentification of the specimen difficult. Copies of the Kodachrome transparency have been seen by authorities in New Guinea, Australia, England and the U.S.A., and resemblances noted to the species *Caretta caretta*, *Eretmochelys imbricata* and *Chelonia mydas*—three of the four turtle species found around the New Guinea coast.

SUMMARY.

An outbreak of poisoning following ingestion of poorly-cooked turtle meat is reported. Of the 43 persons eating this food 17 became ill and five of these died. The toxic agent was not identified.

Note.—The author would be pleased to send a Kodachrome transparency of the incomplete carapace to anyone wishing to attempt a definitive indentification of the species of turtle involved in this epidemic.

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"NEW GUINEA MOUTH"

NEW GUINEA.

Beriberi assumed epidemic proportions on the goldfields. Transport was exceedingly difficult, and only the most portable articles could be carried in. The great majority of the natives therefore lived on polished rice and tinned meat, and this, together with the depressive factors of great altitude (7,000 to 8,000 ft.), constant cold and rain and heavy work, make beriberi inevitable. It was not long before cases of ulcerative stomatitis made their appearance, and it is interesting to note that so common has this symptom been on other goldfields that is is commonly known as 'New Guinea Mouth'.

Verbal and written representations to the miners by medical officers were not successful, so a special diet was made obligatory by law, and the incidence of beriberi began at once to decline."

Report to the League of Nations, 1926-1927.

PAPUA.

"As was the case last year, the deficient rainfall during this year has resulted in a similar deficiency of native food. In all, 20 cases of ulcerated mouth disease (early scurvy) passed through the Native Hospital, Port Moresby, and 17 through the Native Hospital, Samarai. This 'ulcerated mouth' disease has undoubtedly a scurvy basis although the organisms found are almost entirely large spirochaetes usually mixed with fusiform bacilli much as in Vincent's Angina and Noma. The first signs are that the gums bleed if rubbed; they next ulcerate along their margins and the breath gets foetid. Ulceration then spreads on the inside of the cheek, usually adjacent to the molars, and anaemia and general weakness become marked. In some instances the ulceration spreads markedly and may even lead to necrosis of the palate or portions of the jaw-bone. If untreated, death occasionally occurs due either to extensive spread of the disease or to general weakness and debility. Cases not too far gone recover as a rule readily with some form of anti-scurvy treatment, as they did in this instance. Other signs of scurvy are not frequently seen."

W. M. STRONG,
Chief Medical Officer.
Annual Report 1931-1932.

Letters to the Editor

TURTLES AND BOTULISM

Dear Sir,

In your June 1975 edition (v.18, p.125), you published a letter from me about an outbreak of turtle meat poisoning that occurred whilst I was Medical Superintendent of the Kavieng Hospital in August 1974. Several similar outbreaks had occurred in New Ireland and elsewhere. No satisfactory explanation was offered of the nature of the poisoning.

I am very grateful to Dr Greg Lawrence, of the Institute of Medical Research in Goroka, for the suggestion made to me privately that the symptoms described by me in the letter were consistent with those of botulism.

Botulism differs from other forms of food poisoning in that gastro-intestinal symptoms are slight or absent. The condition is more accurately described as an intoxication, and the action of the toxin on the parasympathetic nervous system is responsible for the most prominent symptoms, namely, dilated pupils, aphonia and cranial nerve palsies. General muscular weakness and paralysis is also a feature, and death, which is usual, occurs from respiratory or cardiac failure, or airway obstruction.

Of course few doctors have ever seen a case of botulism, and therefore it would normally pass undetected. Furthermore, bacteriological confirmation can only be made by the isolation of the organism and its toxin in the suspected food.

Botulism has been reported with prepared fish foods; equally, since the toxin itself is heat stable, the turtle may have been already intoxicated and moribund when caught. This could explain the strange advice, 'If the turtle comes to you, do not eat it'.

In view of the doubt about this condition, it might be wise to follow Dr Edmonds' advice, 'Refrain from eating turtles'.

Yours faithfully,

Robert Likeman

REFERENCE

EDMONDS, C. (1974) *Dangerous Marine Animals of the Indo-Pacific Region*. Newport, Wedneil. On botulism, p.165; on turtle poisoning, p.195f.

PSYCHIATRY REJOINDER

Dear Sir,

Kindly permit me to reply to Dr Burton-Bradley's comments on my letter 'Too much Psychiatry' (*P.N.G. Med. J.* 19:181, 1976). Unfortunately he appears to have missed the point of the letter which was to question both the effectiveness and efficiency of the psychiatric services compared with traditional means for dealing with behavioural disorders.

It would be regrettable if he actually finds such questioning merely a 'quaint little diatribe'.

Yours,

B. Hocking.

mission has been difficult to interrupt since spraying started in 1970, there was an average of only four malaria cases per month during the latter half of 1974.

Malaita District has experienced minor outbreaks of transmission in scattered areas every year since spraying started in 1970. This was repeated during 1973 and more seriously in 1974. During mid 1974 East Kwaio, an operationally difficult area, showed signs of resolving, only to break out again in November and spread up and down the coast and also across the mountain barrier to west central Malaita. This outbreak of mainly *P. falciparum* malaria, which was resolving by April 1975, was an object lesson in the need for very speedy action once the parasite gets the upper hand in a semi immune population (as a result of spraying).

During 1973-74 further efforts were directed towards improving the standards of field operations, but delays in supplies and transport problems continued, as always, to threaten the success of the programme. Great efforts were made to improve on the supervision of treatment of all proven malaria cases to avoid the well known relapsing propensity of S.W. Pacific *P. vivax* strains. Remedial measures were intensified around all foci. Mass Drug Administration was extensively used with good results in the Gilbertese communities in Western District, Nggela and N.E. Makira but equivocal results in North Guadalcanal.

Objectives and plans for 1975 include the elimination of all *P. falciparum* Parasitaemia from the Solomons; a special effort in Malaita; an improvement in the detection of imported cases into Western District and an all out onslaught on the north coast of Guadalcanal. Additional supplementary measures will include the use of ultra low volume peridomestic space spraying with malathion and the use of Abate Larvicide. These will be directed at specific foci with the objective of reducing vector densities to a very low level during the low transmission season.

The prospects for the eventual eradication of malaria from the Solomon Islands remain good. It cannot be emphasized too strongly however, that the task is not an easy one and that it requires the wholehearted backing of all agencies in the Ministry of Health, full support by other agencies and last but by no means least, full acceptance and co-operation by the people.

I am grateful to His Excellency, the Governor of the British Solomon Islands, for permission to publish this Progress Report.

Dr. J. G. Avery,
Chief Medical Officer (Community Health), Ministry of Health and Welfare,
HONIARA. British Solomon Islands.

REFERENCE

[1]AVERY, J.G. (1974). *A Review of the Malaria Eradication Programme in the British Solomon Islands* 1970-72. Papua New Guinea Medical Journal (1974) 17 50-60.

TURTLE MEAT AND CONE SHELL POISONING

Sir

Two unusual cases which presented at Kavieng hospital during 1974 illustrate an interesting and disturbing lack of local knowledge about dangers from marine animals.

1. Turtle Meat Poisoning

A meal of turtle was eaten in Panapai village, close to Kavieng, on the afternoon of Sunday 11th August. During the night several children were sick. At about 6.00 a.m. the next day a child of 6 years was brought to the hospital, after having become unconscious following vomiting. There was no response to pain, a flaccid paralysis and dilated pupils. Lumbar puncture and a malaria parasite slide were performed to exclude more common diagnoses, but in spite of supportive measures including intravenous fluids, hydrocortisone and phenergan, the child died quite suddenly at midday. This child was alleged to have eaten the unlaid eggs of the turtle.

During the night 2 more children died in the village. The next morning the teacher brought 18 other children who had eaten the turtle. Fifteen were symptom free; 3 had had attacks of vomiting, and were kept

under observation for 24 hours. In the absence of further symptoms, they were discharged.

Despite careful inquiries we were unable to ascertain the details of the catching and cooking of the turtle, but the shell was recovered and identified as the ERETMOCHELYS IMBRICATA or Hawksbill Turtle. It was not possible to discover whether the poisoning was due to staphylococcal or clostridial contamination, or a toxin in the meat itself.

Several persons associated with this incident recalled the poisonings at Namatanai in December 1965 which resulted in 5 deaths, and on New Hanover in 1957 and isolated incidents had occured at various other places in the New Ireland District over many years. Nobody however knew definitely which species of turtle were dangerous or when. The only positive comment was that "If the turtle comes to you, do not eat it. If you go after it and catch it, it is good for eating." It is by no means clear what was meant by this.

Reviewing the literature (Dewdney 1967, Halstead 1959, Bell 1972,) it appears that several species of turtle, notably the Hawksbill, the Green Sea Turtle (CHELONIA MYDAS) and the Leatherback Turtle (DERMOCHELYS CORIACEA) do became poisonous to eat at times, possibly by a process analogous to ciguatera poisoning in fish. The presence of neurological symptoms generally favours this diagnosis rather than a bacterial intoxication, although this undoubtedly can occur. In spite of the risks and local uncertainty about the nature of turtle meat poisoning, turtles continue to be eaten in large number here, and so presumably intoxication will continue to occur from time to time.

2. Cone Shell Poisoning

A 35 year old man was admitted unconscious after having been diving on a reef in the Kavieng harbour the previous night with the aid of a flashlight he had picked up a coneshell, which he had placed in his trouser pocket. The shell was later identified as CONUS GEOGRAPHICUS. A short while later he felt a sharp prick in his thigh, but thought little of it. While cycling home he felt weak, and went straight to bed. His family later noticed that he was not moving his chest when he breathed, and was not rousable, so they brought him to hospital. On examination he had generalized flaccid paralysis, and was breathing satisfactory but only with his diaphragm. Other observations were normal. No mark could be seen on his thigh.

No treatment was given beyond careful observation. The following morning he was fully fully conscious, although drowsy, and could move his legs. He was out of bed the next day and discharged two days later.

I subsequently showed the shell to every local member of the hospital staff. Only two said they were aware that it was dangerous; one of these was from Manus District, the other, a mixed race person. This suggests poisoning by Cone Shells is very rare since otherwise one would expect local folk lore to have stored the information.

In the majority of cases of stings or bites which present to hospital (3-4 a month in Kavieng), no positive identification is made, because the victim is injured while wading, the injury is to the leg or foot is under water and the attacking creature is not seen. Occasionally an identification can be made if the patient has subsequently speared the animal (e.g. a stone-fish) or if some part of the sting or proboscis is left in the wound.

R. Likeman,
Kavieng Hospital, KAVIENG
The Diseases & Health Service of Papua New Guinea Dept. of Public Health.

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A NOTE ON THE DISTRIBUTION AND PREVALENCE OF ANOPHELES SUBPICTUS GRASSI

Sir—

Anopheles subpictus has been recorded from the following localities - (1) the Port

Moresby area (Hill, 1925, and current surveys by Dept. of Health); (2)/Motu Motu, 150 miles west of Port Moresby (Bang et al., 1947); (3) Mirivasi in the Papuan Gulf (Roberts and O'Sullivan, 1948); (4) D'Entrecasteaux Islands (Spencer, 1965); (5) Wewak (Peters & Christian, 1963); (6) Milne Bay (Lee & Woodhill, 1944); (7) New Britain (Lee & Woodhill, 1944; Spencer, Spencer, & Venters, 1974); (8) Madang (Dept. Health report, 1974).

With such a wide known distribution, this species will undoubtedly be found in many other localities along the Papua New Guinea coastline.

Bang et al. (1947) recorded that this species was "extremely prevalent, came frequently to feed on humans, was present in houses in the daytime, and was found infected" (with malaria).

A. subpictus was found at numerous points around the entire perimeter of the three large islands of the D'Entrecasteaux Group, sometimes in large numbers. Specimens occurred in all types of catch - day and night indoor-resting, day outdoor-resting, leg-biting, exit-traps - and occurred commonly. Larvae were readily found. In 1957 this species constituted 1% of 2228 anophelines taken in leg-biting catches on Goodenough Island in this Group; in 1958 it represented 14% of a total of 1572 anophelines. Breeding was in pools and on the edges of brackish tidal creeks.

As *A. subpictus* occurs in the Port Moresby area it is possible that specimens will be found in aircraft originating from there and landing at Australian airports.

M. Spencer
Formerly Entomologist, Dept. of Public Health, Papua New Guinea

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Date: Sat, 29 Aug 1998 22:02:31 -1000 (HST)
From: "George H. Balazs" <gbalazs@honlab.nmfs.hawaii.edu>
To: Don Buden- Math & Science <mathscience@mail.fm>
Subject: Re: turtle poisoning

Good to hear from you. Yes, indeed, I have some papers more recent than the 70's. Unfortunately, though, very little new is know about this poisoning from hawksbills. Sporadic. Decades can go by in an area with no cases. Then, a whole village will become ill from sharing a single turtle. Some babies will die, along with some elderly. The symptoms are horrible. It's interesting that no cases have ever been know from the Caribbean where hawksbills (at least in the past) have been somewhat numerous and regularly eaten.

I'll pull the papers together and mail to you. Note that most do not refer to this poisoning as being related to ciguatera fish poisoning. Hawksbill normally don't eat algae. Their most common diet item is sponges (which as you know contain weird toxic compounds).

I'm leaving on early Tuesday for a week of field work at French Frigate Shoals. So the mailing will go out to you shortly after my return.

Aloha, George


```
*****************************************
*       George H. Balazs, Leader        *
*   Marine Turtle Research Program      *
*  National Marine Fisheries Service    *
*         Honolulu Laboratory           *
*          2570 Dole Street             *
*   Honolulu, Hawaii 96822-2396 USA     *
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*Email: gbalazs@honlab.nmfs.hawaii.edu* *
*****************************************
```


On Sun, 30 Aug 1998, Math & Science Department wrote:

> George
>
> I have an ms on the reptiles of Sapwuahfik Atoll in progress and which is
> based largely on my 5-week visit this past summer. Last year, several
> Sapwuahfik islanders died in two separate incidents of turtle poisoning
> involving Eretmochelys imbricata. I have some background information on
> chelonitoxication, but most of it from the 1970s and earlier. Little was
> known of the actual cause of poisoning then, the general concensus being
> that the toxins were derived from algae ingested by the turtles, in the
> manner of ciguatera in fish. George Zug indicated you might be able to
> provide updated information. If you know of any recent papers addressing
> the issue of epidemiology, I would very much appreciate your sending me
> xeroxes of one or two that I could cite as background information. Library
> resources here at COM are rather limited.
>
> Don Buden
>
>
> Title III Math/Science Connection
> Department of Mathematics and Natural Sciences
> College of Micronesia-FSM Palikir
> P.O. Box 159 Kolonia
> Pohnpei FM 96941-0159

PLATE 9.54
A captive hawksbill turtle eating an adult *Chironex fleckeri*.
(Photograph courtesy of Ben Cropp Productions, Port Douglas, Queensland)

PLATE 9.55
A [illegible] fish photographed in the act of eating a young (inverted) box jellyfish *Chironex fleckeri*.
(Courtesy Ben Cropp Productions, Port Douglas, Queensland)

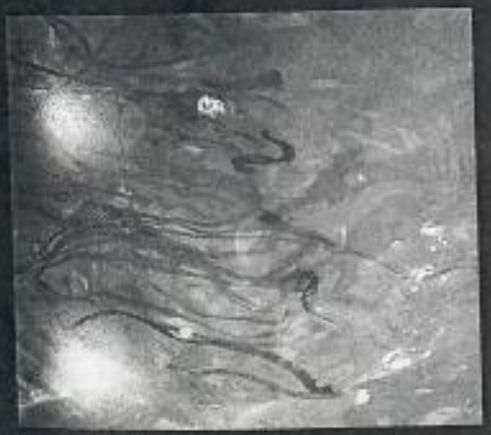

PLATE 9.56
A large adult *Chironex fleckeri* occupies almost the entire field of the photo, swimming from left to right. Even under such 'ideal' conditions for spotting (clear sunlit calm seawater) the animal is still difficult to see. Compare with Plate 5.8.
(Courtesy of Dr Bob Hartwick, James Cook University of North Queensland).

PLATE 9.57
Microscopic section (x400) of an unfired *Chironex fleckeri* tentacle piece, stained to show the large, plum-coloured, cigar-shaped, dangerous-venom-bearing microbasic mastigophore nematocysts.
(Compare with Plate 6.12)
(Photograph courtesy of Dr Bob Hartwick, James Cook University of North Queensland)

PLATE 9.58
A major Northern Territory *Chironex fleckeri* sting (in which specific *Chironex fleckeri* antivenom was administered), with a less common sting distribution involving the lower back, as well as the posterior upper thighs. Protection afforded by the swimming trunks is evident. Note the vesiculation on the welt just above the natal cleft. (Courtesy of Menzies School of Health Research, Darwin)

PLATE 9.59
A preserved specimen of *Chironex fleckeri* captured in Brunei in 1993.
(Specimen courtesy of Major [Dr] R. Hooper, Labuan, Sabah)

CONE SHELL

INDO-PACIFIC (INCLUDING JAPAN), INDIAN OCEAN

(KOHN 1963; HALSTEAD 1988)

Fatalities have occurred, but the number is uncertain. Seven deaths are listed by KOHN (1963), as shown in Table 3.3A, but up to 15 outside Australia are claimed (KOHN unpublished observations in WILSON & GILLETT 1980). HALSTEAD (1988) states a 'crude mortality rate of about 25 per cent' on the basis of 'more than eight deaths' from 'more than 28 cases'. *Conus geographus* is responsible for the majority of confirmed deaths, with *Conus textile* responsible for one death and suspected in one other (KOHN 1963; SUTHERLAND 1983; HALSTEAD 1988). Recently another two deaths in Japan from *Conus geographus*) have been advised (Table 3.3B) (TOMIHARA 1994, unpublished observations) [Chapter 20, page 415].

Of the four deaths in Okinawa (Tables 3.3A and B), the 1927 death was a five-year-old boy stung on the finger while clamming on a beach; the 1935 death was a 32-year-old male stung on the right thumb while clamming in the sea; the 1944 death was in a 13-year-old male stung on the left finger while fishing in the sea; and the 1954 death was in an 11-year-old stung on the left hand while clamming on the beach (YASHIRO 1939; TOMIHARA 1994, unpublished observations).

POISONOUS TURTLE FLESH (CHELONITOXIN)

MALAYA, SRI LANKA, INDIA

(HALSTEAD 1970 Vol 3)

The number of fatalities is unknown, but a review by SILAS and BASTIAN FERNANDO (P1986, published by the Regional Centre of the Central Marine Fisheries Research Institute, Marine Fisheries PO, Mandapan Camp, Tamil Nadu, India) documents (in some detail) 95 Indian deaths (including 36 children, three of them breastfed) out of a total of more than 723 patients. Chelonitoxin is apparently very toxic; a mortality rate of 28 per cent in outbreaks is reported (HALSTEAD 1970 Vol 3), and well-documented autopsy reports exist (HALSTEAD 1988).

ASIATIC PORPOISE FLESH POISONING

CHINA

(MACGOWAN 1887), and

WHITE WHALE FLESH POISONING

ARCTIC

(STEFANSSON 1944)

Fatalities from ingestion are claimed but no precise details are known.

TABLE 3.3A
DOCUMENTED CONE SHELL ENVENOMATION FATALITIES, BASED UPON KOHN 1963.

Date of sting	Species	Location	Original description
unknown	*Conus textile*	Banda, India	Rumphius (1703)
14 May 1859	*C. textile*?	New Hebrides (now Vanuatu)	MacGillvray (1860)
unknown	*C. geographus*	Loyalty I (now New Caledonia)	Gill (1876)
unknown	*C. geographus*	Fiji	Cleland (1912)
28 June 1927	*C. geographus*	Okinawa, Japan	Yasiro (1939)
25 June 1935	*C. geographus*	Hayman I, Nth Qld, Australia	Iredale (1935)
29 June 1935	*C. geographus*	Okinawa, Japan	Yasiro (1939)

Venomous and Poisonous Marine Animals: A Medical and Biological Handbook

Medical Editors:

John A. Williamson
Peter J. Fenner
Joseph W. Burnett

Biology Editor:

Jacqueline F. Rifkin

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INTOXICATION ALIMENTAIRE COLLECTIVE PAR CONSOMMATION DE TORTUE DE MER DANS LE DISTRICT D'ANTALAHA

RANAIVOSON G.[1], CHAMPETIER DE RIBES G.[2], MAMY E. R.[3], JEANNEROD G.[2], RAZAFINJATO P.[1], CHANTEAU S.[4]

RESUME : *En Décembre 1994, une intoxication alimentaire collective par consommation de tortue de mer a touché environ 60 personnes, sur la côte nord-est de Madagascar. Les signes cliniques prédominants étaient digestifs (nausée, vomissements, dysphagie, stomatite aiguë) et pouvaient persister plusieurs semaines. Le taux d' attaque de l' intoxication a été de 48% avec un taux de létalité de 7,7%.*

De tels accidents, bien que rares à Madagascar, nécessitent l'organisation structurée d'une surveillance des intoxications après consommation de produits de la mer, afin de mettre en place des mesures préventives adaptées.

Mots clés : Intoxication alimentaire collective - Toxine flore et faune marines - Tortue de mer - MADAGASCAR.

ABSTRACT: *"Mass food poisoning following turtle consumption in the district of Antalaha - Madagascar" : in December 1994, a mass food poisoning through ingestion of turtle affected about 60 persons, on the north-eastern coast of Madagascar. The prevailing clinical signs were digestive (nausea, vomiting, dysphagia, acute stomatitis) and might persist during several weeks. The poisoning attack rate was 48% with a lethality of 7,7%. Such accident, even if rare in Madagascar, requires a structured organization to control sea products poisoning and to set up adequate prevention measures.*

Key-words : Mass food poisoning - Marine toxins - Turtle - MADAGASCAR.

INTRODUCTION

Les intoxications alimentaires par consommation de tortue de mer sont bien connues dans la région Indo-Pacifique : Philippines, Sri Lanka, Indes et d'autres pays du sud-est asiatique (1). Les symptômes varient en fonction de la quantité de viande ingérée et de la personne (2). Généralement les premiers signes surviennent de quelques heures à quelques jours après la consommation de tortue : les symptômes initiaux consistent habituellement en nausée, vomissements, diarrhée, pâleur, douleur épigastrique, froideur des extrémités, vertiges, céphalées, stomatite aiguë; puis hypersialorrhée, dysphagie, papulo-pustules et ulcérations sur la langue pouvant persister des mois, parfois somnolence, coma, et décès pouvant atteindre 28% des personnes intoxiquées (2), essentiellement les enfants et les sujets âgés .

A Madagascar, les intoxications après consommation de certains poissons sont connues et relativement courantes. Par contre les intoxications après consommation de certaines tortues semblent rares et n'ont pas fait à ce jour l'objet d'étude.

MATERIEL ET METHODES

Du 19 au 22 Décembre 1994, une tortue de mer du nom vernaculaire de "*Fanoara*" (*Eretmochelys imbricata*, CARET), a été consommée (après cuisson) par environ 120 villageois, habitant à 27 km au nord de la ville d'Antalaha (Province d'Antsiranana, dans les *fokontany* d'Amboahangy et d'Andrasaingy) . La tortue avait été prise vivante le 18 au soir, alors qu'elle était sur la plage. L'alerte a été donnée le 27 Décembre, lorsque deux femmes du premier village ont été hospitalisées dans le Centre Hospitalier de District (CHD) d'Antalaha et y sont décédées dans un tableau assez semblable. Ce n'est que le 6 Janvier que les services centraux du Ministère de la Santé à Antananarivo ont été informés d'une intoxication alimentaire collective après consommation de tortue de mer, et une enquête épidémiologique a pu être réalisée les 10 et 11 Janvier 1995.

L'enquête descriptive a été réalisée auprès des sujets qui avaient consommé de la tortue en cause, à partir d'un questionnaire préimprimé.

Une étude comparative de type cas-témoin a ensuite été réalisée entre les sujets malades et les non malades pour lesquels un questionnaire devait être complété. Reprenant les données de la littérature et les signes cliniques des personnes décédées après avoir consommé de la tortue en cause, a été défini comme cas toute personne ayant consommé de la tortue entre le 18 et le 23 Décembre 1994 et présentant dans les heures/les jours/les semaines qui ont suivi, plusieurs des symptômes suivants qui ne soient pas liés à une pathologie intercurrente évidente :

[1] Service de Surveillance Epidémiologique (DLMT) - Ministère de la Santé. [2] Conseiller technique en [3] Service Sanitaire de District d'Antalaha [4] Institut Pasteur de Madagascar.

- signes buccaux : stomatite aiguë, sensation de brûlure de la langue et des lèvres, hypersialorrhée, dysphagie, ulcération buccale, haleine fétide

- signes digestifs : nausée, vomissements, diarrhée

- signes neurologiques : sensation ébrieuse, vertige, somnolence, coma

- signes généraux : céphalée, pâleur, sueurs, tachycardie, refroidissement et/ou fourmillement des extrémités.

Une dysphagie isolée survenue dans les 72 heures après le repas en cause a aussi été retenue pour 3 cas.

Les seules variables qui peuvent être comparées sont: âge, sexe, date du repas, partie de la tortue consommée.

Les données ont été traitées sur les logiciels Epiinfo, Access et Excel.

RESULTATS

Le questionnaire n'a pu être complété que pour 66 personnes sur les 120 personnes qui auraient consommé de la tortue (soit environ 55% des consommateurs).

En prenant en compte les informations fournies par le médecin du District d'Antalaha et les médecins de l'hôpital d'Antalaha, l'interview des responsables des 2 *fokontany* (Amboahangy et Andrasaingy) et d'une quarantaine de personnes de ces villages, et l'analyse des questionnaires, il est possible de donner les éléments suivants:

1- L'agent causal

Une tortue de mer de grande taille (80 cm sur 50 cm, environ 25 à 30 kg) a été trouvée vivante sur la plage : le "pêcheur" (venant d'un village éloigné de 20 km) l'a tuée et dépecée sur la plage, avant d'en vendre les morceaux. Il n'a pas été possible de retrouver ce vendeur qui se cache, mais qui selon sa famille aurait aussi des symptômes liés à l'ingestion de viande de cette tortue.

Les gens du village précisent qu'il s'agit d'une tortue de type "*Fanoara*" connue comme étant occasionnellement toxique : habituellement cette toxicité est recherchée par l'observation d'un prurit en mettant du sang de la tortue au contact de la peau. Dans le cas présent, le "test" a été négatif. La carcasse n'a pu être retrouvée (elle est avec le pêcheur), seulement 2 écailles nous ont été fournies et quelques petits morceaux d'écaille ont été retrouvés sur la plage où elle a été dépecée. Aucun reste de viande de tortue n'a pu être retrouvé en raison du long délai entre la date du repas et celle de l'enquête.

Il est à noter qu'une intoxication collective après consommation de tortue de mer était survenue en 1993 dans un village situé à 10 km au nord d'Amboahangy et en 1990 à Amboahangy. La saison Novembre à Mars est connue comme étant la saison à risque d'intoxication après consommation d'animaux marins.

2- Analyse descriptive des questionnaires

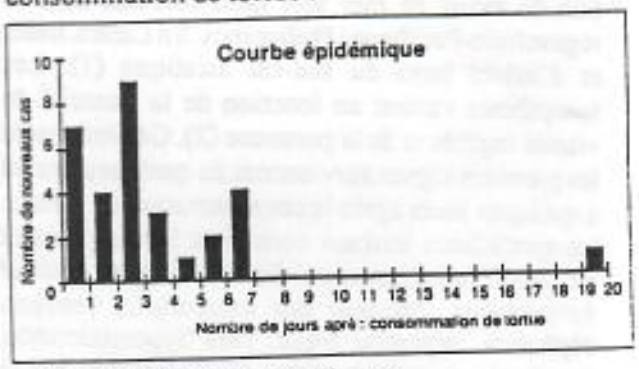
Age et sexe : L'âge des 66 sujets ayant consommé de la tortue variait de 8 mois à 80 ans, avec une médiane à 13 ans. 53% étaient de sexe féminin et 47% de sexe masculin.

Période de consommation de tortue: du 19 au 22 Décembre 1994.

Mode de préparation et morceaux consommés: le temps de cuisson a varié de 2 à 10 heures, quels que soient les morceaux mangés . Les gens ont mangé de la chair et/ou des oeufs ; la consommation s'étant passée 3 semaines avant l'enquête, il était impossible de faire préciser la quantité consommée. Par ailleurs, il n'a pas été possible de retrouver des restes de la tortue pour une analyse toxinique.

Taux d'attaque : 32 personnes sur les 66 rentrent dans la définition d'un cas au moment de l'enquête, soit un taux d'attaque de 48%.

Courbe épidémique : 22% des malades ont présenté des signes dans les 24 heures qui ont suivi la consommation de tortue, 64,5% des malades ont présenté des signes dans les 72 heures. Un malade a présenté les premiers signes 20 jours après la consommation de tortue .



Description des symptômes :

- dysphagie dans 50% des cas
- nausées, vomissements dans 38% des cas
- stomatite aiguë dans 34% des cas
- papules sur la langue dans 31% des cas
- sensation de brûlure des lèvres dans 31% des cas
- ulcération buccale dans 28% des cas
- somnolence, asthénie dans 22% des cas
- tachycardie dans 19% des cas
- céphalées dans 19% des cas
- vertige dans 19% des cas
- fièvre dans 13% des cas
- hypersialorrhée avec haleine fétide dans 13% des cas
- dans 3 cas, sueur, pâleur ou fourmillement des extrémités

- dans 3 cas, diarrhée
- dans 3 cas, une polyadénopathie cervicale inflammatoire a été notée, avec fistulisation dans un cas (non vu au cours de l'enquête).

Evolution :

- *Guérison* : sur les 32 personnes ayant présenté des symptômes, 15 étaient guéries au moment de l'enquête. La guérison était survenue dans un délai de 1 à 7 jours après le début des signes dans 8 cas, et dans un délai de 8 à 16 jours dans 7 cas .

- *Persistance des signes* : 17 personnes avaient encore des signes au moment de l'enquête, essentiellement à type de dysphagie et de stomatite.

- *Décès* : 5 personnes sont décédées, soit un taux de létalité de 7,7%. Les cas de décès étaient :

• *une fillette âgée de 8 mois allaitée par sa mère qui avait mangé de la tortue ; la fillette a eu des vomissements intenses 29 heures après consommation de la tortue par sa mère et elle est décédée 4 heures plus tard .*

• *son frère âgé de 2 ans a mangé de la chair de tortue; il a eu des vomissements et de la diarrhée 34 heures plus tard et est décédé 2 heures après.*

• *deux femmes âgées respectivement de 48 et 60 ans ont été hospitalisées dans des tableaux à peu près identiques avec : signes généraux (tachycardie, sueurs, céphalées, vertiges), signes digestifs (nausée, vomissements, diarrhée), signes buccaux (dysphagie, stomatite aiguë, hypersialorrhée, haleine fétide), TA normale, poly-adénopathie cervicale, puis coma et décès. Les décès sont survenus respectivement 9 jours et 14 jours après le repas en cause. Une autopsie a été réalisée pour l'une d'entre elles et des prélèvements envoyés à l'Institut Pasteur de Madagascar pour analyse.*

• *une femme (âgée de 50 ans) non hospitalisée avait présenté selon ses proches des signes généraux (asthénie, somnolence, fièvre, anorexie), des signes buccaux (stomatite aiguë, dysphagie, ulcération buccale), des signes digestifs (vomissements) et son état s'était dégradé jusqu'à son décès 18 jours après le repas en cause.*

3- Comparaison malades/non malades

Seule la variable "type de morceau consommé" a été associée à une différence statistiquement significative : les sujets qui ont consommé de l'oeuf de tortue ont été plus souvent malades que ceux qui n'en ont pas mangé (57% *versus* 23%, p=0,03).

CONCLUSION

Les signes cliniques décrits dans cette étude sont concordants avec les données de la littérature. Le taux d'attaque d'intoxication clinique sur l'échantillon étudié est de 48%, et le taux de létalité parmi les sujets intoxiqués peut être estimé à 7,7% .

Trois semaines après le repas en cause, la moitié des personnes intoxiquées ont gardé encore quelques symptômes de type dysphagie et stomatite.

La toxicité potentielle de certaines tortues (type *Fanoara*) est connue dans cette région, puisque de tels accidents sont déjà survenus en 1990 et 1993 . Cependant les techniques traditionnelles pour identifier la toxicité chez la tortue en cause n'ont pas été performantes dans le cas décrit.

L'organisation structurée de la surveillance de tels phénomènes semble nécessaire pour en améliorer la connaissance et mettre en oeuvre les mesures préventives adaptées .

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Fiji / W. Samoa

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Hawksbill poisoning thought to be caused by copper pollution. - Honolulu

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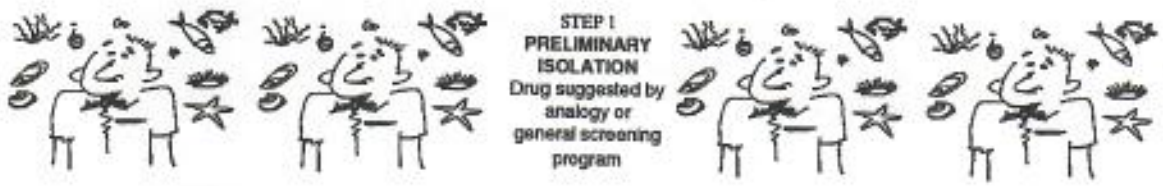


Folk medicine and horticulture

Marine organisms are used in folk medicine by coastal people of northern Mindanao and the Visayan islands of Bohol, Cebu and Negros in central Philippines. Of particular interest are concoctions used to treat malaria, bleeding, fertility problems, and worm infection (table below lists those related to aquaculture). There could be truly effective drugs from marine organisms.

Sea urchins contain bioactive substances. Mangroves contain tannins, phenolic compounds that exhibit antimicrobial activity. Seaweeds contain phenols and terpenoidal compounds.

In horticulture and agriculture, Filipino coastal dwellers have used brown seaweeds as soil ameliorant and growth enhancer. Studies point out that seaweed extracts contain plant growth-promoting substances. These are now exploited commercially. Other studies indicate the possible use of seaweeds directly as fertilizer. In northern Cebu, starfish is used as coconut fertilizer; it reportedly induces blooming and produces healthier trees. Studies has also been made on the use of sea urchins and seaweeds as vermifuge.

Common name	Local name	Treatment or use
Green seaweed	Lato, ar-arucip	For treatment of rheumatism. Seaweed is boiled and its soup drunk. For treating or preventing goiter. Seaweed is eaten raw or prepared as salad.
Green seaweed	Lukay-lukay	For treatment or prevention of goiter or *bugon* and colds. Seaweed is eaten raw or prepared as salad.
Brown seaweed	Boto-boto, lusay-lusay, aragan	For treatment of goiter and other glandular troubles. Seaweed is boiled, the soup drunk. Young shoots may be added to *paksiw* dishes (recipes consisting of fish cooked in vinegar, ginger, garlic and salt).
Brown seaweed	Samo, aragan	Dried seaweeds are burned with or without small pieces of rubber to drive away insects particularly those that infect rice fields. Dried seaweeds may also be hung on trees -- like the *upo* or *Lagenaria siceraria* and the *langka* or jackfruit *Artocarpus integrifolia* -- that are infested with worms. Seaweeds are believed to drive away the insects. These may also be mixed with soil as conditioner or with rice bran as hog feed.
Red seaweed	Gulaman	For treatment of stomach disorders; also used as laxative. Decoction is prepared and drunk.
Red seaweed	Guso, tamsao, cottonii	Decoction is used as foliar spray to enhance flowering and crop growth.

Common name

Giant clam
Black sea urchin

Sea urchin

Sea cucumber

Grouper

Rabbitfish

Green turtle, haw
bill turtle, leath
back turtle

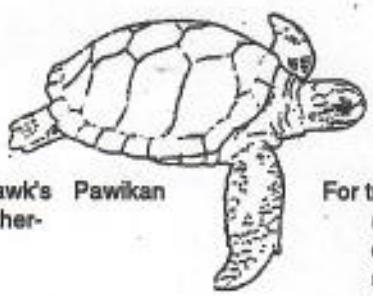
Crocodile
Mangrove

Seawater

Hot mud from po
and mangrove
White sand from
the sea
Oysters

Washing of fish (
and scales)

References: (1) Re
Fortes, WRY Licua
isms. SICEN Leafl

Common name	Local name	Treatment or use
		
Giant clam	Taklobo	For malaria; the meat is eaten raw or boiled.
Black sea urchin	Tuyom, salunggo, tayong-tayong	As pig vermifuge. The fluid inside the urchin test and the aristotle's lantern is used.
Sea urchin		As purgative for man and pigs. Sea urchin is chopped or pounded, boiled, and the soup is drunk.
Sea cucumber	Bahay-bahay, balat	For errant husbands. The sea cucumber is dried, then, placed in the trousers (inside pockets or seams) of the husband. Believed to cause impotence.
Grouper	Lapu-lapu, lig-lig	For faster healing of wounds of women who gave birth. Blood from the tail of the fish is extracted and drunk.
Rabbitfish	Danggit, samaral	For treatment of wound caused by fish spines. Fish liver is applied on the wound.
		
Green turtle, hawk's bill turtle, leather-back turtle	Pawikan	For treatment of asthma. The flesh is cooked *adobo*-style (with vinegar, soy sauce, garlic, laurel leaves, pepper corn and salt) and eaten. The flesh may be broiled slightly burned, placed in water, and the soup drunk. As aphrodisiac. The turtle eggs are believed to stimulate sexual drive.
Crocodile	Buaya	For sexual potency. The sexual organ is cooked and eaten.
Mangrove	Pagatpat, miyapi	For stomach aches. The bark is boiled and the extract drunk or used to wash wounds. Tannins from boiled mangrove is considered an omnipotent medicine. Used as antiseptic.
Seawater	Tubig sa baybay o dagat	Used for faster healing of wounds. During summer in eastern Visayas, newly circumcised boys ages 7-10 bathe in the sea to heal their wounds faster. For treatment of partial paralysis caused by stings of poisonous sea urchins or fishes. Hot seawater is applied on the wound, or the area is immersed in hot seawater.
Hot mud from ponds and mangrove areas		Used for faster healing of wounds and sores. Hot mud is applied on wounds and sores.
White sand from the sea		For treatment of wounds caused by thorns of marine animals. Sand is chewed and applied externally to the wound.
Oysters	Sisi, talaba	Shells are ground and applied to plants as fertilizers or soil ameliorant. Ground shells are mixed with grain for use as feeds for fowls.
Washing of fish (blood and scales)		Applied to ornamental plants to enhance growth and blooming.

References: **(1) Research Gems.** UP Diliman, 1991. UP, Diliman, Quezon City. (2) PM Aliño, GJB Cajipe, ET Ganzon-Fortes, WRY Licuanan, NE Montaño, and LM Tupas. *Some traditional medicinal and horticultural uses of marine organisms.* **SICEN Leaflet 1.**

Farming notes

(with reference to folk medicine and horticulture on pages 6-7, this issue)

Grouper, rabbitfish and oysters

The grouper *Epinephelus coioides*, the rabbitfish *Siganus guttatus*, and the oyster *Crassostrea* spp. are among the 20 or so commodities prioritized for research at SEAFDEC/AQD for 1995-1997. Accomplishments in their farming techniques are discussed in the ADSEA proceedings edited by Bagarinao and Flores (see page 22, this issue).

For the grouper, work on these areas still have to be done: (1) refinement of broodstock management and breeding techniques, (2) studies on reproductive biology and endocrinology, (3) improvement of hatchery and nursery techniques with alternative live food and water management, (4) studies on sources of wild seed, (5) improvement of holding and transport techniques for wild juveniles, (6) determination of nutrient requirements, (7) feed development for nursery and grow-out, (8) health management, (9) documentation and improvement of existing culture techniques, and (10) economics of monoculture and polyculture.

For rabbitfish: (1) refinement of broodstock management, (2) feasibility studies for searanching. (3) market study and socioeconomic assessment of existing industry, (4) cage culture as alternative livelihood, and (5) collation of traditional knowledge on rabbitfish biology, fishery, and culture.

For the oyster: (1) spatfall forecasting, (2) evaluation and refinement of culture technology, (3) transplantation and transfer of culture technology, and (4) product development.

The giant clams

All seven known species of giant clams are found in the Philippines. However, only the smaller attached species *Tridacna crocea*, *T. maxima*, and *T. squamosa* are widely distributed. Of the four unattached species, only *Hippopus hippopus* occurs in some numbers while the other three, *H. porcellanus*, *T. derasa*, and *T. gigas* are virtually extinct due to overexploitation.

Both shell and meat are utilized although the shell is a more important commodity. All species are harvested with women playing an equally important role as the men. The clams are harvested from reef areas which are common property, without any management program at present.

Research on giant clams in the Philippines is undertaken principally by two institutions:

- Silliman University Marine Laboratory in Dumaguete City and
- University of the Philippines Marine Science Institute in Bolinao, Pangasinan

Studies have included resource surveys, growth, and mariculture. These research programs are part of a larger program that include the James Cook University in Townsville, Australia, the University of Papua New Guinea, and the Ministry of Primary Industries in Suva, Fiji. It is supported by the Australian Centre for International Agricultural Research.

Mariculture. Raising laboratory-reared clams in ocean nurseries was tested in 7 sites but most successfully in Pamilacan (an island southwest of mainland Bohol) in central Visayas. The ocean nursery in Pamilacan was within an established marine reserve, inside the reef, which is a mixed seagrass community. The site is partially protected from the monsoon trade winds, which can destroy cages, overturn clams, and chip off shells. *H. hippopus* juveniles (<12 to 14 months old) were placed in a simple fence-like enclosure of nylon net held in place by ipil-ipil or bamboo posts. Survival after 17 months was about 74%; remaining clams grew an average 3.65 mm each month.

Further reading: JW Copland and JS Lucas. 1988. **Giant clams in Asia and the Pacific.** ACIAR Monograph No. 9. 274 pages. (This monograph describes the research conducted by Silliman University and the University of the Philippines, among others.)

The marine turtles

In the Philippines, marine turtles now face a threat to their survival. They are hunted in the sea or slaughtered on the beach as they lay their eggs. The eggs and meat are taken as food while their skins and shells are processed into various by-products.

The government has prohibited the trade of marine turtles or its by-products; regulates the egg collection of communities that have traditionally depended on marine turtles as source of food; declared sanctuaries in Tawi-tawi (the Turtle Islands), Palawan (El Nido, Bacuit Bay), and Antique (Caluya); and deputized provincial officials as conservation officers or game wardens.

Marine turtles live up to 60 years or more. At maturity, the males develop more elongated, curled claws and much longer tails than females. Marine turtles are air-breathing although they can dive for long periods and can swim powerfully. They have no teeth and use a sharp, horny beak and jaws to tear and bite their food. Their senses of sight, taste and touch are well-developed. Due to the absence of vocal cords, the only sounds they can make are hissing, grunting noises while exhaling, usually during courtship and mating.

Of the eight species of marine turtles occurring worldwide, five are present in the Philippines. One species is the green turtle *Chelonia mydas*. The green turtle derived its name from the color of its fat. It has many names in the local dialect but is more popularly called **pawikan** (Tagalog). Adult females measure 54-121 cm carapace length and weigh 113-182 kilograms. It is carnivorous during its first year of life, becoming exclusively vegetarian when adult. Algae and seagrasses are its favorite diet.

The Department of Environment and Natural Resources (DENR) notes that many gaps still exist in the study of marine turtle biology and ecology. So far, only the nesting process of marine turtles has been studied extensively. DENR also maintains hatcheries to protect eggs against poachers and predators during their incubation period. These eggs are collected from beaches. Approximately 500,000 marine turtles have successfully emerged from these hatcheries and have been released to the sea.

DENR enjoins all to contribute and support marine turtle conservation:

- do not kill or injure marine turtles
- do not use dynamite or cyanide when fishing
- do not gather marine turtle eggs
- do not litter or throw garbage in their habitat
- do not buy or sell turtle eggs, turtle meat, stuffed turtles and its by-products such as guitars, combs, bangles, earrings, and rings
- report people engaged in these illegal activities to the authorities
- if you see a turtle with a metal tag in its front flippers, write down the serial number and species, the date and place the turtle was spotted. Never remove the tag; the turtle may be spotted somewhere else and this would help in the research on migratory routes. Send this information to: Pawikan Conservation Project, Protected Areas and Wildlife Bureau, NAPWNC, Quezon Avenue, Quezon City 1100.

Further reading: **Marine Turtles in the Philippines** produced by the DENR. 10 pages.

The "killer" crocs

The fear of crocodiles seems universal, especially for saltwater species that are known to attack humans. Freshwater species has no such record. Crocodiles are among the oldest creatures on earth, having survived 200 million years. They are important in regulating the food chain in aquatic ecosystems, and their feces is known to spur the growth of plants that are eaten in turn by fishes. They are protected under the Convention on International Trade in Endangered Species of which the Philippines is a signatory.

The Crocodile Farming Institute is a joint project of the Governments of the Philippines and Japan through the DENR and the Japan International Cooperation Agency. It is based at Barangay Irawan, Puerto Princesa City in Palawan. The Institute aims to conserve two endangered species of crocodiles -- the Philippine crocodile *Crocodylus mindorensis* and the Indo-Pacific *Crocodylus porosus* -- and to develop farming technology for local communities. By the end of 1992, its croc population had reached almost 1,500 of which 90% were bred in captivity. The Institute believes in the potential commercialization of crocs as a dollar-generating industry given the prevailing prices of its hides ($8 per cm^2 in the world market) that are used to manufacture bags and shoes. Its meat is also considered a delicacy in some countries. Farming crocs has been a profitable venture in Thailand, Australia, and Zimbabwe for several decades.

The Institute built a prototype backyard croc farm inside the institute's 10-hectare complex. The structure is worth ₱18,000 and consists of three adjoining bamboo pens with concrete pools in the middle surrounded by compact soil. The biggest pen is a 25 m^2 enclosure 5 ft high fitted with iron sheets all around as an added precaution.

In 1993, the Institute began pilot testing its community-based farming technique. It provides 25 crocs, each about a year old, to qualified cooperatives who raise the crocs for three years. Breeders provide enough food (leftover fish or meat unfit for human consumption) and water.

Farming crocs is intended to be a sideline (the breeder spends two hours a day to care for them). A breeder can earn ₱77,000 net in three years (US $1 - ₱25).

The Institute publishes a quarterly publication **CFI News**. Send requests to RP-Japan Crocodile Farming Institute, PO Box 101, Puerto Princesa City 5300, Palawan.

original attached

Kinugasa
& Suzuki

A study on the cause of the outbreak of poisoning by sea turtle meat in Taiwan

Masaru Kinugasa health technician
Wasaburo Suzuki

Forward

In 1939, 4.26, about 1:00 AM. 2 fishermen caught a sea turtle at an off-shore of Gongu beach, Shinchiku state in Taiwan and served it among friend.

As soon as the news of poisoning was informed we rushed there and did the investigation.

Out of 57 poisoned, 7 died, 9 serious 41 minor.

We found a certain poison. Thus it seems possible that some sea turtle has a certain poison.

1. The clinical symptoms of the patient

a) The symptoms of the not serious patient

Many of them felt sick and heavy head only.
The mildest cases were heavy head and sick feeling.
But some of them vomited. A less mild cases were dryness inside the mouth, few diarrhoea.
The pulse was generally slow. None had fever.

were quite swollen and scattered spots of effusion of blood along the vessels generally. The mucous membrane were rough and inflamed but no ulcer were found. There were some blood in the food in the stomache, some had bile in it. The blood vessels on the mucous membrane were considerably swollen.

Intestines

The most obvious change were observed in this area. In the small intestines, the blood vessels were considerably swollen everywhere, scattered spots of conjestion, and in every case there were some spots of effusion of blood. Some were like lines, circles or oval shape, from 2 cm diameter to the size of a hat pin, all the kinds of sizes and shapes. Many were seen at the upper part of the small intestine. The duodenum were filled with bile.

Heart

The surface had slightly swollen coronary arteries. Inside had some fluid blood. The valve and the mucous membrane had spots of conjestion, but no unusual change.

Liver

The surface and inside had map-like or tree branch-like conjestion.

Kidney

not unusual

4. With all the circumstances, the sea turtle was believed to be not poisoned

5. The sea turtle was found not rotten.

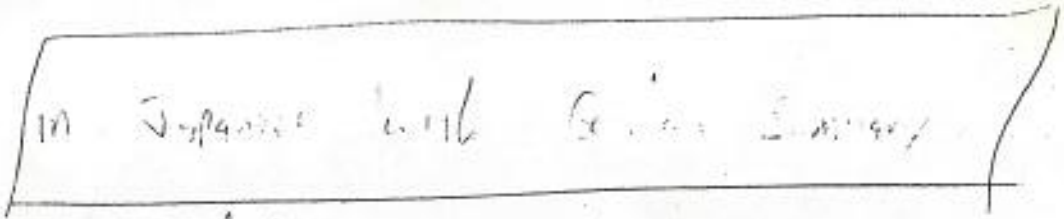
6. The bacteriological examinations were negative.

7. The chemical examination
There were no salmonella family.
But a certain toxin was admitted, however it was not clear what it was.

8. Animal experiment
The toxin stimulates vagus nerves with a small amount, and slows down the heart beat. With large amount, the vagus nerves will be paralized and heart beat gets faster.
With extreme amount, the heart stops, probably the heart muscle become puralized.

over.

Kinugasa, M. and W. Suzuki, 1940. Über untersuchungen der ursache der massenhaften vergiftung nach dem genuss von fleisch einer an der küste von koryo in der prafektus sintiku gefangenen seeschildkröte. Taiwan Igohkai Zassi 39(74): 66-74.



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臺灣新竹州後龍海岸ニ於テ捕獲セル海龜肉ノ食用ニ因ル多數中毒發生ノ原因檢索ニ就イテ

新竹州警務部衛生課(課長下村博士)
衛生技師 衣笠勝
鈴木和三郎

(昭和14年10月10日受付)

目錄

緒言

昭和14年4月26日午前1時頃臺灣新竹州竹南郡後龍庄外埔海岸沖ニ於テ、同所居住者漁師許大炮及朱某兩名ノ捕獲セル或種海龜肉ヲ食用セルガ、多數(57例、內死亡7、重症9、輕症41)ノ中毒者ヲ出セリト報セラレタルヲ以テ、余等ハ時ヲ移サズ現地ニ急行シ、其中毒原因ニ就キ本論ノ如キ調査ヲ遂ゲタルニ、一種ノ固有毒ヲ認メタリ。仍テ或ル種ノ海龜中ニハ一種ノ固有毒ヲ保有シ居ル可キコトヲ考察シ得タルヲ以テ茲ニ報告セントス。

第1章 中毒患者ノ臨床症狀ニ就イテ

(a) 輕症者ノ症狀

輕症者ノ症狀ハ單ニ惡心竝ニ頭重ヲ訴フルニ過ギザルモノ多ク、特ニ神經質ノ患者ニアリテハ多數ノ重症者竝ニ死者アルヲ見聞シテ、精神不安ヲ感ズルモノ少カラザルガ、之等ノ者ヲ除キテ思考スルニ、極輕度ノ場合ニハ頭痛、惡心ノミナルモノ及嘔吐ヲ催セル者アリ。更ニ稍々症狀ノ強キ者ハ、口腔內ノ乾燥感アルモ下痢ヲ來セルモノ少シ。脈ハ一般ニ遲脈ヲ呈スル者多キモ、發熱セル者1例モナク、肝、脾ヲ觸レズ、神經系統ハ僅ニ瞳孔ノ散大セル者少數ニ認メタルニ過ギス。其他眼球及耳鼻咽喉科領域ノ變化及炎症々狀竝ニ皮膚溢血等ヲ認メズ。又黃疸ヲ認メタル者ナシ(第一表參照)。

第一表の一 中毒者ノ臨床的症狀表（輕症者）

氏名	性	年齢	自覺的症狀	他覺的症狀	備考
呂氏來好	女	61	昭和14年4月26日午後6時食同28日朝發病、頭重、口腔咽頭乾燥感時ニ嚥下時軽度ノ胃部苦悶	熱ナシ Milz (−) Leber (−) 口腔、咽頭舌一帯發赤ス	前揭ノ外陳氏虫♀ 11 歲 朱和技♂ 18 歲 黃氏香♀ 3 歲 林氏江♀ 14 歲 陳 乾♂ 22 歲 陳光得♂ 26 歲 陳氏富♀ 15 歲 朱 木♂ 6 歲 陳氏歎早♀ 7 歲 黃氏朱♀ 9 歲 陳有得♂ 66 歲 ハ惡心頭重、鼻咽頭ニ乾燥感アリ、他覺的ニハ熱ナク脈ハ臨診時ニ緩除ス
張氏猫	女	13	症狀前者同様ニシ稍々重症ニテ一時ハ意識不明ノ時アリシモ漸次回復ス	熱 36.9 脈 電氏ノ時ハ脈搏 140 回復後遂ニ脈搏 62 ヲ算ス	
鄭氏燕	女	13	4月26日正午食28日早朝發病頭重ノ口腔咽頭乾燥感頭重胸内苦悶	熱ナシ 脈 60 Milz (−) Leber (−)	
朱永盛	男	15	頭重惡心アルモ他ニ異常感ナシ	熱 35.5 脈 60 Milz (−) Leber (−)	
陳氏玉靈	女	12	前記症狀ニ一致ス	熱 36.5 脈 58	
陳氏球	女	9	前上ノ他口腔、咽頭ニ熱感アリ	熱 36.6 脈 56 Milz (−) Leber (−)	
呉氏	女	11	同前	熱 36.5 脈 58 Milz (−) Leber (−)	
洪氏粉	女	14	同前	熱 35.8 脈 50	
朱氏紫琴	女	7	前上ノ他惡心嘔吐、四肢厥冷、頭重、鼻汁、眩暈、一時起坐不能	熱 37.0 脈 74 Milz (−) Leber (−)	
朱登山	男	10	同前	熱 37.0 脈 78 Milz (−) Leber (−)	

第一表の二 中毒者ノ臨床的症狀表（重症者）

氏名	性	年齢	自覺的症狀	他覺的症狀
朱氏玉銀	女	17	4月26日正午食同28日發病惡心、嘔吐、鼻汁流出、眩暈、舌痙攣、口腔ニ涎シキ灼熱感及疼痛感アリ、四肢末端ノ麻痺感即チシビレ感強ク一時起坐不能	顔面蒼白、一般症狀重篤、顔面冷汗額頭ヨリ流下ス 熱 37 度脈ハ 140 内外ヲ算スルモ緊張良不止ハ 3—7—10 ノ間隔ニ変ル
趙氏偸	女	32	前記同様時ニ四肢末端ノシビレ感强シ	同前
黃氏好	女	38	前記ノ外胸内苦悶强シ	同前
朱氏香	女	11	多量ニ食ス前記ノ外一時意識不明ニ陥リ四肢末端屈曲ノ痙攣アリ	顔面蒼白、口過チアノーゼ顔面特ニ前額ノ盗汗滿シ 熱 37.1 脈搏數 150 微弱
朱氏蘭	女	18	同前	熱 37.2 脈搏數 192
趙氏足	女	20	同前	

(b) 重症者ノ症狀

各例ニ就イテ多少ノ差アルモ、大體ニ於テ熱發ハ存在スルモ其ノ程度ハ極ク輕度ニシテ、熱發

ヲ自覺スル者少ナシ。顕症ニ頭痛、口腔、舌帶、咽頭ノ乾燥感灼熱感ハ共ニ殆ンド全例ニ於テ認メラレ、又惡心、嘔吐モ强弱ノ差ヲ有スルモ、殆ンド全例ニ之ヲ有セリ。脈搏ハ本症候群中特有ナルモノニシテ、發熱ニ比シ遲脈ヲ呈スル事、恰モ腸チフス」ノ症候ニ正反スルノミナラズ、興味アルコトハ猛症者ノソレト全ク相反スル作用ヲ見ルコトナリ。即チ體溫 37°C 内外ニシテ脈搏 150 ヲ算スル者少カラズ、季肋下部ニ於テハ膨滿感アリ、下腹部ニ於テハ腸蠕動運動ヲ喚起スルモノヽ如ク、雷鳴ヲ聞カザルモ下痢ヲ有スル者アリ。口腔内ノ乾燥感、鼻咽頭ノ乾燥感、全身冷感、四肢厥冷ヲ併有スル者少カラズ。又他覺的ニモ之等ハ粘膜面ニハ溢血斑ヲ伴ハザルモ、一般ニ充血セリ。神經症狀トシテ瞳孔散大ヲ來シ、又瞳孔反應微弱、其ノ他全身違和感、重態感强シ、脾ハ觸知セザルモ肝ハ一横指乃至二横指ヲ呈セル例アルモ、一般ニ必發的ニ非ザルガ如シ。同部ノ壓痛ハ各例トモ陰陽共ニ明瞭ナラズ。尿ノ檢査ヲ爲ス機會ヲ逸シタルハ甚ダ遺憾トスル所ナリ。大便モ入手セラレザリシガ、本中毒死ニ因ル解剖死體ヨリ小腸内ノ内容物ニ就テ後述ノ細菌學的檢査ヲ施行セリ。尚膀胱内蓄溜尿ヲ採リ蛋白ヲ檢シタルモ陰性ナリキ。

第 2 章　中毒症狀ノ發生及其ノ死亡ニ至ル迄ノ時間

紫蟹肉食用ノ時ヨリ初發症狀、即チ自覺症狀ヲ來ス時間ハ各例一様ナラズ。早キハ 8 時間、遲キハ數 10 時間ヲ經テ發スル例アリテ平均値ヲ求メ難シ。然レドモ大體ニ於テ平均 16 時間ヲ要スルモノ多シ、是レ個人的體質或ハ食用ノ多少及性、年齡ノ相違ニ因スルハ勿論ナリ。尚死亡ニ至ル時間ニ就テハ、更ニ區々ニシテ發病ニ至ル期間ハ 24 時間ヲ經過セルモ、發病後僅ニ 3 時間 30 分ニシテ死亡セル例アリ。或ハ 80 時間ヲ經過セルモノ或ハ夫レ以上ニ及ブ者アリテ一様ナラザルモ、一般ニ死亡ニ至ル時間ハ短カラザルガ如シ。勿論强心、解毒等ノ適當ナル醫治ヲ加ヘタルニモ拘ラズ、死亡セル者ノ平均時間ハ 43 時ヲ要セリ。之ヲ要スルニ本疾患ハ自覺的ニハ頭重、口腔咽頭ノ灼熱感、胸部壓迫感ヲ有スレドモ、他覺的ニハ著ルシキ症狀ヲ缺除シ、只僅ニ一般ニ顏面蒼白、發汗ヲ見ルノミナルガ、時ニ重症者ニアリテハ四肢厥冷セリ。而シテ最モ特異トスル處ハ、一般ニ熱ハ缺除スルカ、若クハ極輕度ノ上昇ヲ示スニ過ギザルニ反シ、脈ハ速脈ヲ呈スル者多キコトナリ。如斯血行系ニ於テ著名ナル所見ヲ呈スルモノヽ如クニシテ、即チ發熱ニ比較シ心音頻數ナルコトナリ。恐ラクハ著ルシク本毒作用ノ發現ヲ見シカ、遂ニハ全心筋麻痺ノ經過ヲ取ルニ非ズヤト信ゼラル。即チ本臨床的所見ヨリシテ本毒ノ作用ハ其ノ始メ迷走神經ヲ鼓舞シ後之レヲ麻痺スルカ、或ハ交感神經ヲ鼓舞シ後之ヲ麻痺スルカニアルベシ、或ハ又心筋自體ニ前記ノ如キ作用ヲ呈スルモノナラン。而シテ前記ノ臨床的症狀並ニ既知動物毒ノ性質ヨリ推理スルニ恐ラクハ其ノ微量ニ於テ迷走神經ヲ鼓舞シ、後之ヲ麻痺スルノ性ヲ有スルモノニ非ラザルカ。本論ニ就キテハ動物實驗ノ條下ニ之ヲ論ズ。

第 3 章　中毒死體ノ病理解剖學的所見

3 例（其ノ一、朱登英當 3 年、死後 23 時間、其ノ二、朱氏雲秧當 9 年、死後 16 時間、其ノ三、許氏楡當 11 年死後 12 時間）ニ就テ之ガ總括的所見ヲ記ルニ左ノ如シ。

胃、表面ハ一般ニ小灣部ニ於テ怒張セル血管多數ニシテ而モ其ノ血管ニ沿ヒ局圍ニ溢血斑散在セリ。一般ニ胃粘膜面粗鬆ニシテ且ツ腐爛セルガ潰瘍ヲ認メタルモノ 1 例モナク溢血斑及胃内容物ニ血塊ヲ認メ、又黒汁ヲ滿タセルモノアリ。胃粘膜面ニ配布セル血管ハ著シク怒張セリ。

腸、本臓器ニ於テ病的變化最モ著明ニシテ、特ニ小腸ニ於テ主ル所配布血管ノ怒張、充血斑ヲ散在性ニ認ムルノ他各例トモ溢血斑スラ存在セル箇所ヲ發見ス。其ノ形狀及大サハ或ハ線狀ニ或ハ圓型ニ或ハ楕圓型ニ。大ナルモノハ直徑 2cm ニ及ブモノアリ、小ナルモノハ縫針頭大ニ至ル迄デ千差萬別ナリ。特ニ小腸ニ於テハ上部ニ多ク認メラルヽモ廻盲部ニ於テモ認メラル。尚十二指腸部ニハ膽汁ヲ以テ充滿シ腸壁切開ヲ行フヤ、多量ノ膽汁流出セリ。（寫眞參照）

心臓、表面ハ冠狀動脈稍々怒張ス其他所見少ク、内面ハ尚少量ノ流動性血液ヲ保有ス。水洗後心内膜面ヲ診ルニ著ルシキ所見ヲ認ザルモ、瓣膜及粘膜面ニ充血斑ヲ認ムルモ割面ニ著變ナシ。

肝、表面及内面共ニ地圖狀或ハ樹枝狀ノ充血斑ヲ認ム、割面ハ溢血斑ト思ハル、箇所アリ。

腎、一般ニハ混濁尿ヲ認メザルモノ多シ。表面、割面、内面共ニ異常ヲ認メズ。

第 4 章　海龜肉内ニ他ノ毒物ノ混在アリヤ

何等カ怨恨關係ニヨリ他殺ノ目的ヲ以テ他ノ毒物ヲ混在セシヤ否ヤニ就イテハ、先ヅ實情調査ヲ論理的ニ行フニ午前 1 時頃地曳網ニカヽリシ該海龜ヲ許、朱兩名共同ニテ捕獲シ得タルガ兩名ニ於テ等分シ（目前ニ於テ）許、朱ハ其ノ家族ニ食用セシメタル事實ニシテ、特ニ許大炮ハ自身之ヲ食用セリ。朱某ハ自ラ捕ヘタル大ナル動物ハ之ヲ食セザル習慣ヲ持シテ食セザリシモ、然カモ自己ノ妻、娘其ノ他家族全部ニ之ヲ食用セシメタル外、近在ノ者特ニ親シキ者ニ之ヲ分與セリ。其ノ他怨恨關係ナキ事實並ニ今日迄同樣或種無毒ノ龜ノ食用ニ就イテモ、略今回同樣ニ行ヒシ事數度ニ及ビ、且ツ彼等ノ自宅及附近ニ斯ル毒物ノ存在ナシ。仍テ當時ノ實情ハ推理的ニ斯ル毒物ノ混在ヲ否定シ得ルモノナリ。

第 5 章　海龜肉ノ單ナル腐敗ニヨル中毒ニ起因スルヤ

該龜ヲ捕獲（4 月 26 日午前 1 時）シ屠殺（午前 9 時）後食用ニ供セル時間ハ前後ヲ通ジ、僅ニ 3 時間ニシテ、然カモ當時北臺灣ノ 4 月 26 日ナルヲ以テ氣温低ク推定温度 18 度内外ナリ。且ツ余等 4 月 27 日午後 7 時頃龜肉料理ノ殘品ニ付キ檢査スルニ未ダ腐敗シ居ラザリキ。

第 6 章　細菌學的感染ノ有無ニ就イテ

食用ニ供セル龜肉ノ尚殘存セルモノ竝ニ重症者ノ血液及該中毒死體ノ胃、腸、腎ノ內容物ヲ檢體トシ、先ヅ遠藤氏培養基竝ニ普通寒天培地ニ移シ、之ヲ孵卵器內ニ(攝氏 37 度)納メルコト 24 時ニシテ生ジタル「コロニー」ヲ見ルニ、Salmonella 族ト思ハルヽモノ認メザリシモ、確實ニ大腸菌ト思惟セラルヽ「コロニー」ヲ除キ、全コロニー」ニ就キ綿密ニ既知血清及重症者朱氏第 18 歲、趙氏足 20 歲ナル者(何レモ女性)ノ血清トノ間ニ就キ凝集反應ヲ試ミシニ、悉ク陰性ナリシガ、更ニ中毒死體ノ腸內容物タル檢體ヨリ生ゼル「コロニー」ニハ假性凝集(腸チフス)ヲ呈セルモノアリタルヲ以テ、更ニ緻密ニ型ノ如ク鑑別培養ニ附シタルニ、之ヲ否定シ得タリ(第2表參照)。

第 2 表ノ一

氏名	朱玉葉	趙氏足	朱氏雲紀		朱登英		許氏織				備考
檢査物	血液	血液	胃內容物	腸內容物	胃內容物	腸內容物	胃內容物	腸內容物	腎內容物	料理セル龜肉	(−)ハ「コロニー」ノ發生ナキモノ (+)ハ「コロニー」ノ發生ヲ示ス ブイヨン及膽汁ハ遠藤、普通寒天、血液寒天ノ各盤ニ移植檢査セリ
培養基ノ種類 遠藤氏寒天	−	−	+	+	+	+	+	+	+	+	
血液寒天	−	−	+	+	+	+	+	+	+	+	
普通寒天	−	−	+	+	+	+	+	+	+	+	
ブイヨン	−	−	+	+	+	+	+	+	+	+	
牛膽汁	−	−	+	+	+	+	+	+	+	+	

第 2 表ノ二

氏名	菌種	血清 朱氏玉葉	趙氏足	チフス	パラA	パラB	パラC	パラK	赤痢混合	ニユーポート	ロンドン	ミユラトシヨテト	馬流産菌	ブレスラウ	スタンレー	ゲルトネル混合	豚コレラ混合	鼠チフス
朱氏雲紀	胃檢出菌	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
	腸檢出菌	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
朱登英	胃檢出菌	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
	腸檢出菌	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
許氏織	胃檢出菌	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
	腸檢出菌	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
	腎檢出菌	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
—	料理セル龜肉	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
備考	試驗管內 50 倍ヨリ 800 倍迄稀釋凝集ヲ行ヘリ																	

第7章 化學的檢索

1) 中毒ノ原因ナリト認メラル、食物。

午前時ニ屠殺シ、許、朱、兩家ニテ分チ肉ト內臟トヲ一緒ニ少量ノ落花生油ト共ニ煮シ鹽及醬油ヲ以テ調味セルモノナリ。

2) 殘食物ノ形態。

多量ノ黃色脂肪樣物質ノ下ニ黑褐色ノ少量ノ液ヲ有スレ共他ハ鷄卵大ニ切リタル肉片ニシテ味覺ヲ刺戟スル臭ヒヲ有シ腐敗臭ナク弱酸性ヲ呈ス。

3) 食物殘品ニ對スル毒物檢索。

A. 本品ハ微ニ酸性ヲ呈スルヲ以テ「アルカリ」ヲ以テ中和シタル後酒石酸々性トナシ水ヲ加ヘテ重量敷上ニ致シ後濾過シタル酸性ノ液ニ就キ、「スタース、オット法ニヨリ次ノ操作ヲ遂ゲ。

第一　酒石酸々性液ニ「エーテル」ヲ加ヘ浸出シ若クハ抽出ス。

第二「エーテル」ヲ以テ浸出シ盡シタル酸性ノ水液（第一）ニ「ナトロン溶液ノ過剩ヲ加ヘ再ビ「エーテル」ヲ以テ浸出シ抽出ス。

第三「エーテル」ヲ以テ浸出シ盡シタル「アルカリ性ノ（第二液）ニ「クロールアンモン」ヲ加ヘテ後、「アミールアルコール」ヲ以テ浸出ス。

第四　第三ノ殘液ニ炭酸瓦斯ヲ通シ砂浴ニ乾固シ其ノ殘渣ヲ「ソックスレット浸出裝置ニ納メ、「アルコール及クロ、ホルム兩液ヲ以テ抽出ス。

以上四種ノ抽出シタルモノヲ夫々通法ニヨリ精製シタル液、順ヲ追ヒ精密ニ夫々常法ニ從ヒ檢索セルニ、何レモ反應陰性ナリキ。

B. 可檢物 200 g ヲ同量ノ稀酒精ヲ以テ 24 時間抽出セル酸性ノ液ヲ重量敷上ニ蒸發シ數回通法ニヨリ、精製セルモノヲ 500.00 cc ノ水ニ溶解シタル液ニ就キ次ノ反應ヲ試ム。

I. 本液ノ 10.0 cc ヲ「フエリング溶液中ニ入レ熱スルニ之ヲ還元シテ赤褐色沈澱ヲ生ズ。

II. 本液 20.0 cc ニ「アンモニア性硝酸銀溶液ヲ加ヘ熱スルニ黑褐色ヲ呈ス

C. 更ニ可檢物ヲ常法ニ從ヒ有機物質ヲ破壞シタル液ニ無機毒ノ檢索ヲ試ムルモ何レモ之ヲ認メズ。

以上化學的操作ノ結果ニ徵スルニ、本食物中ニハ「アルカロイド及プトマイン竝ニ無機的毒物ヲモ之ヲ證明セサルモ、酒精ニヨル抽出物中 B ノ試驗ニ對スル反應ハ一種ノ「トキシン」ノ存スルコトヲ認メ得タリ。

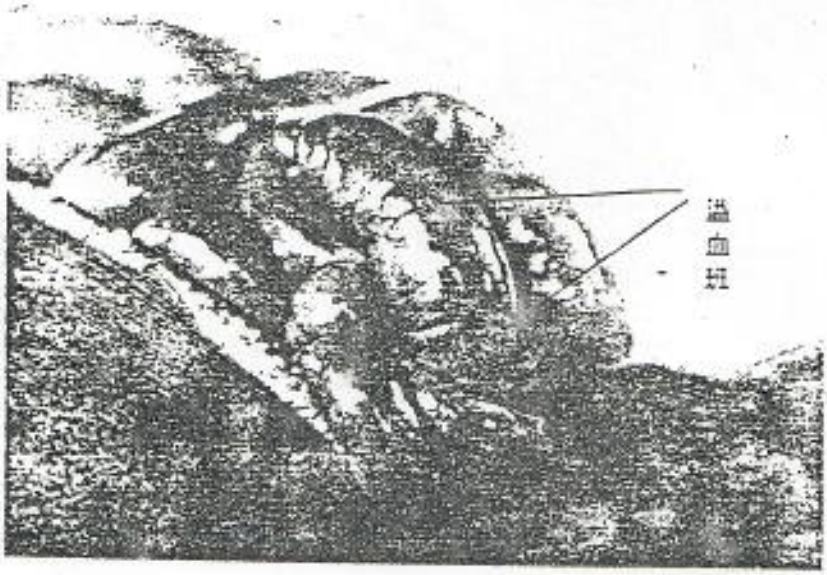
第8章 動物實驗

可檢物ヲ試驗液トシ、動物實驗ニ移セリ。使用セル實驗動物ハ海豚體重一頭ハ 1.5 瓩、他ノ一頭ハ 1.3 瓩ニテ都合二頭及ビ一頭ノ「マウス竝ニ鯽六匹ニ就キ實驗セリ。可檢毒試驗液ハ浸出毒微量（$^{1}/_{100}$）ナルヲ以テ（中毒者自身及家族ノモノヽ言ヲ以テ推理スルニ恐ラクハ鷄肉ヨリ來

ロ內臟器中ニ多ク含有セラレ居ルモノナランカ）可及的可檢毒試驗ヲ多量ニ使用セザルベカラズ。從ツテ小動物マウス等ヲ可トスルモ前章ニ於テ既述セルガ如ク、本中毒例ノ臨床的所見ヨリ考察スル時ハ、特ニ本毒ハ其ノ血行系ニ作用スルガ如ク，思考セラルヽニ就キ、蛙ノ心臟ニ就イテ本可檢毒試驗液ノ作用ヲ觀察セリ。而シテ可檢毒液ハ料理ヲ施セル該鼈肉ヨリ製セル B 液ヲ以テセリ。該液ニ 20.0 cc ヲ海猿ノ皮下ニ注入シ、對照トシテ他ニ 1.45 瓩ノ海猿及 1.3 瓩ノ海猿ニ生理的食鹽水ヲ同樣 20.0 cc ヲ皮下ニ注入セリ。注入後可檢毒液ヲ注入セル一號及 2 號ハ共ニ殆ンド時ヲ同ジウシ、約 15 分ニ於テ數回ノ嘔吐ヲ來セルモ、尙斃死ニ至ラズ。然ルニ對照海猿 3 號及 4 號ニ於テ何等カヽル症狀ヲ見ザリキ。「マウス」ニ於テハ可檢液 2.0 cc ヲ注入セルガ見ル可キ變化ナシ。仍ツテ翌日更ニ同液 10.0 cc ヲ 1 時ニ皮下ニ注射シタルニ 30 分後ヨリ不快症狀アルガ如ク、前後左右ニ運動スルノミナラズ、口邊ヲ前手ヲ以テ恰モ拭フガ如キ運動ヲナセルガ約 20 分ニシテ數回ノ嘔吐ヲ來セリ。（但シ內容物ニ乏シク粘液樣水樣液及胃液ノ他少量ノ顆粒狀食物殘渣ヲ吐出セリ。）其ノ後著シキ變化ナク 30 時間後ニ於テ斃死セリ。剖檢セルニ海猿ニ於テハ 2 頭トモ內臟ニ變化ヲ認メズ。「マウス」ニ於テハ肺、心臟、小腸ニ充血斑ヲ認メタルモ著明ナラズ。然カモ對照マウス無キヲ以テ變化ノ程度ヲ云々シ難ク、且ツ組織標本ヲ作成セザリシヲ以テ詳細不明ナリシハ遺憾トスルトコロナリ。又蛙 6 匹ニ夫々 1 號ヨリ 6 號ノ番號ヲ附シ 1 號、2 號、3 號、4 號ハ之ヲ抽出セル粘液（B 液）ヲ注射シ、5 號、6 號ハ之ヲ對照トシテ使用セリ。而シテ該蛙ヲ 1 號及 2 號ハ大腦ヲ切除シ、他ノ 3 號、4 號ハ「クロロホルム」麻酔ヲ行ヒテ腹壁ヲ切開シ心臟ヲ露ニ附著ノマヽ見易キ樣露出シ實驗セルガ、始メ可檢毒液ヲ 0.5 瓦筋肉內ニ注入後直チニ心臟運動稍々緩徐トナリ、更ニ心臟附近ニ倍量ヲ注入スル時ハ、更ニ緩徐トナルガ如ク更ニ同量ヲ心臟內ニ注入スル時ハ、遂ニ心運動ハ遲トナリ脈搏數ハ注射前ニ比シ多數ヲ算スルガ如シ。然レドモ之ヲ心筋內ニ前記實驗量ノ 10 倍以上ヲ注射スル時ハ、又緩徐トナル。更ニ可檢毒液 10.0 或ハ 15.0 ヲ與フル時ハ遂ニ心臟ハ其ノ運動ヲ 1 時停止ス。而シテ時間ヲ經ルニ從ヒ再ビ諏動ヲ始ルモノアリ。此ノ場合漸次諏動强クナリテ、遂ニ逆ニ諏動ハ注射前ヨリモ速ニナルニ至ル。然レドモ甚ダレキ多數ヲ與フル時ハ、遂ニ心臟ヲ麻痺スルガ如シ。此ノ時ハ內臟器特ニ肝表面及ビ腸壁ニ出血斑ヲ生ゼリ。

以上ノ事實ヨリ考察スルニ該トキシン」ハ矢張リ先ノ推定通リ、其ノ少量ニ於テ運走神經ヲ刺戟ス。從ツテ心動緩徐トナリ更ニ其ノ毒量多量ニシテ其ノ刺戟强キトキハ運走神經ヲ麻痺シ、反ツテ心動ヲ速カナラシムガ如シ。然レドモ實驗ノ示スガ如ク超多量ヲ注射スル時ハ、又再ビ心動停止スルニ至ルハ恐ラク心筋自體ノ麻痺ヲ招來スルモノヽ如シ。而シテ實驗ニ使用セル 1 號、2 號、3 號、4 號ハ共ニ其ノ大腦切除ヲ行ヘタルト又「クロロホルム麻酔ニヨリタルモノモ其ノ毒物ノ實驗的成績ハ完全ニ相一致ス。

圖附文論木鈴笠衣



總　括

1) 或種海鼈食用ニヨル食餌中毒ニ就キ科學的原因探索ヲ行ヘリ。

2) 該海鼈肉ニ他ノ毒物混在ノ有無ヲ檢シ、其ノ然ラザルヲ確メ得タリ。

3) 該海鼈肉ノ細菌學的感染ノ有無、特ニ Salmonella 族ヲ求メタルモ、其ノ然ラザルヲ確認セリ。

4) 該肉中毒患者ノ臨床的症狀ヲ或程度ニ明瞭ニシ得タル結果ハ其ノ原因ノ中毒ニヨルヲ思ハシメタリ。

5) 中毒死體ノ剖檢的所見ヲ求メ其ノ中毒ニヨルモノトノ信念ヲ高メタリ。

6) 化學的ニ該鼈肉内ヨリ詳細不明ナルモ「トキシン樣毒物ヲ檢出シ得タリ。

7) 動物實驗ノ結果ハ前項化學的試驗成績ニ一致セルガ如キ作用ヲ認メタリ。

結　論

以上ノ檢索ノ結果ヲ綜合スルニ本中毒原因ハ海鼈體内ニ存在セシ毒物ニ基因スルモノニシテ其ノ毒物ハ恐ラク一種ノ「トキシン」ナル可シト判定セラル。仍テ或種海鼈中ニ「トキシン樣物質ヲ保有シ居ルカ又ハ或ル期間之ヲ保有スルモノナル可シト考察シ得ラル、處ナリ。尙本調査研究ハ何分事件發生地ガ遠隔地ナル事、急ヲ要スルヲ以テ尙研究ニ充分ナラズシテ、誠ニ輕下痢樣ノ感アルヲ遺憾トスル處ナルガ、他日更ニ之ガ詳細ナル調査研究ヲナサン。

文　獻

1) 下條久馬一，今日ノ「バクテアス論，日本傳染病學會雜誌 第11卷 第10號及第12號 (昭和11年7月—同13年9月)　2) ロベルトヲツトー，動物試驗法　3) 丹波敬三，微判化學　4) 目野五七郎，和漢藥物學　5) 藤島廉太，藥物學　6) 原三郎，實驗病理學本草新目　7) 竹內松次郎，細菌學及免疫學　8) 食餌中毒トプロトイス屬ノ相互的關係ニ就イテ日本傳染病學會雜誌第10卷 田村正五郎　9) 杉田慶介，食餌中毒例ヨリ檢出セル「プロテウス屬類似一新菌型ニ關スル研究 慶應醫學會雜誌 第37卷 第2號 昭和13年3月

Über Untersuchungen der Ursache der massenhaften Vergiftung nach dem Genusz von Fleisch einer an der Küste von Koryo in der Präfektur Sintiku gefangenen Seeschildkröte.

Von

Masaru Kinugasa und Wasaburo Suzuki.

Aus der Hygienischen Unterabteilung, die Abteilung für Polizeiangelegenheiten.

Es trug sich in der letzten Zeit zu, dasz viele Vergiftungsfälle (57 Fälle, von denen 7 starben, 9 schwer und 41 leicht erkrankten) nach dem Genusz von Fleisch einer in der Nähe an der Küstengegend Koryo (Kreis Tikunan) in der Präfektur Sintiku gefangenen Seeschildkröte (Karette?) entstanden, und dasz wir bald nach dem Empfangen der Nachricht über dieses Ereignis nach dem Entstehungsorte der Vergiftungsfalle eilten, um von verschiedenen Seiten die Fälle zu untersuchen und folgende Ergebnisse zu erhalten:

1. Wir machten chemische, in solchen Fällen erforderliche Untersuchungen.
2. Die Untersuchung der Reste des gekochten, in Betracht kommenden Fleisches auf die anderen, etwaigen, absichtlich gemischten Gifte und das Verhör der damaligen Sachlage von den Patienten ergaben, dasz kein Gift darin gemischt war.
3. Wir untersuchten das Restfleisch der betreffenden Schildkröte, das Blut von damit vergifteten Patienten und den Inhalt von Magen, Darm und Nieren der Todesfälle auf etwaige Bazillen, besonders auf Salmonellagruppe, und konnten feststellen, dasz die Folge der Untersuchungen negativ ausfiel.
4. Wir konnten auch die klinischen Symptome der betreffenden Patienten bis zu einem bestimmten Grade klar stellen; sie deuteten uns an, dasz die Ätiologie der Erkrankung in Vergiftung zu suchen ist.
5. Obduktionsbefunde lieszen unser Glauben an Vergiftung immer fester machen.
6. Chemisch konnten wir eine Art von einem Toxin ähnlicher Giftsubstanz, deren nähere Eigenschaften noch nicht zu erklären sind, in dem Restfleisch herausfinden.
7. Die Ergebnisse des Tierversuches lieszen die Wirkung erkennen, welche mit der Folge der obengenannten chemischen Untersuchung übereinstimmte.

Schluszfolge:

Aus den obenerwähnten Ergebnissen läszt es sich schlieszen, dasz die Ursache der betreffenden Vergiftung auf das Gift im Fleisch der Seeschildkröte zurückzuführen ist, welches vielleicht als Art Toxin angenommen wird. Daher wird es auch vermutet, dasz das Fleisch einer Art Seeschildkröte entweder immer oder in einem gewissen Stadium einen Toxin ähnliechen Stoff enthalten werde.

Unsere Untersuchungen sind leider nicht vollständig angestellt worden, weil die Sache in einem abgelegenen, im Verkehr nicht begünstigten Orte geschah, und der Bericht darüber Eile beanspruchte, um allgemeine Aufmerksamkeit auf diese Sache richten zu lassen. Wir werden künftig Gelegenheit haben, von neuem dieselbe Sache noch näher zu untersuchen.

U.S. DEPARTMENT OF COMMERCE
National Oceanic and Atmospheric Administration
NATIONAL MARINE FISHERIES SERVICE
Southwest Fisheries Center Honolulu Laboratory
2570 Dole St. • Honolulu, Hawaii 96822-2396

June 29, 1987 F/SWC2

Dr. K. E. G. Krishnan
Consultant Physician
The Ministry of Health
Labasa Hospital
Fiji

Dear Dr. Krishnan:

I am most appreciative for your letter of 9 June 1987 which provided substantial detail on the recent Fijian turtle poisoning tragedy. Thank you very much for taking the time to respond to my inquiry in such a comprehensive manner.

Turtle poisonings of this type are known to have occurred sporadically and quite unexpectedly at widely separated locations over the past two centuries. In addition, certain Pacific islanders, such as the Hawaiians, have traditionally called the hawksbill turtle "kapu," thereby suggesting it was not suitable for consumption. Unfortuantely, as the excerpts I sent you indicated, almost nothing is known about what causes a turtle to be poisonous, nor what constitutes the best treatment for the victims.

The case in Fiji is exceedingly well documented as the result of your fine efforts. Consequently, I would like to see this information made available to a larger audience. I wonder if you would permit me to have these facts published in the Marine Turtle Newsletter? Naturally you would receive full credit. With a brief introduction and minor modification, the letter you sent me could be published as written. There are no set style standards for the Marine Turtle Newsletter, and notes of an informal nature are frequently printed.

If you could allow me to submit this information, there are two points that it would be good to clarify.

1. Approximately how large was the hawksbill? Since 43 people shared the turtle, it would appear to have been fairly large, although each portion could have been very small. Would the turtle have been 100 or 200 lbs in weight or even larger? Any estimation of size that you can provide will be valuable to include in the documentation.

2. You stated that 200-300 turtles are caught and eaten per year in the region of Nakudamu. Approximately how many of these turtles are hawksbills? Are the hawksbill turtles that are commonly eaten as large as the one that caused the recent poisoning?

Again, thank you for writing to me. I look forward to hearing from you again when your time permits.

Sincerely,

George H. Balazs
Zoologist

THE MINISTRY OF HEALTH
GOVERNMENT BUILDINGS
SUVA, FIJI

TEL. NO. 81444

REF. NO.

LABASA HOSPITAL
FIJI

DATE :

9th June, 1987

Mr George H. Balazs,
Southwest Fisheries Center,
Honolulu Laboratory,
2570 Dole Street,
Honolulu, Hawai 96822-2396.

Dear Mr Balazs,

Thank you for your letter dated 1st May, 1987 and also for the referal reprints. Sorry for the delay in replying. As you required I am giving you below the details of the turtle poisoning happened in Fiji in April 1987.

Three turtles were speared off shore by a villager on 11.4.87 at Nukudamu in Fiji. Two of them were Green Sea Turtles and one Hawkesbill type. One of the Green turtles was eaten by a family in another village with no incidence of poisoning. The other Green Turtle was found decomposed after cooking and was discarded.

All the poisoning happened in Nukudamu village was after eating the Hawksbill turtle. 43 peple ate the turtle meat; from children of 5 years to adults of 78 years. 24 of them had symptoms of poisoning. The shoulder portion of the turtle was eaten by a family of 11, of which 3 of them developed only mild symptoms. The visceral and the rest of the turtle was eaten by 32 people of which 4 died, 4 severely ill, 5 moderately ill and another 8 mildly ill; none under the age of 14 was ill.

The 4 fatal cases were of 19, 33, 70 and 78 years old. The 19 years old was a female and the other males. The deaths occurred on the days 3,5 and 10.

The intervals between the time of eating the meat and the beginning of symptoms were from four hours to 10 days.

The main symptoms were nausea, vomiting, abdominal pain, muscular and joint pains, weakness, drowsiness with tendency to sleep and sore throat and mouth.

The turtle was cooked by boiling with minimum water added.

An average of about 200 - 300 turtles are caught and eaten per year by the villagers of Nukudamu and around. Sea turtles were not found to have laid eggs in that area this year.

The turtle poisoning was never reported in Fiji before; but according to the elderly villagers of Nukudamu, 56 people died in a neighbouring village after turtle poisoning in 1905.

I may be able to send you more particulars later.

Yours sincerely

K E G Krishnan

(Dr K.E.G. Krishnan)
Consultant Physician, Labasa Hospital

becoming increasingly big business. Shell ornaments are popular, especially among people of Bengali origin.

Near the jetty at Katchal island where I utilized the stopover period of a inter-island ferryboat to take a quick plunge in the sea, I excitedly beheld the first colony of garden eels (troglodyte eels) that I had seen, on a sloping sandy bed in about 15 ft of water. As I approached them, they simultaneously retreated tail-first into their sandy burrows, swaying like stalks of vegetation in the gentle swell. Dr. Hans Hass has recorded the presence of troglodyte eels from deeper water off Great Nicobar island, but whether or not these were of the same species as the ones I saw, I am unable to confirm.

Close to the town of Wandoor in South Andaman, the intertidal fauna is particularly rich. Large chitons cling to spray-moistened rocks; sea cucumbers of at least five species are found in the shallows. A small pale white octopus crawled over rocks exposed by the tide.

South of the hamlet of Pulo Babi on Great Nicobar island, I twice observed avian predators — perhaps Nicobar Serpent Eagles — snatch up octopi from a reef exposed at low tide. In one instance the bird was forced to drop its prey after partaking of a bite or two, because of the mollusc's weight. Despite having a chunk missing from its mantle, I found the octopus to be alive and active after its fall.

I was fortunate enough to see civet cats (Paradoxurus tytleri) on two occasions: Once at day break on uninhabited Tarmugli island at a distance of ten feet as it leisurely climbed to the top of a tall tree, and another individual at dusk as it searched for titbits among crevices in the exposed reef on Rutland Island, much as I had observed wild pig do in Little Andaman. On both occasions the civets displayed a degree of apparent unconcern about the proximity of a human being that was startling to me.

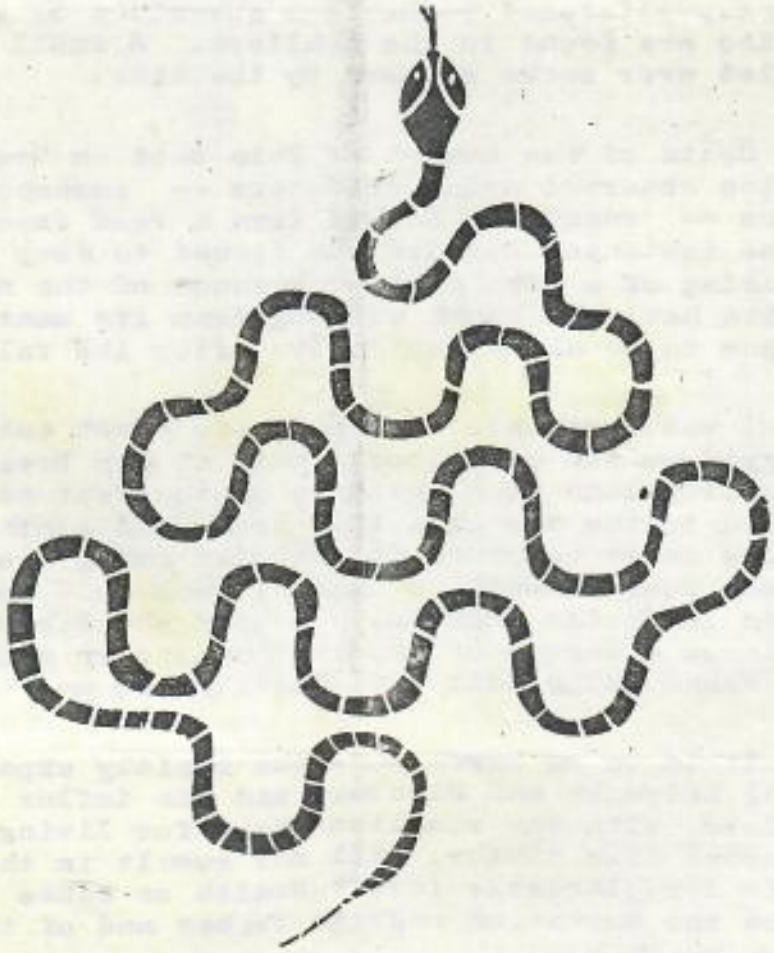
It is to be hoped that the rapidly expanding population in the Andamans and Nicobars and the influx of refugees and settlers, with the resultant need for living space and resources like timber, will not result in the undermining of its irreplaceable forest wealth or cause the disappearance of the the surviving negrito tribes and of their culture.
Satish Bhaskar.

TURTLE MEAT KILLS THREE

Tuticorin, June 17, 1980 (UNI)

Turtle meat took three lives — one directly and two indirectly — today. Two suckling infants aged six months and one year died after their mothers had taken turtle meat, and a seven-year old girl who took the meat also died today, official sources said.

HAMADRYAD



Seventy nine people (all fisherfolk) including several women of the fisherman colony of Trespuram who had taken turtle meat on Sunday (June 15, 1980) were treated at the Government headquarters hospital here. 57 were treated as out-patients and 22 were still in hospital.

From the 'Indian Express' Wednesday, June 18, 1980.

It appears likely that the turtle in question was a Hawksbill seaturtle (Eretmochelys imbricata), a species which has been indicated recurrently as causing deaths in India and Sri Lanka. The following instances have been recorded:

On 6th and 7th August 1977, nine persons — two adults and seven children of varying ages — died in the village of Manappad, southern Tamil Nadu from eating, on 3rd August, the meat of a seaturtle whose head was described as being somewhat aquiline, and as resembling a parrots beak. The seaturtle was also known locally as "Natchely Ammai" which means "turtle with a mouse-like head" and had a yellow plastron whereas that of the seaturtle species that was usually consumed (in all likelihood, the green turtle, (Chelonia mydas) was always white. On this occasion, some of the fishermen's advice against consumption of the meat, on the basis of it being of an occasionally poisonous variety, went unheeded. In 1970, deaths occurred at the village of Periathalai, 7 miles from Manappad, from the consumption of turtle meat.

In 1972, about 20 persons died in Thazai village from Hawksbill meat poisoning. (Valliappan and Pushparaj, 1973).

Deraniyagala (1953) cites instances of deaths in Sri Lanka in June 1921 at Manaaitivu (24 persons) and on December 3, 1941 at Habaraduva "Its toxicity is though to be due to the diet of the animal at the time; accordingly fishermen chop its liver and throw it to the crows before cooking its flesh. If the crows refuse it, the animal is discarded. Another test is to mix the raw flesh with slaked lime which turns greenish if the flesh is poisonous".

Valliappan and Pushparaj cite additional tests that some Tuticorin fishermen employ: the turtles blood drips off quickly if the meat is nonpoisonous and thickens on the knife blade if poisonous. A drop of blood on the skin itches and the spot becomes inflamed if the meat is poisonous.

Among symptoms of Hawksbill meat poisoning are:

Neurological symptoms like vertigo, twitching of the muscles leading to convulsions, coma and finally death. Ulceration throughout the buccal cavity, severe itching sensation in and sloughing of the upper layers of the tongue. A sensation of obstruction in the chest, respiratory failure followed by cardiac failure.

In the absence of knowledge of the exact type of poison involved, patients were given high doses of tetracycline, massive doses of vitamin C and corticosteroids and were put on plenty of fluids and diuretics. Where treatment was started before the collapsing stage, cases responded very satisfactorily to the administration of 'Siquil' as an antiemetic, "Anthisan" tablets for food allergy and "Terramycin" injection for the infection. In all cases where death occurred, one to four days elapsed from the time the meat was consumed.

The above data were kindly supplied by Berchmann Moraes and Dr. B.V. Balaji of Manappad, and by Drs. S.C. Thanupillai, G.C.I.M. and Dr. Ramasubramaniam of Udangudi.

S.B.

* * *

MUGGER (Crocodylus palustris) RELEASES IN ANDHRA PRADESH & TAMIL NADU

Andhra Pradesh

On 7th April 1980, the Andhra Pradesh Crocodile Conservation project released 33 mugger crocodiles (11 males and 22 females hatched in June 1977) into the Kinnerasani reservoir situated within the Kinnerasani Wildlife Sanctuary. This sanctuary is located 300 km north-east of Hyderabad. The released crocodiles all ranged from 1 to 1.3m in size. Follow up monitoring survey of the released crocodiles was carried out in August 1980. Some have shown a upstream movement of over 15 km during this monsoon time.

During previous surveys in this reservoir only a few (less than five) resident muggers were reported. No breeding has taken place in past years. Since, the released muggers are all of Gir (Gujarat) origin and are a very slow growing strain (1.2m in three years!) it was decided not to mix them up with the resident Andhra Pradesh wild breeding stock occuring in the Krishna and Godavari rivers and some other tributary rivers. The remaining 58 Gir muggers of 1977 origin are being released in Pakhal Wildlife sanctuary and again in Kinnerasani sanctuary. These releases are planned for the coming winter (November 1980 to February 1981).

Tamil Nadu

The second large scale mugger release by the Tamil Nadu Crocodile Conservation Project was carried out at Hoggenakal in May 1980. (The first release was

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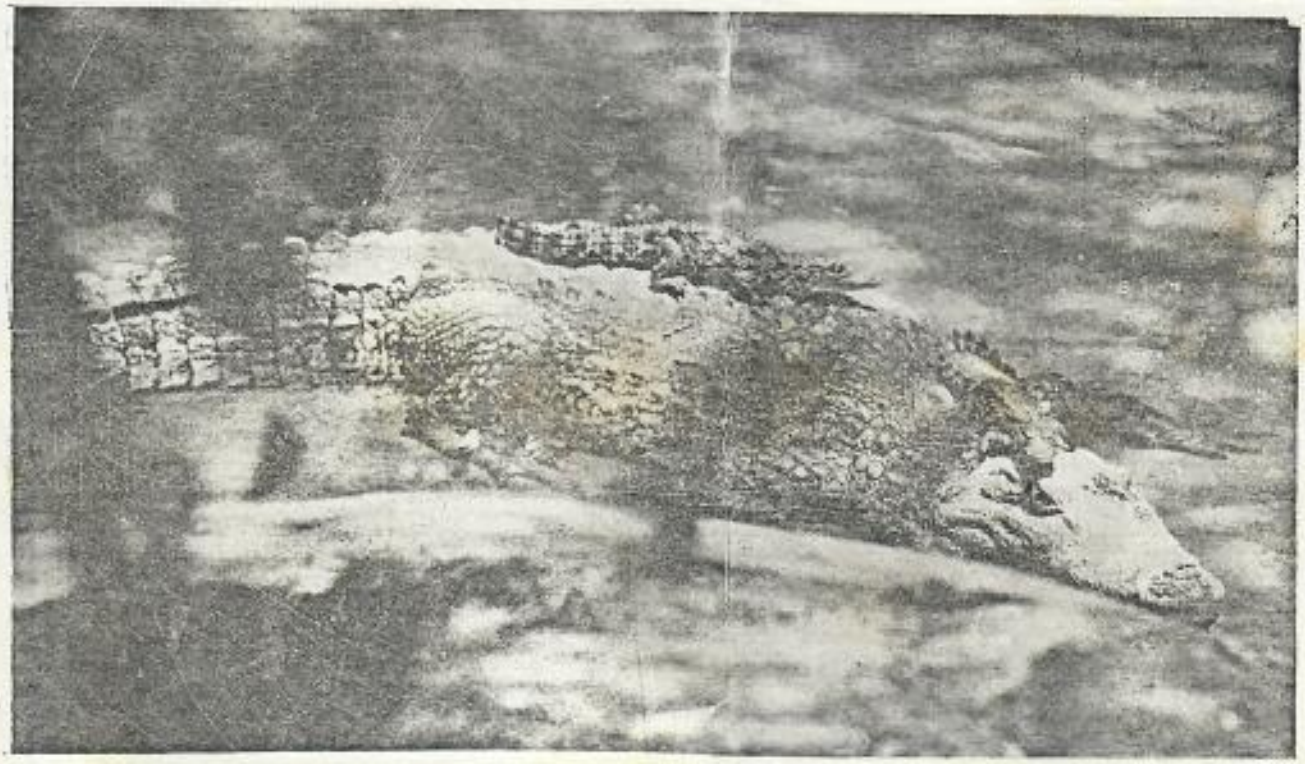
Cheques should be made to the Madras Snake Park Trust



Newsletter of the Madras Snake Park Trust, Guindy Deer Park, Madras-600 022. Edited by Zai Whitaker.

Department of
Environment and Heritage

QUEENSLAND
NATIONAL PARKS
AND WILDLIFE
SERVICE

160 Ann Street • Brisbane Queensland • PO Box 155 • North Quay Qld 4002
Telephone (07) 227 7111 • Facsimile (07) 227 7676

Enquiries to

Telephone

Your reference

Our reference

7 November 1990

Mr George Balazs
US Department of Commerce
National Oceanic and Atmospheric
Administration
National Marine Fisheries Service
Southwest Fisheries Centre
Honolulu Laboratory
2570 Dole Street
Honolulu
Hawaii, 96822-2396
UNITED STATES OF AMERICA

Dear George Mr Balazs

Re: Background information on Marine Turtle Poisoning

As a follow up to the recent deaths in Kiribati of persons who had eaten a toxic turtle, a summary of turtle poisoning is attached.

Both myself and George Balazs would be interested in receiving additional reports of cases of turtle poisoning (past or present) to add to our information base on this topic.

There is still much to be learned on this topic. Take care if hawksbill turtles are eaten. Cases of possible green turtle poisoning need to be checked for accurate species identification.

Thank you.

Yours sincerely

Col

Col Limpus
Principal Conservation
Officer (Research)

Please note that this letter to you was not at your request.

George. a recycled letter me over.

P.S. Was suprised to read Peter's letter. Sylvia had gone ahead and ordered 1000 tags for saying SPREP - Did she consult with you on coming up with an ideal return address?

11/13 — Seems like some collaboration or advice between the 3 of us should have taken place.

Col -

Nice write-up you did here for turtles in TOXIC Animals - Australia. No date appears 1987 on it, but presume it was very recent. Be alert, though, that I'll probably "rib" you alot about your ~~[illegible]~~ statement "In other countries, as a general rule eat only Green Turtle." Word! what a ~~[illegible]~~ recommendation! It is for people that eat turtles.

I've got quite a few more cases of poisning documented by literature sources and personal correspondence, including Tonga, Fiji and Samoa. Like lots of other things, one of these days I'll get it out.

October 25, 1982 F/SWC2:GHB

Dr. Turner, Director
LBJ Hospital
Pago Pago, American Samoa 96799

Dear Dr. Turner:

As I described during our telephone conversation on October 15, I would like to obtain information on the poisoning from turtle flesh that occurred some years ago on the island of Aunuu. Specifically, I would like to know 1) the month and year, 2) the number of people involved and mortalities, if any, 3) the symptoms of poisoning, 4) the kind of turtle eaten--probably a hawksbill (Eretmochelys imbricata) or green turtle (Chelonia mydas), 5) where and under what circumstances the turtle was captured, and 6) the approximate size of the turtle. I realize that it probably will not be possible for you to obtain all of these data but whatever you can send to me will be greatly appreciated.

Thank you again for your offer to help.

Sincerely,

George H. Balazs
Fishery Biologist

bc: Balazs
HL

GHB:iht

October 25, 1982 F/SWC2:GHB

Mr. Jack Erle
Aunuu Village
Pago Pago, American Samoa 96799

Dear Mr. Erle:

You will undoubtedly recall that we met during early October when I visited Aunuu to talk about sea turtle and the poisoning that occurred some yearsago. I greatly appreciate the help you provided at that time. Interesting enough, since returning to Honolulu I have uncovered some confusing literature references to the poisoning. I am therefore writing to you with the hope that you can obtain some answers for me that will clarify this important case. Perhaps you will be able to obtain answers to my questions from your Father-in-Law, Mr. Folitsu, or one of the other elders of your village. My questions are as follows:

1. In what year (and month, if known) did the poisoning take place?

2. How many people actually died?

3. What kind of turtle gave the poisoning? Green turtle (laumei) or hawksbill turtle (laumei uga)?

4. Where was the turtle caught? Nesting on the beach at Aunuu or swimming in the water somewhere away from the island?

I am sorry to trouble you with these questions but, as I mentioned, there is a lot of confusion here at the University of Hawaii over the facts. You are the best person I know living in the village that can find accurate answers to these questions. Thank you for your help.

Sincerely,

George H. Balazs
Fishery Biologist

Christchurch, NZ

Turtle meat fatal

Four Fijians in the second largest island of Vanua Levu have died from food poisoning after eating turtle meat. Another 11 people have been admitted to hospital. Reports from the aren said more people were showing signs of illness because they continued to eat turtle meat.

The Press 7 May 1987 p. 24

L.A. TIMES MON. JUNE 13, 1988.

Sea Cure

Scientists Enthusiastic About Potential Medical Compounds From Deep

By JAMES C. BORG

HONOLULU—The ancient Hawaiians called it "the deadly seaweed of Hana," and declared it "*kapu*," taboo.

Today the rare and extremely poisonous soft coral, known as *Palythoa toxica*, has become one of a growing number of sea creatures that may be a new source of modern medicine.

The research with palytoxin, while at an early stage, is indicative of renewed scientific enthusiasm in finding potential cures from the oceans.

"We see the sea as [containing] many interesting and exotic compounds, some of which have exciting possibilities as drugs," said Steve Brauer of the Hawaii Biotechnology Group, which is working with palytoxin as part of a $900,000 federal small-business innovative research grant. "The process of culling them out is a massive task and one which is just beginning."

One extract at an advanced stage of research is manoalide, named after the University of Hawaii at Manoa and derived from a sponge found at the Pacific island of Palau.

Discovered in 1977 by Paul Scheuer of the University of Hawaii, it was demonstrated by a group of University of California scientists to have potent pain-killing and inflammation-fighting properties. Allergan Pharmaceuticals of Irvine now has an option to license the product commercially and has begun testing it for toxic side effects.

R.L. BOWERS

A manned mini-sub collects underwater organisms for research, above. At right, the polyps of *Palythoa toxica*, a poisonsous coral found only in a small tidepool on the eastern coast of Maui.

The UC program is a collaboration between organic chemists John Faulkner and William Fenical at Scripps Institution of Oceanography in La Jolla, Phillip Crews of UC Santa Cruz and pharmacologist Robert Jacobs at UC Santa Barbara.

"My feeling is, manoalide or one of its relatives or descendants will eventually be available for treatment of certain forms of inflammatory disease," such as arthritis, said Jacobs.

Other promising compounds:

■ **Didemnin B**—Isolated from tiny Caribbean sea squirts called tunicates, Didemnin B is the first wholly natural marine product to enter clinical trials with cancer patients as a potential anti-tumor drug. Pioneered by Kenneth Rinehart at the University of Illinois at Urbana, Didemnins A and B also have shown promise as inhibitors of oral and genital herpes, flu and fever viruses.

■ **Pseudopterosins**—This class of anti-inflammatory compounds, developed by Fenical and colleagues at Scripps, comes from fernlike soft corals known as sea whips. Both manoalide and pseudopterosins offer unique metabolic approaches to controlling inflammation, said Fenical. Pseudopterosins chemicals also have proved effective as painkillers in animals tests.

■ **Dolastatin 10-G**—Robert Pettit and colleagues at Arizona State University painstakingly extracted this compound from an Indian Ocean sea hare, a mollusk not known to develop cancer. Supply problems have hampered research with dolastatins and another group of compounds called bryostatins, which were discovered by Pettit in a barnacle-like "false" coral that attaches to ships and piers. Bryostatins have shown promise against leukemia in mice and human ovarian cancer.

■ **Punaglandins**—These natural extracts from a soft coral found in Hawaii and Eniwetok in the Marshall Islands were found to be too toxic, but a similar synthetic version is currently under clinical trials as an anti-tumor drug in Japan.

Often compounds that prove too poisonous in their natural states for humans still can lead to new and promising chemical models.

"In many cases the natural product provides a good template, and then if undesirable side effects show, they can manipulate things pretty well and try to eliminate them," said Scheuer, emeritus professor of chemistry, who helped identify punaglandins in 1982.

One anti-cancer drug, Ara-C, was modeled chemically after a substance isolated from a Caribbean sponge in the 1960s.

□

Until World War II, land plants provided the raw material for nearly all pharmaceutical research. Some of the more effective drugs have properties well known to ancient peoples. For example, the key ingredients in aspirin and quinine came from tree barks while painkillers such as morphine and codeine came from opium poppies.

With the discovery of penicillin in 1928 from mold, lower forms of plants became a source of new medicines. Thanks to easy access of land plants and the process of fermentation, which allows large-scale production, research and development of land-based drugs moved rapidly.

But the terrestrial medicine cabinet now looks about as full as it's going to get, scientists believe. Hence the renewed interest in the seas—home to an estimated 80% of all life forms.

Some of the more interesting discoveries have come from the so-called "sessile" sea creatures that cling to rocks, hulls, piers or the seabed. These often have developed chemicals as a defense against predators.

Research into organic marine chemicals has been slow for a couple of reasons. First, only a small fraction of the ocean has been explored; second, even after being identified as "biologically active" in humans, many compounds cannot be easily produced in quantities necessary for testing, much less wholesale marketing. Interest in marine pharmaceuticals ebbed in the late 1970s, but has revived in the last five years or so, much of it building on university research funded by the Commerce Department's Sea Grant Program.

In 1986, the National Cancer Institute launched a large-scale program to collect new specimens of marine life for screening for anti-tumor agents. The institute awarded contracts worth $3.6 million to SeaPharm Inc., a private research company based in Princeton, N.J., for the collection of 10,000 deep- and shallow-water specimens.

SeaPharm works with the Harbor Branch Oceanographic Institution in Ft. Pierce, Fla., to collect organisms using the Johnson-Sea-Link manned mini-sub and other underwater technologies. Since it began operating five years ago, the company has screened some 11,000 compounds for signs of activity against tumors, viruses and fungi and for effects on the immune system.

The company has applied for more than 30 patents and hopes to begin clinical testing of an anti-cancer compound from a South Pacific sponge by mid-1989.

At the University of Hawaii, researchers led by chemist Richard Moore have been studying hundreds of varieties of blue-green algae for drug potential. In 1986, under a five-year grant from the National Cancer Institute, Moore and colleagues began to grow another 1,000 strains of blue-green algae for tests against the AIDS virus and 100 types of cancer.

□

Palythoa toxica, found only in a small tidepool on the eastern coast of Maui, has had a dark history in Hawaii.

In December, 1960, on the day that specimens were first collected, despite warnings by native Hawaiians that the tidepool was taboo, a mysterious fire gutted the university's marine biological laboratory.

Three years later, pure palytoxin was isolated, but its chemical description, ultimately by University of Hawaii chemist Moore in 1981, required technologies that did not become available until the mid-1970s.

Today, researchers at Hawaii Biotech hope to attach the palytoxin molecule to a lab-grown monoclonal antibody that will attack specific cancer cells while leaving healthy tissue alone. But developing such an "immunotoxin" has proven difficult.

No marine pharmaceuticals are expected to make it through the regulatory process and onto the market until at least the early 1990s. But that prospect has not dampened the enthusiasm of many in the field.

"I believe that there is a very large untapped resource of chemical entities from the ocean," said Thomas Matthews, a microbiologist at Syntex Corp. in Palo Alto. "I also believe that they are going to play a major role in the drugs of the future."

Borg is a Honolulu-based science writer.

NATIONAL MARINE FISHERIES SERVICE
HONOLULU LABORATORY
P. O. BOX 3830
HONOLULU, HAWAII 96812

August 9, 1983

F/SWC2

Mr. Semisi Fakahau
Fisheries Officer
Fisheries Division
Ministry of Agriculture,
Fisheries and Forests
P. O. Box 14
Nuku'alofa, Tonga

Dear Mr. Fakahou:

The Director of our Laboratory, Mr. Richard S. Shomura, recently told me about the case you reported at the SPC meeting concerning a poisoning in Tonga from eating a hawksbill turtle. As a researcher of sea turtles in the Pacific islands, I am very much interested in learning more about this case. Your kind assistance in helping our Laboratory obtain information will be greatly appreciated. As far as I know, this is the the only report of hawksbill poisoning in Tonga. Are you aware of any other cases that may have occurred in the past?

Your answers to the following questions will be helpful to our research:

1. Where did the poisoning occur in Tonga, and on what date?

2. How many people became sick, and how many died?

3. Was the hawksbill a large or a small turtle?

4. Where was it captured?

5. What is the name and mailing address of the medical doctor, or health officer, that attended the case?

I look forward to hearing from you about this important matter. Again, thank you for your assistance and cooperation.

Sincerely,

George H. Balazs
Wildlife Biologist

GHB:11

bc: HL
Balazs

DEPARTMENT OF HERPETOLOGY
AMERICAN MUSEUM OF NATURAL HISTORY
NEW YORK, NEW YORK 10024 • PHONE 212 873-1300/352

MESSAGE

TO George Balazs

DATE 10 May 1986

—George. Terribly embarrasing -- I lost (temporarily) the abstract & citation of the man who wanted to do nutritional studies of sponge inH. Remember the poultry science pub. on protein content of Chondrilla nucula? It's one of very few modern refs. on subject. Would you mind sending it to me again. ☹ I'm still not unpacked, believe it or not! Good news - I got a grant from the Lerner Fund (AMNH) and am off to Panama shortly for Nov. on chem. ecol. of sponge pred'n. in Hbills! Am anxious to get in the field.

BY Best regards, Anne

P.S. Above is for me — not just library business. Overdue, as usual! Did you get the bad loom I sent?

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Thank you -

Geoz

NORMAN W. BEBERMAN, M.D.
ANDERS G. J. RHODIN, M.D.

Orthopaedic Associates, P.C.

Telephone: 345-1144

Burbank Professional Building
Nichols Road
Fitchburg, Mass. 01420

April 12, 1984

Dear Dr. Balazs,

I saw your note in the latest Marine Turtle Newsletter about sea turtle poisoning in Tonga, and wanted to write you. Though I have never personally seen any cases, and do not know of any unpublished ones, I thought you might be interested in the short bibliography I have compiled over the years concerning this subject. My interest in sea turtle poisoning stems from not only my medical background, but also my interest in Papua New Guinea where I spent several months involved in both medical work and herpetological collecting. As you may be aware, I am also involved in turtle research. I have enclosed some of my recent sea turtle papers for your interest.

Perhaps there are some of the following articles that you were not previously aware of. Some of them are somewhat difficult to obtain. Should you need copies, then I have them here and can send you xeroxes.

Bierdrager, J. 1936. Een geval van massale schildpadvergiftiging in Nw. Guinee. Geneesk. Tijdschr. Nederland-Indie 76:1933-1944.

Deraniyagala, P.E.P. 1939. The Tetrapod Reptiles of Ceylon. Vol. I. Testudinates (p.191)

Dewdney, J.C.H. 1967. Turtle meat poisoning — the New Ireland epidemic, 1965. Papua New Guinea Med. J. 10:55-58.

Likeman, R. 1975. Turtle meat and cone shell poisoning. Papua New Guinea Med. J. 18:125-126.

Likeman, R. 1977. Turtles and botulism. Papua New Guinea Med. J. 20:93.

Romeyn, T. & G.T. Haneveld. 1956. Turtle meat (Eretmochelys imbricata) poisoning in Netherlands New Guinea. Doc. Med. Geogr. Trop. 8:380-382.

Siegenbeek van Heukelom, A. 1936. Doodelijke vergiftiging na het eten van een schildpad, gevangen bij Billiton. Geneesk. Tijdschr. Nederland-Indie 76:1945-1947.

Silas, E.G. & A.B. Fernando. 1984. Turtle poisoning. In: Silas, E.G. et al. (Eds.), Sea Turtle Research and Conservation. Bull. Cent. Mar. Fish. Res. Inst. 35:62-70.

I hope these references will be of some use to you. Best of luck in the completion of the project. I would be very interested in seeing your manuscript when it is completed.

536-9302

Sincerely,

Anders Rhodin

Anders Rhodin

Documenta Medicina Geographica et Tropica, continuing Doc. Neerlandica et Indonesica de Morbis Tropicis [illegible]

January 25, 1985 F/SWC2:GHB

Dr. Peter Barss
Medical Superintendent
Provincial Hospital
Alotau
Milne Bay Province
Papua New Guinea

Dear Dr. Barss:

Many thanks for your recent response to my inquiry about the incidence of poisoning from sea turtles eaten in your area. As yet Dr. Anders and I have not published any papers on this little-known subject. We are still in the process of gathering a sufficient data base. The distribution of 32 questionnaires to surrounding areas that you mentioned could produce relevant information for our study. Thank you for initiating this effort.

I have enclosed copies of two articles on turtle poisoning that you may not have seen. The first one deals with a 1954 case in the Philippines where 11 people and a dog died from eating a hawksbill turtle. The other article was just published last year and reviews a number of cases occurring in India. It would appear that the hawksbill is responsible for most poisonings, but is only displayed in certain animals. The reports of poisoning by the green turtle may often be misidentification. The scute count of the carapace is identical for both the hawksbill and green turtle. The carapace shown in one of the articles you sent me is difficult to identify from a photo for this reason. However, the mentioned fact that scutes had been taken from the carapace leads me to believe it was a hawksbill. Green turtle scutes are usually considered worthless and no one wants them. Often the carapace is all that remains from a village feast involving a turtle; everything else is consumed. Because of problems of species identification, I am also sending you an illustrated poster and other materials that may prove useful.

I look forward to hearing from you again if and when more information becomes available.

Sincerely,

George H. Balazs
Wildlife Biologist

GHB:ey
cc: Balazs
HL

NATIONAL MARINE FISHERIES SERVICE
HONOLULU LABORATORY
P. O. BOX 3830
HONOLULU, HAWAII 96812

August 9, 1983 F/SWC2

Mr. Semisi Fakahau
Fisheries Officer
Fisheries Division
Ministry of Agriculture,
Fisheries and Forests
P. O. Box 14
Nuku'alofa, Tonga

Dear Mr. Fakahou:

The Director of our Laboratory, Mr. Richard S. Shomura, recently told me about the case you reported at the SPC meeting concerning a poisoning in Tonga from eating a hawksbill turtle. As a researcher of sea turtles in the Pacific islands, I am very much interested in learning more about this case. Your kind assistance in helping our Laboratory obtain information will be greatly appreciated. As far as I know, this is the the only report of hawksbill poisoning in Tonga. Are you aware of any other cases that may have occurred in the past?

Your answers to the following questions will be helpful to our research:

1. Where did the poisoning occur in Tonga, and on what date?

2. How many people became sick, and how many died?

3. Was the hawksbill a large or a small turtle?

4. Where was it captured?

5. What is the name and mailing address of the medical doctor, or health officer, that attended the case?

I look forward to hearing from you about this important matter. Again, thank you for your assistance and cooperation.

Sincerely,

George H. Balazs
Wildlife Biologist

GHB:11

bc: HL
Balazs

Telegrams : Fisheries NUKU'ALOFA
All letters to be addressed
FISHERIES DIVISION, M.A.F.F.,
Telex: Gentel 66225, Fisheries
Telephone : 21-399

Ministry of Agriculture, Fisheries & Forests
P.O. Box 14,
Nuku'alofa,
Kingdom of Tonga.

....13th October, 1983.....

Our Reference ....F3/3/4/635.....

George H. Balazs
National Marine Fisheries Service
Honolulu Laboratory
P.O. Box 3830
Honolulu
HAWAII 96814

Dear Balazs

Please accept my apologies for the late reply of your letter dated 9 August, 1983 regarding your interest on the Hawksbill poisoning in Tonga in early this year.

Today (13 October) the Agriculture Extension Officer working in Nomuka contact me with the following information.

- The poisoning occurs in Nomuka Island (Position Lat.20°.17'S. Long 175°.48' W on the 12 March 1983.
- 18 people became very sick and two person died
- The approximate size of the Hawksbill Turtle is 2'4" long and 1'2" width.
- Caught at Hakaufisi Reef situated at the Western side of Nomuka Island
- The medical Officer that attended the case;

  Lata 'i Muli
  Medical Assistant
  Nomuka
  Ha'apai
  Tonga Island

Please note that the medical officer that attended the case is not a qualified Doctor.

Tevita F. Lātū
for Principal Fisheries Officer

TONGA GOVERNMENT MINISTRY OF AGRICULTURE F & F

TFL:af

MASS POISONING FROM TURTLE MEAT

by PILLAI ET AL.

Mass Poisoning from Turtle Meat. Pillai et al. (*J Ass Phys India* 10:181 [April] 1962) studied 130 patients who had food poisoning from consuming turtle meat. This happened in the coastal town of Quilon in May, 1961. The aquatic turtles belonged to the group of *Eretmochelys imbricata* and had grown up in the open tropical seas where they became poisonous from feeding on toxic flagellates during the summer months. Turtle meat is considered a delicacy on the south coast of Kerala from May to July.

Incubation period for poisoning in man was from immediately to 2 weeks. The toxic agent was a nitrogenous curare-like compound that produced muscular respiratory paralysis. Twelve families ate the poisonous meat. Those that had prepared a turtle curry after boiling and decanting the meat did not become sick. On the other hand, those that had prepared a curry without decanting became sick. Only 32 of the 130 patients had serious or moderate symptoms. They consisted in severe vomiting, pain in the throat, drowsiness, general weakness, and sunken and congested eyes. The throat was congested, the liver enlarged, and there was tachycardia with fall of blood pressure. Temperature varied between 99° F. and 101° F. One man developed a sudden behavior disorder; 6 pregnant women aborted. Pulmonary edema just before death occurred in 18 patients. All of them had been in a coma, and 2 children had convulsions.

Autopsy findings in 5 instances showed some lesions in the stomach and intestines; fatty changes in the liver; the heart was flabby; the cerebral cortex edematous; and the kidneys congested. The neurons showed degenerative changes.

Jnl of Assoc. of Physicians, Bombay.

From:
Journal of the American Medical Assoc.
Vol 181 No 9 Sept 1, '62
Page 140

Requested Pillai papers

11 April 1986

George -

You can't say I never send you anything exciting. This "Portuguese man-o-war" I found ~~found~~ washed up on a beach at Islip, Long Island, N.Y. It was on a beach inside Great South Bay, not on Atlantic. It was apparently one of the painted, metallic ones. It had lots of air in it when I found it — in fact I thought it was a man-o-war washed up. It still has air in it. Very persistent, I'd say. 1st hand evidence of balloon problem, no? Hope you like it.

(OVER)

Of the Nukualofa only I Answered the Questions.

1. No Hawksbill or Turtle poisoning before at Nomuka or in Tonga. *letter to you from Tevita Latu says 2 died. Did you get to the bottom of this?
2. ① Twenty-One people were ate the hawksbill, eighteen were very sick and three died, and three of them were not sick, and Mostly of the very Sick and those people died who ate the Viscera.
3. The three people were died Name and Sex/Age
   ① ELENOA ONGOONGO — F/8.
   ② SIONE MISINALE ONGOONGO - M/1yrs 7months.
   ③ MATEAKI FONUA IVAHA LATAPU M/9months 29days.
4. Symptoms:- Headache, Vomiting, Weakness, Lost of Power, Pain in the joints and all the body.
5. Said after an hour of eating, Short Headache and Vomiting and after 24 hours and death occur.
6. Boiling
7. More likely Hawksbill and Turtle. = green turtle? (the real turtle, they say in Caribbean)
8. No Hawksbill or Turtle lay eggs at Nomuka.

Most of the people who got very sick and those who died were ones who had eaten the viscera?

slight?

I hope that this short report will meet your inquiries. Thanks to

FOOD POISONING DUE TO TURTLE FLESH

(A Study of 130 cases)

V. Kumara Pillai,* M. Balaraman Nair,** K. Ravindranathan,† C. S. Pitchamoni,‡

INTRODUCTION

Food poisoning due to consumption of shell fish is not uncommon. According to Deklary[3] many persons have definite idiosyncracy to shell fiesh, generally manifested as urticaria and alimentary symptoms. Apart from outbreaks of Typhoid fever due to consumption of infected shellfish, collected from sewage-polluted areas, cases of food-poisoning have been reported due to consumption of mussels, oysters, crabs, lobsters and turtles. The turtles belonging to the *Eretmochelys imbricata* ('*Alungama*' in Malayalam) species are commonly caught from the west-coast and the flesh is consumed by many. Buckly and Parges[1] have reported that this species is met with in Arabia, Malaya-Peninsula, Malaya Archipelago, Australia, Formosa, Samoa, Guiana, Bahamas and Guatemala. The species *Dermochelys coriacea* is seen in the Cape of Good Hope, Indian Ocean, New Zealand and Solomon Islands. Gala[2] reports that the tetrapod reptile fish is usually eaten by the poor class of Tamil fishermen in the north of Ceylon. It is said that the flesh becomes poisonous after the turtles have eaten certain algae and deaths have been reported by consumption of these turtles occasionally.

Halstead has reported such poisoning and the symptoms are diarrhoea, nausea and vomiting, sore throat and sore lips, feeling of constriction in the throat, marked debility, fever, boils, hallucinations, irresistable somnolence, coma and death due to respiratory paralysis. The onset of symptoms is almost immediate or may be delayed upto a week or two. Death may occur within 12 hours after ingestion or may be delayed upto two weeks in some cases; nothing is reported regarding the nature of the toxin and its antidote.

According to Dreisbach[4] "Mussels, clams and oysters growing on the open ocean become poisonous during the warm months (May to September) from feeding on certain dinoflagellates including *Gonyaulax Cartenella*. One mussel clam or oyster may contain a fatal dose of the poison. More than ten deaths have been reported in the U.S. literature from this type of poisoning. The poisonous principle contained in the shellfish is a nitrogenous compound which produces a curare-like muscular paralysis. The principal manifestation of shellfish poisoning is respiratory paralysis. After ingestion of poisonous shellfish, the following are observed to occur: numbness and tingling of lips, tongue, face and extremities and nausea and vomiting progressing to respiratory paralysis. Convulsions may or may not occur. Laboratory findings are not characteristic".

* Professor of Medicine, Medical College Hospital, Trivandrum.
** Lecturer in Pathology, Medical College, Trivandrum.
† Tutor in Medicine, Medical College Hospital, Trivandrum.
‡ House Surgeon, Medical College Hospital, Trivandrum.

1962. J. Assoc. Phys. India. 10(4):181-187

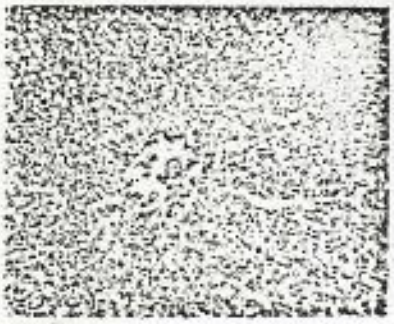
Locket[6] reports that during certain seasons of the year (June to October) mussels and clams contain a powerful heat stable neurotoxic agent. Death from respiratory failure occurs in a high proportion of persons poisoned (Sommer and Meyer 1941). The mortality rate would appear to lie between 5%-10% of cases poisoned. Apparently the toxic agent is due to the presence of a dino-flagellate *Gonyaulax-Cartenella*. Poisoning due to Pacific coast mussels had been known to the American Indians. These Indians had noticed that when shellfish were eaten after being collected when the ocean waves were luminous in the hot weather, they caused illness and death. It is due to a unicellular, microscopic organism. This plankton organism is most abundant in the summer and at times they multiply producing the so-called 'red water' during the day and luminous at night. A little sea water may contain 40 million of them and a far less number is highly poisonous. Patients may die of paralysis, gastrointestinal irritation or allergy. There is vomiting, swelling of the face and feet, pricking sensation of hands and feet, convulsions and spasms, dilated pupils and respiratory failure. The poison is of the nature of an alkaloid which is heat stable and the lethal dose for man is 10-20 mg. of the pure poison.

According to Oettingen[7], in mussel poisoning, the toxic effects are due to allergic idiosyncracy or infection by salmonella or another toxin which shows seasonal variation. This poison resembled the "fugu" poison of Japan isolated from livers and gonads of Tetradon. This is probably a quarternary or tertiary amine. Its concentration increases during the spawning season and produces numbness of the mouth, numbness and pricking of fingers and toes, ataxia and impaired speech. The patients feel exhilarated and are not often aware of the seriousness of their condition. Occasionally there may be nausea, vomiting and diarrhoea, respiratory disturbances, paresis of musculature and death from respiratory paralysis in three to ten hours. In the process of cooking, poison is accumulated in the broth and is destroyed by soda bicarb.

Thienes and Haley[6] report that "four or five mussels cause symptoms, twelve severe, more than this fatal. A purified extract is lethal to mice in a dose of .06 microgram per gram of body weight. It occurs in *Gonyaulax Cartenella* which are eaten by mussels in spring and summer rendering them poisonous. The poison may be a quarternary ammonium base. Absorption from the intestine is rapid. It is found in red sea water. Cooking does not destroy this poison. Symptoms are parasthesia of oral mucous membrane and limbs, tingling or numbness or anaesthesia. Muscle, tendon or joint sense is disturbed, there is, unsteadiness, mild paralysis and giddiness and difficulty in speaking. Severe poisoning results in respiratory failure. Heart and circulation are little affected. Death occurs within a few minutes to 12 hours. Prognosis is good after 12 hours. The toxin is rapidly excreted by the kidneys." Pathological findings were petechial haemorrhages of the upper gastrointestinal tract, damage to the ganglion cells of the medulla and anterior horn cells of the spinal cord.

g. 1. Section through oesophagus showing hydropic degeneration of the epithelium and ulceration.

Fig. 2. Section through wall of the small intestine showing marked submucous oedema and separation of muscle bundles by oedema fluid.



g. 3. Section through liver showing fatty changes and commencing centrilobular necrosis.

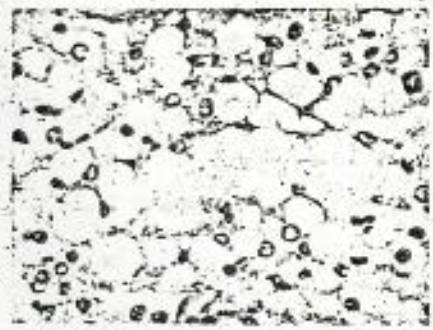


Fig. 4. High power view of section through liver showing extensive fatty changes with formation of fat cysts.

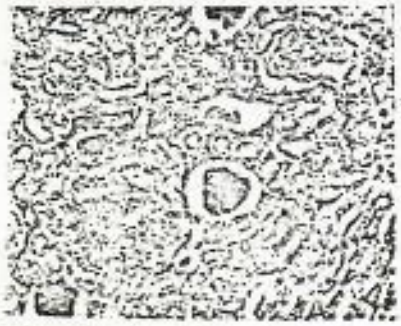


Fig. 5. Section through kidney showing cloudy degeneration of tubules.

Fig. 6. Section through floor of the fourth ventricle showing subependymal ring haemorrhages.

1962. J. Assoc. Phys. India. 10(4):181-187

MATERIAL AND METHODS

A clinico-pathological study of 130 cases of food-poisoning due to consumption of the flesh of the turtle belonging to the group of *Eretmochelys imbricata* was done, which occurred in May, 1961 in the coastal town of Quilon, Kerala State. Of the 130 cases only 32 had severe or moderate symptoms and others had mild symptoms, but all were admitted to Quilon District Headquarters Hospital, for observation and treatment.

History of the cases:

On the morning of Saturday (27-5-1961), 5 turtles were caught alive by fishermen. The flesh of one of them which alone proved poisonous was shared by 12 families and was consumed the same afternoon. The preparation made was a curry which in some houses was prepared after boiling and decanting. No incidence of poisoning occurred in these families and in those who ate the other four turtles. Among those poisoned all had taken curries prepared without decanting after boiling but were symptom free on the first day except one child, who vomited several times the same evening. Another child from the same family started vomiting on Sunday morning i.e., on the next day and later developed fits and died on the way to hospital. It was on Sunday that most of the persons who consumed the flesh of the poisonous turtle without decanting after boiling, started vomiting.

Symptoms:

The presenting symptoms in all these cases were vomiting and severe constipation. Vomitus was bilious in nature and contained plenty of mucus, but no blood. All of them complained of pain in the throat and general weakness. Even those persons who were admitted with mild symptoms had pain in the throat. Six of the patients who were pregnant women aborted before death. One case, an adult male, who was convalescing, suddenly developed behaviour disorder, but improved later with treatment.

Physical findings:

Thirty-two of the patients were drowsy with sunken and congested eyes and had moderate fever, the temperature varying between 99-101 degree F. No allergic manifestations were noted in any of the cases.

Alimentary system: Tongue was dry and coated and longitudinally fissured. Two or three days later all showed severe glossitis. This finding was also seen in the 98 cases admitted with mild symptoms. Throat was congested. Abdominal examination did not reveal anything abnormal in the beginning except in one boy who had a palpable, firm liver which was not tender. In the 2nd week, in 32 patients the liver was found to be palpable upto three-fourth of an inch below the costal margin, but it was not tender.

Cardiovascular System: Slight tachycardia was noted in all the 32 cases with fall of B.P. (systolic pressure varying between 84-94 mm. of Hg.). Nothing else abnormal was noted. E.C.G. was normal.

Respiratory System: Did not reveal anything abnormal except in 18 of e comatose patients who had pulmonary oedema just before death.

Central Nervous System: All were drowsy, 18 of them were comatose. wo cases (both children) had convulsions before death. Pupils were not re-ting to light in 32 cases with severe symptoms. Deep reflexes were diminish-l. Plantar reflex was flexor. Fundus occuli was normal in all the cases.

Genito Urinary System: Nothing abnormal was detected.

ourse and Complications:

Children developed convulsions before death and adults died of coma. he total mortality in this series amounted to 18 and all died due to respira-ory paralysis, the pulse and the heart sounds were good till the end and the CG taken 30 minutes before death in one case was normal. Others who ere serious in the beginning improved with the symptomatic treatment given, ut as mentioned earlier, liver became palpable in 32 of them.

nvestigations:

1. Urine: Normal in all cases.

2. Blood: (a) Total leucocyte count 6,000-7,500/cmm., (b) differential ount was normal, (c) E.S.R. 10-15 mm/1st hr., (d) Westengren Blood Urea —was raised in five cases but not significantly.

3. Liver function tests—normal.

4. C.S.F. The tension was raised in five cases, but biochemical analysis howed nothing abnormal.

5. E.C.G.—normal.

6. Report from Public Health Laboratory:

(A) Bacteriological and serological examination from blood and motion for common food-poisoning salmonella and other organisms proved negative.

(B) Chemical analyser's report:

The usual inorganic or organic poisons were not found but the extract of the stomach contents and liver on injecting into mouse and frog killed them. Control animal showed no symptoms.

7. Autopsy findings in 5 cases (1 woman, 1 child, and 3 men): Serious cavities contained slight excess of fluid. (Fig. 1) Oesophagus showed ulcera-tion of mucosa. Gastric mucosa was congested and oedematous. Small intes-tines contained thick bile. (Fig. No. 2) The intestinal mucosa and sub-mucosa showed marked oedema. The muscle bundles were separated out by oedema fluid. Large intestine contained thick mucus and bile and the mucosa showed oedema and ulceration. Liver: soft, friable, showed patchy conges-tion. Microscopy (Fig. No. 3 & 4) showed practically all the liver cells to have fatty changes with formation of fat cysts. There was centrilobular con-gestion and necrosis of the adjacent liver cells. Heart was flabby and showed

subepicardial petechial haemorrhages. Kidneys were intensely congested and tubules showed cloudy swelling (Fig. No. 5). The cerebral cortex was oedematous. The cortical vessels were intensely congested. The floor of the fourth ventricle (Fig. No. 6) showed congested vessels and petechial haemorrhages. The neurones showed degenerative changes.

DISCUSSION

Incidence of turtle flesh poisoning has seldom been reported from any part of India. A few cases were reported from Tuticorin. But so far no clinico-pathological study, as done in the present series, has ever been reported nor has there been an incidence of such poisoning in as large a number of cases reported from anywhere in the world. Turtle flesh has been a delicacy among a large section of people living along the coast of south Kerala as in other parts of the world. During certain seasons of the year (namely the Monsoon months of May, June and July) large numbers are trapped and eaten curried. Their size varies from 2—4 feet in length and each affords a day's food for ten to fifteen families. It is reported that large numbers are exported from Tuticorin in Madras State to Ceylon. It is strange, therefore, that incidence of poisoning has not occurred in Ceylon. The explanation offered is that the turtle becomes poisonous only after eating poisonous algae. Incidences of poisoning due to such algae—fed turtles, though common in the Pacific Isands has occurred only once in this part; probably such algae-fed turtles are not found in these areas. It is possible that on account of the heavy monsoon winds which prevailed in May last, poisonous turtles have been carried by heavy seas and got trapped accidentally. Three or four other turtles trapped along with the poisonous one did not prove harmful. Those exported to Ceylon from the Madras State are stored and fed for more than 7 days with a view to get rid of the poison in case any of them had fed on algae and this has been successful in preventing poisoning in those parts.

In the present series of cases, those who boiled and decanted the flesh did not develop poisoning or escaped with very mild symptoms. This and the fact that the flesh was eaten fresh, exclude poisoning due to the products of the flesh itself; also, some who had eaten the head part of the turtle escaped with mild or no symptoms showing that the belly part of the turtle contained something which the turtle had consumed and was the poisonous element.

The fact that the flesh was curried and consumed shows that the poison is heat stable and Locket[5] has stated that it is of the nature of an alkaloid. The poison is both an irritant and a neurotoxin as evidenced by the ulcerations from the mouth downwards, the damage to the liver, congestion of the medulla of the brain, which produced coma and respiratory failure. The fact that the jerks were feeble with flaccid muscles with no evidence of spinal cord involvement shows that the poison had a curare-like action. The enlarged liver during convalescence and the cellular necrosis seen during autopsy suggest the likelihood of a post-necrotic cirrhosis developing at least in some cases later in life.

In view of the above widespread damage the poison has produced as reported also by Oettingen[7], it is a strange finding that many of those who had seen the poison refused hospitalisation until they developed serious symptoms were persuaded by others.

It is said by Gala[2] that in countries where *Eretmochelys* poisoning was common, experienced fishermen fed crows with the raw liver of turtles to see whether they were poisonous before eating them. Poisoned patients were given soup from the carapace and plastron of chelonia mydas.

Since soda bicarb rendered the poison innocuous, stomach wash with soda bicarb would have been efficacious in the present series, but was not possible since the patients did not report in time until dangerous symptoms developed.

SUMMARY AND CONCLUSIONS

(1) Incidence of 130 cases of poisoning due to eating of turtle flesh is reported.

(2) A clinicopathological study is made.

(3) Course and nature of the poison are discussed.

(4) Preventive and curative measures suggested.

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PLEASE RETURN TO BALAZS

FILE - INVERTS

American Museum of Natural History

13 November 1985

Dear George:

Thanks for your letters, articles, and the nice hawksbill slide. I've already had a chance to use the latter. I am finally getting settled at the AMNH, and we've now just moved into an apt. in the city. This means 4 hours/day not spent commuting! It's quite a change to be here -- rather exciting, actually, although I miss people + places (like our home) in Gainesville. My position at the museum is a courtesy appt. (= no $), so I am looking for a job -- at least ½ time. I really need time to write up my diss'n. and continue my research.

Sorry for not sending you info. on Chondrosia chucalla. Actually, I don't know anything about that particular species, although I'm sure it's very similar to ones in the Caribbean (i.e., C. collectrix) that I know very well. I didn't assign a specific name to any of the Chondrosia material I found in caribbean hawksbills, but it was very common in my samples. I was aware of your finding sponges in Hawaiian greens, and mentioned it in my dissertn, but I didn't get into specifics because they were green turtles. Chondrosia is unusual in (1) lacking spicules + (2) spongin fibers, but I

Central Park West at 79th Street New York, New York 10024, U.S.A. Telephone (212) 873-1300

think you can discount the first component as a feeding deterrent, in light of the hawksbill's diet with many species having >50% silica. You might be interested to know that it's the only (genus) species I've ever heard was consumed by humans — there is a mention of Dalmatian people eating it in one of de Laubenfels' old papers. The lack of spongin fibers is in keeping with patterns observed for hawksbills. I am still at a loss to explain this. I have reviewed species id's for all sponges consumed by turtles, and except for Den Hartog's S. Atlantic record, all fit the pattern. I am working on a manuscript describing patterns and will send you a draft for your comments. Incidentally, I was able to get samples from h'bill's in Oman (thru Pervan) and S. Africa (through G. Hughes). Also got a batch from Mona Island, ALL sponges. And, apparently, keeping to patterns I've documented for the Caribbean. I'm really excited about the whole subject — in fact, I'm attending an Int'l. sponge symp. next week at Woods Hole, and chairing a session on ecology of coral reef sponges! What an odd assignment for a turtle person!

I enclose a recent publication on collagen aspects of h'bill diets. This discussion is highly relevant to Chondrosia — I mention this genus in the text. It is one frequently used in research on collagen fibrils, because of its high content. The collagen imparts a very tough, rubbery consistency.

I never really talked after my trip to Tahiti. I met Beau out there, and heard about his recent surveys out to Scilly. He's left Tahiti by now, and apparently nothing much is happening regarding Fr. Polynesian turtles. Turtles seem to be very low priority, (esp. w/the Greenpeace affair!) and the idea to somehow exploit Scilly has ~~somehow~~ not materialized. He did say the govt was relocating the human settlement to the best nesting area -- to escape mosquitos at the current site. He was worried what effect this would have on poaching, which occurs at the present, anyway. I tried to get a copy of his Tahiti paper, but he promised to send it along and never did. He was sort of an odd guy -- he chaired one session in his barefeet, with shorts (gym) and a flowered shirt. Very laid back, I'd say!

Are you planning to attend the Costa Rica turtle meeting in December? Wish they had announced it a bit sooner. I'd like to go, but I don't really have funds. Did you attend the Galveston ridley mtg? I didn't.

WATS II is rolling along. I don't know what Fred + cronies are up to, really. I had an invtn. for a meeting in Miami last week, but I couldn't go. I hope they go about it in a sensible fashion - I really hate to see conservn. money all tied up in one big wad. I'm not sure what Wats II will provide in addition to WATS I, ~~which~~ The latter was a success, but seemed like a once-in-5yr. exercise in P.R.

nt mean to write so much, but once I get fired up....

Do you know Jim Parrish? He was at the mtg. in Tahiti, and I later wrote him for details of sponge-feeding fish. He was extremely helpful. Please give him my regards if you have occasion to talk to him.

I am applying for jobs around the U.S. for next fall. Do you know of anything in Hawaii? I applied for one at Port Aransas, as a marine scientist at the Marine Science Institute of U of T at Austin. Am keeping my fingers crossed. Please let me know if there's anything worth pursuing.

Harvey Hirth called yesterday to tell me about the tons of plastic beads on Tortuguero beach. I told him about the styrofoam precursor business. He may be getting in touch w/ you about this to see what you've learned.

Well, all for now. I'll try to keep in touch a little better.

Regards,

Anne

My phone at AMNH is 212-873-1300 ext. 352 secretary or 415 herp laboratory
I don't have an extension at my desk, so have various alternatives!

Peter's ext. 287

home 212-628-4130

CSIRO
MARINE LABORATORIES

Division of Fisheries Research
Division of Oceanography

Castray Esplanade, Hobart, Tas. 7000

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A Division of the Institute of Physical Sciences

GPO Box 1538, Hobart, Tas. 7001
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REJ: KE

17th February, 1986

Mr. George Balazs,
U.S. Department of Commerce,
National Oceanic and Atmospheric Administration,
National Marine Fisheries Service,
Southwest Fisheries Center,
Honolulu Laboratory,
P O Box 3830,
HONOLULU HAWAII 96812

Dear George,

In response to your request for comments on the doings of the Hawaiian Sea Turtle Recovery Team:

I'm not convinced I can serve any real purpose here. My current involvements don't permit any useful work on turtles. I haven't gotten anything of note from my Torres Strait work - which has concentrated on dugong and traditional sea rights because these are the two major problem areas in traditional fisheries there. Colin Limpus knows far more about turtles in the Strait than I do. I'm also being pulled into research in temperate waters and getting spread entirely too thin.

So I think we should leave it at my responding to requests for specific information or advice only in the event that you feel I might be able to be useful.

call ANNE

One subject that intrigues me and that I will write a bit about in my final report is toxicity of hawksbills. Only a few people in Torres Strait will eat them because of past deaths. Those who do are particularly careful to remove a white gland (?) from on the trachea for it is believed to contain the poison. Do you know anything about this - and what the gland might be?

Sorry I can't be of more help to you.

Yours sincerely,

R. E. Johannes

P.S. Thanks for bugging Nitta about the publication of my report.

File - Hawksbill Poisoning

CSIRO
MARINE LABORATORIES

Division of Fisheries Research
Division of Oceanography

Leach Street, Marmion, W.A.

A Division of the Institute of Animal and Food Sciences
A Division of the Institute of Physical Sciences

PO Box 20, North Beach, W.A. 6020
Telephone (09) 447 1388 Telex 93366

2nd August, 1984.

Mr. George Balazs,
National Marine Fisheries Service,
Southwest Fisheries Center,
Honolulu Laboratory,
P.O. Box 3830,
Honolulu, Hawaii 96812,
U.S.A.

Dear George,

Thanks for the various pieces of information you've sent.

I recall reading somewhere that you are looking for information on poisonous hawksbills. You can use the following as "Johannes, pers. comm." if you wish.

In Torres Strait hawksbills are occasionally poisonous - and no other species (they eat lots of greens). A number of people have died from eating hawksbills. Today many people are afraid to eat them, but those who "know" how to cut them up still do. "Proper" cutting up involves tilting the inverted turtle to one side so that the guts slide to the opposite side from where the incision is made, so as to avoid cutting the guts. Then the turtle is tipped in the opposite direction when the cut is being made on the other side. The most dangerous part is generally considered to be a whitish gland on the trachea. My field officer watched a turtle being butchered this way, ate some of it (said it tasted terrific - better than green turtle) collected the "organ" and sent it to Dr. Clive Wilkinson at AIMS who may or may not get around to sending it off for analysis to somewhere in the U.S. He has a theory that the poison may be derived from toxic sponges. There was some talk among the fishermen that maybe the gall bladder was bad news too - but most of the guys who seemed most knowledgeable seemed to feel that this white gland was the bad one.

Just returned from an expedition to Northern Arnhemland where I was working with Aboriginal fishermen who routinely eat greens, flatbacks, loggerheads, ridleys, and, to a lesser degree, hawksbills. They also say that hawksbills, and only hawksbills, occasionally kill people or make them sick. They claim that a good fisherman can tell whether a hawksbill is poisonous or not by the color of the plastron - if it has a dark yellow-red tinge it's poisonous. (These guys are not very inspiring informants, so I'd take that with a grain of salt.) They also say that the eggs are sometimes poisonous - again, only in hawksbills. If the yolk is dark yellow to reddish they are poisonous. If lighter yellow they are OK. The poisoning is sufficiently rare (and Aborigines so incredibly omnivorous) that many people will take their chances and eat hawksbill eggs or flesh - or at least that's what I'm told.

# #

I was also told that greens lay at this time of year and both day and night. I was a bit sceptical on both counts, on the former count

on about July 20

because greens lay only during the months around Xmas to the east in Queensland and to the west at North West Cape. However, an hour after I was told this I found a green digging a nest at 2.45 p.m. on a bright sunny day. It was killed and eaten. It had a full complement of eggs ready to lay and another set of "yolks" that were virtually as large as the shelled eggs.

Cheers,

R.E. Johannes

P.S. would very much like to see what you come up with on this subject.

from Edmonds, Carl, 1976
Dangerous Marine Animals of the Indo-Pacific Region
Diving Medical Centre Monograph on Identification First Aid and Medical Treatment.
Wedneil Publ. Newport Australia

TURTLE AND TORTOISE POISONING

ORDER : Chelonia

SPECIES & SYNONYM :
Caretta caretta — Loggerhead turtle
Eretmochelys imbricata — Hawksbill turtle
Chelonia mydas — Green turtle
Dermochelys Coriacea — Leatherback turtle
Pelochelys bibroni — Soft shell turtle

GENERAL

In Australia the marine species of Chelonia are termed turtles and the freshwater or land species are called Tortoises. Both have been used as food, and some of those in the north Australian waters weigh over 200 kilograms. One would have thought that the unpleasant consequences of ingestion would have made them obsolete as foodstuff, but still this animal is consumed by some ships crews, natives, etc., and is particularly reported around the Malay Archipelago and New Guinea.

There is no way of determining whether the turtle is poisonous except by trial and error. Some authors believe the poison to be similar or identical to ciguatera. Autopsy findings include hepatocellular damage, to the extent of acute yellow atrophy of the liver, necrosis of the kidney, haemorrhages and ulceration of the bowel.

CLINICAL FEATURES

- Symptoms commence a few hours to several days after ingestion of the turtle.
- Anorexia, nausea, vomiting, abdominal pain and diarrhoea in many cases.
- Abnormal sensations around the lips, mouth, tongue, throat, etc., may extend to include dryness or increased salivation and difficulty in swallowing, mouth ulcers and inflammation may supervene and become extensive — lasting for weeks or months before healing is completed.
- Weakness, sweating, pallor, vertigo, headache.
- A generalised red itchy rash may later peel.
- Difficulty in breathing, tightness in chest, may extend to severe respiratory distress, central cyanosis (bluish tingue to lips) and death.
- Liver damage may result in jaundice, liver enlargement and tenderness, coma and death.
- Other manifestations may mimic ciguatera poisoning (page 166).

- Mortality rate is over 25%.
- Renal failure may result in a decreased urinary output and then the development of uraemia over the next few days.

FIRST AID

1. If the patient is fully conscious, induce him to vomit by inserting fingers in his throat. Syrup of Ipecac U.S.P. 8 ml orally may aid in this. Treat any other potential victims (e.g. other people who ate the turtle) in the same way.
2. Rest and reassurance.
3. Resuscitation if needed. This will usually be in the form of mouth to mouth respiration (page 197) in those patients who develop severe respiratory distress, unconsciousness with cyanosis (bluish colour), etc.
4. Hospitalisation and observation is always needed until recovery.

MEDICAL

1. First Aid as above. Gastric lavage (apomorphine 2—8 mg s.c.) or other emetics if the laryngeal reflex is unimpaired.
2. With respiratory involvement, the ideal treatment is to perform endotracheal intubation and control respirations. The use of an endotracheal tube will also prevent the aspiration of vomitus, particularly likely under the conditions of a bulbar paralysis with gastrointestinal symptoms. If this is not achieved, then maintain respiration by any method at your disposal..

   Assisted intermittent positive pressure respiration (e.g. with a Bird respirator) may be of value when there is only a mild impairment. If there is a rising arterial CO_2 level or an increasing respiratory rate, assistance with respiration is required but oxygen supplementation is not needed.

   When there is a more severe degree of respiratory depression with symptomatic distress and/or cyanosis, an increasing arterial CO_2 and a decreasing arterial O_2, it would be prudent to completely control respirations by the use of endotracheal intubation and mechanical ventilation. Monitoring of serial arterial O_2, CO_2 and pH levels is required. The patient should be maintained on the regime for at least 6 hours and then gradually weaned from the respirator over the next 12—24 hours.
3. Ensure fluid and electrolyte replacement and administer medication by intravenous means (record vital signs, serial haematocrit, S.G., electrolytes, C.V.P., E.K.G., urinary output and analysis, etc.).
4. Sedation should be achieved with non-respiratory depressants, e.g. diazepam 10 mg i.v. repeated as required. Small doses of opiates may be needed for pain.
5. Steroid cover, e.g. hydrocortisone 100 mg i.v. 6 hourly during the danger period is possibly of value — but is non-specific in its effect.
6. Treat the bowel disorder symptomatically.
7. Monitor the clinical, biochemical and electroencephalographic manifestations of hepatocellular damage, and correct these by the customary medical dietetic and antibiotic techniques.
8. Monitor the clinical and biochemical manifestations of renal failure and correct these by dialysis as required.

PREVENTION

Refrain from eating turtles.

October 26, 1983 F/SWC2:GHB

Mr. Lata 'i Muli
Medical Assistant
Nomuka
Ha'apai
Tonga

Dear Mr. Muli:

Mr. Tevita Lātū recently sent me information on the hawksbill poisoning that occurred in March of this year at Nomuka Island. Your name was listed as the medical officer that attended the case. I am therefore writing to you in an effort to obtain some additional facts. It would be greatly appreciated if you would answer the following questions to the best of your knowledge.

1. Has hawksbill poisoning ever occurred before at Nomuka Island, or anywhere else in Tonga?

2. How many people total ate the hawksbill? I understand that 18 people became very sick and 2 died. Were there people that ate portions of the turtle but did not become sick? Could the sickness or deaths be attributed to eating a particular part of the turtle, such as the viscera?

3. What were the approximate ages of the two people that died? Were they male or female?

4. What were the symptoms of the poisoning?

5. How soon after eating the hawksbill did the people become sick? How soon did the deaths occur?

6. How was the hawksbill cooked?

7. Approximately how many hawksbills, and other turtles, are captured and eaten each year by the people of Nomuka Island?

8. Do hawksbills, or any other sea turtles, lay their eggs at Nomuka?

Thank you for any help that you can give to this inquiry. I have enclosed a color poster showing the different kinds of sea turtles. This may be of use to you in your work. I look forward to hearing from you at your earliest convenience.

Sincerely,

George H. Balazs
Wildlife Biologist

bc: Balazs, HL GHB:iht

Telegrams : Fisheries NUKU'ALOFA
All letters to be addressed
FISHERIES DIVISION, M.A.F.F.,
Telex: Gentel 66225, Fisheries
Telephone : 21-399

Ministry of Agriculture, Fisheries & Forests
P.O. Box 14,
Nuku'alofa,
Kingdom of Tonga.

13th October, 1983

Our Reference Fs/3/4/635

George H. Balazs
National Marine Fisheries Service
Honolulu Laboratory
P.O. Box 3830
Honolulu
HAWAII 96814

Dear Balazs

Please accept my apologies for the late reply of your letter dated 9 August, 1983 regarding your interest on the Hawksbill poisoning in Tonga in early this year.

Today (13 October) the Agriculture Extension Officer working in Nomuka contact me with the following information.

- The poisoning occurs in Nomuka Island (Position Lat.20°.17'S. Long 175°.48' W on the 12 March 1983.
- 18 people became very sick and two person died
- The approximate size of the Hawksbill Turtle is 2'4" long and 1'2" width.
- Caught at Hakaufisi Reef situated at the Western side of Nomuka Island
- The medical Officer that attended the case;

Lata 'i Muli
Medical Assistant
Nomuka
Ha'apai
Tonga Island

Please note that the medical officer that attended the case is not a qualified Doctor.

Tevita F. Latu
for Principal Fisheries Officer

TONGA GOVERNMENT MINISTRY OF AGRICULTURE

Health Centre
Nomuka
Haapai
Tonga
February 7. 1984

To George H. Balazs.
Wildlife Biologist
Honolulu Laboratory
Hawaii

Dear Sir,

Very sorry for late to Reply your letter date October 26. 1983 I just Received the letter on January 30. 1984 and the copy receiving on February 6. 1984. Therefore I am going to Answered the Question according for the information of the Scientist.

A diving fisheries from Nomuka sailed to a reef named Hakau Fish for fishing at Our Neighbour and they got many Hawksbills and Turtles more Twenty altogether and One of the Hawksbills they got is Poisoning.

Answered the Questions

1. No Hawksbill or Turtle Poisoning before at Nomuka or in Tonga.
2. ① Twenty-One people were ate the hawksbill, eighteen were very sick and three died; and three of them were not sick, and Mostly of the very sick and those people who ate the viscera. died
3. The three people were died Name and Sex/Age
   ① ELENOA ONGOONGO — F/8.
   ② SIONA MISINALE ONGOONGO 11/1yrs 7months.
   ③ MATEAKI FONUA I VAHA LATAPU M/9months 29days.
4. Symptoms:- Headache, Vomiting, Weakness, Lost of Power, Pain in the joints and all the body.
5. Said after an hour of eating, Short Head Ache and Vomiting and after 12 hours and death occur.
6. Boiling
7. More Mainly Hawksbill and Turtle.
8. No Hawksbill or Turtle lay eggs at Nomuka.

I hope that this short report will meet your inquiring, Thanks very much for your Color Post showing the different kind of Seaturtles.

Sincerely,

COPY

1-16-84
Dear Mr. Muli - Your help with these questions will be greatly appreciated - George Balazs

U.S. DEPARTMENT OF COMMERCE
National Oceanic and Atmospheric Administration
NATIONAL MARINE FISHERIES SERVICE
Southwest Fisheries Center
Honolulu Laboratory
P. O. Box 3830
Honolulu, Hawaii 96812

October 26, 1983 F/SWC2:GHB

Mr. Lata 'i Muli
Medical Assistant
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Sincerely,

George H. Balazs

George H. Balazs
Wildlife Biologist

SPC FISHERIES NEWSLETTER
NO.26, JULY-SEPT. 1983 (NOUMEA, NEW CALEDONIA)

Spiny lobster culture research programme accelerated after encouraging experimental results

The Central Marine Fisheries Research Institute, Cochin, India, has stepped up its ongoing research programme on the technical aspects of spiny lobster culture following the development of a promising technique which greatly increases the animals growth rate.

The Institute's pulication 'Marine Fisheries Information Service', No. 43, contains two articles claiming very encouraging experimental growth rates obtained in specimens of Panulirus homarus from which the eyestalks had been cauterised. In many crustaceans the eyestalk serves as a centre for hormone production or control in the antagonistic hormonal system which controls moulting, reproduction and growth. Supression of one hormonal process can be used to encourage over-expression of another, and variations of the eyestalk ablation technique have long been used in experiments on crabs, prawns and lobsters to variously promote or inhibit moulting, reproduction and growth and thus gain an understanding of the processes involved.

In the Cochin experiments groups of eyestalk ablated P. homarus were measured for growth against 'control' groups of lobsters which were not treated, were given an excess of food and housed in the same way. Growth in lobsters is a function of the frequency of moulting and the weight increase which occurs at that time, and eyestalk ablation increased both these factors. A three to sevenfold increase in weight was obtained in eye-ablated lobsters relative to the controls. Growth rate is dependent on size, larger animals showing a higher daily weight increment. Thus juvenile eyestalk ablated lobsters recorded an average increase of 1.02 g/day, increasing to 2.50 g/day in maturing and mature lobsters. Equivalent figures for the control animals were 0.35 g and 0.36 g/day respectively. The maximum weight gain of 4.6 g/day was obtained in an ablated individual.

Panulirus homarus is present throughout the SPC region, but is probably the least common of the five species to be found here. However, the technique may be applicable to other species. The Institute has met with success in similar preliminary experiment carried out with P. ornatus, the largest of the species of the SPC region, and P. polyphagus, but P. argus and P. cygnus did not give similar results.

Announcement of the experimental results in India led to a spate of letters and articles, with titles such as 'Lobsters Are Human Too', in India's national daily papers, questioning the ethics of such experiments, which were considered against the country's traditions. The researchers defend their work as comprising only standard experimental methods, and point out that eyestalk ablation is an investigative technique and perhaps a step towards enabling growth rate acceleration by hormonal treatment.

Researcher seeks information on Hawksbill Turtle poisoning

Following a report of a possible case of poisoning from eating a hawksbill turtle (Eretmochelys imbricata) in Tonga, researcher George Balazs of the U.S. National Marine Fisheries Service, Hawaii, is seeking information on other known or suspected cases of turtle poisoning in the Pacific. Dr Balazs, who works at the South West Fisheries Center, Honolulu Laboratory, P.O. Box

3830, Honolulu, Hawaii, would be very grateful to hear from anyone who has any knowledge of incidents of turtle poisoning in any locality.

The following notes on turtle poisoning are extracted from the FAO Synopsis of Biological Data on the Hawksbill Turtle, by W.N. Witzell.

"Hawksbill meat is generally eaten throughout its range, although the meat is often reported to be of poor quality, being dark and oily with a strong flavour. The hawksbill is reportedly poisonous in many areas throughout its range, but often without documented cases. In many instances the hawksbill has been accused of being poisonous, possibly because of its strong flavour, which is believed to vary according to diet.

Hawksbills are not eaten, or are reluctantly eaten because of their reputed toxicity, in the following locations: Gulf of Guinea, Australia, Sri Lanka, Gulf of Siam, Colombia, India, Mauritius, New Guinea, Mexico, West Africa, Seychelles, Senegal, Sudan, Oman. Documented cases of hawksbill turtle poisoning are relatively rare, having been reported from the following locations: Taiwan, Gilbert Islands, Ryukyu Islands, Philippines, Australia, Solomon Islands, New Guinea, Sri Lanka and India.

In reviewing the cases of turtle poisoning and summarizing all pertinent information, one research worker found that poisoning was seasonally sporadic with a geographically intermittent distribution, and could result from eating either the meat, fat, viscera or blood. Symptoms generally appear from within a few hours to several days, depending upon the amount ingested and the person. Major symptoms are: nausea, vertigo, vomiting, diarrhoea, severe gastric pain, sweating, coldness of the extremities, and a dry, burning sensation of lips, mouth and throat. Additionally the tongue develops a white coating with tiny pustules, and victims may suffer from headaches and general lethargy. There are no antidotes, consequently treatment is symptomatic. The overall case fatality rate is 28 per cent, with the oldest and the youngest persons reportedly being more susceptible. Prevention is difficult, since toxic turtles are impossible to differentiate from non-poisonous turtles, the best method being to feed a sample to a dog or cat and wait at least 24 hours. The origin of the toxin is not known but it is believed to be caused by ingestion of poisonous algae, jellyfish or even sea snakes".

Turtle headstarting scheme in Palau

The Government of Palau is currently sponsoring a headstarting programme for hawksbill turtles. Three technicians work within the programme, hatching about 1000 turtles per year and releasing them after 6 months.

FAO World Fisheries Conference announced

The Food and Agriculture Organization (FAO) of the United Nations (UN) have scheduled a major fisheries meeting, entitled the World Conference on Fisheries Management and Development, for June/July 1984, in order to examine the technical, economic and social problems raised by the new regime of the ocean, and the future potential and needs of the fishery sector. The conference, open to all member nations of FAO, the UN and its specialised agencies, and the IAEA (International Atomic Energy Agency) will be held in Rome from 27th June to 6th July.

SOUTH PACIFIC COMMISSION

FISHERIES NEWSLETTER

No. 26 July - September 1983

This issue

Following on the heels of the SPC 15th Regional Technical Meeting on Fisheries, this issue includes a good deal of material which stems directly from its results and recommendations, or which relates to subjects of particular interest raised during discussions. Two articles (numbers 3 and 5 on the contents list below) were produced in response to specific requests for information made by the meeting.

Our regular selection of 'News from around the Region' is steadily expanding and gaining broader coverage, largely thanks to the efforts on the part of readers and correspondents who have sent in notes, reports, press clippings and other details of items of interest. The editors sincerely appreciate any material received, and we thank all those who have contributed so far.

Contents

ACTIVITIES

SPC 15th Regional Technical Meeting on Fisheries

This year's technical meeting, attended by 42 official delegates and 38 observers, ran well despite a very full agenda and heavy workload for many participants.

TURTLE MEAT (*ERETMOCHELYS IMBRICATA*) POISONING IN NETHERLANDS NEW-GUINEA *

by

T. ROMEYN & G. T. HANEVELD

Public Health Service and Royal Netherlands Navy Medical Corps, Hollandia, Netherlands New-Guinea

INTRODUCTION

The Papuans are keen on hunting turtles in the seas around New-Guinea as their meat is considered a delicacy. LOVERIDGE (1948) described four species of turtles in these waters. Publications from various countries reported on poisoning—usually mass poisoning—by this meat; cases, often ending fatally, occurred in India, Ceylon and the Philippine Islands. As regards the East-Indian archipelago, VAN HASSELT (1922) mentioned poisonous turtles around New-Guinea. The Public Health Service of the Netherlands East-Indies instituted an enquiry into the matter (*Mededelingen*, 1933) and received several positive answers; some doctors reported their personal experience, others from hearsay. The symptoms were dizziness, nausea and vomiting, in a few cases with fatal outcome. BIERDRAGER (1936) recorded an instance of mass poisoning in about 52 people, five of whom died, after eating the meat of a large turtle (of the Cheloniidae species) on the Isle of Japen north of New-Guinea. The pathologist, BONNE (cited by BIERDRAGER, 1936), found fatty degeneration of the liver and kidneys. SIEGENBEEK VAN HEUKELOM (1936) also described degeneration of the liver, pancreas and kidneys.

PERSONAL EXPERIENCE

On August 24, 1954, a number of people in Kaipuri (Schouten Islands) ate turtle-meat. The government doctor arrived a week later; in the meantime one man had died after two days and another after six days. Four others suffered from vomiting, cramps and unconsciousness one week after the meal; they took a long time to recover.

All cases of turtle-poisoning in Netherlands New-Guinea and Indonesia were caused by *Eretmochelys imbricata* (hawk's bill turtle, vernacular name *tuduruga sisir*). This turtle may reach a length of 85 cm and a weight of about 20 kg; the jaws are curved like a bill or beak—hawk's bill—and the head has four prefrontal horn plates. The carapace has four overlapping horn plates, in contrast to *Chelonia mydas* (green turtle or soup turtle).

SYMPTOMS

(1) *General:* The symptoms usually begin about twelve hours after the meal: a feeling of distress often coupled with vomiting, dizziness and a burning sensation in the throat, sometimes also of the tongue, gums and lips. Headache and abdominal

* Originally published in Dutch in *Nederlandsch Tijdschrift voor Geneeskunde*.

References p. 382

pain, sometimes diarrhoea. Mild cases may show only a few of these general symptoms.

(2) *Mouth and throat* affection becomes manifest after about two days; the tongue is white-furred, the mucous membranes of the mouth and throat are swollen and sometimes swallowing becomes difficult. From two to ten days afterwards red elevations, the size of a pin's head, appear on the tongue; these are the swollen papillae, especially near the point and along the edges. The papillae may still be visible after two months.

(3) *Nervous system:* somnolence occurs at an early stage. The patients may react when spoken to, but they fall asleep again immediately. This somnolence may lead rapidly to death. Occasionally the patients are restless. There is thermolability; some authors mentioned hypotony and hyporeflexia, but others did not observe these symptoms.

Course and prognosis. The severity of the affection is proportionate to the amount of meat eaten, but even in serious cases the prodromal symptoms do not start until 12 hours after the meal, though they are more intense then. Vomiting stops after a few days.

The light cases show the mouth and throat symptoms only; in severe cases the clinical picture is dominated by disturbed consciousness. None of the nine somnolent patients seen by BIERDRAGER (1936) recovered. One of the Kaipuri patients was unconscious for a week and then recovered slowly. In cases of prolonged somnolence the development of ulcerating stomatitis is possible, with intense *foetor ex ore*. KARIADI (*Mededelingen*, 1933) reported such a case; it ended fatally.

Treatment. In the first stage of the disease the stomach and intestinal tract should be emptied by emetics, gastric lavage, clysters and/or laxatives. In the later stages treatment can only be symptomatic, by administration of excitants. The symptoms of the mouth and throat may be alleviated by boracic glycerin and rinsing with permanganate of potassium.

What is the cause of the toxicity of the hawk's bill's meat?

Some authors assume that the turtle's meat is poisonous in certain seasons only; this is probably incorrect as cases have occurred in nearly every month of the year. Possibly only a certain variety of the species is poisonous, as is the case in many kinds of fishes. For the West-Indian *ciguatera*, a well known type of fish poisoning, one of the theories is that the toxin is the result of the dietetic habits of the fish (ARCISZ, 1950)

The hawk's bill is a carnivore; with its sharp beak it tears crabs, molluscs, perhaps also seaweed from the coral-reefs. Several toxic and irritating species exist among the coral fauna and vegetation, as evidenced *e.g.* by coral dermatitis manifesting itself by redness, oedema and cutaneous paraesthesia. Both fish and turtle poisoning might derive its origin from coral fauna or vegetation; extensive coral formations exist near all localities where turtle poisoning has been reported.

Neither a bacterial origin of the toxin nor a relationship with toxic products from the generative organs of the turtle have as yet been proved. Thus the problem of poisoning by turtle meat is still unsolved.

SUMMARY

In Netherlands New-Guinea, two patients died two to six days after eating turtle

References p. 382

meat; a week after the meal four other persons developed symptoms, including vomiting, cramps and unconsciousness; recovery was very slow.

Comparison with cases described in the literature reveals that there are two important groups of symptoms, *viz.*, symptoms of the mouth and throat (swelling, redness of the buccal mucous membranes, white coating, and protracted swelling of the papillae of the tongue) and nervous disturbances, especially somnolence.

In the case of nervous disturbances the prognosis is less favourable. All cases were attributable to the carnivorous hawk's bill turtle (*Eretmochelys imbricata*). The toxicity of its meat is probably due to the poisonous coral vegetation on which the turtle feeds.

Treatment is chiefly symptomatic.

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ARCISZ, W. (1950), *Ciguatera, special scientific report in fisheries*, no. 27, Washington, D.C. – BIERDRAGER, J. (1930), *Geneesk. Tijdschrift v. Ned.-Indië*, **76**, 1933. – HASSELT, F. J. F. VAN (1922), *Trop. Natuur*, 157. – LOVERIDGE, A. (1948), *Bull. Museum comp. Zoology*, **101**, 315. – *Mededelingen van de Dienst Volksgezondheid in Ned.-Indië* (1933), **22**, 121. – SIEGENBEEK VAN HEUKELOM, A. (1930), *Geneesk. Tijdschr. v. Ned.-Indië*, **76**, 1954.

B.R. 519 (RESTRICTED)
GEOGRAPHICAL HANDBOOK SERIES
FOR OFFICIAL USE ONLY

PACIFIC ISLANDS

VOLUME I

GENERAL SURVEY

August 1945

Gt. Brit.
NAVAL INTELLIGENCE DIVISION.

in food or drink contaminated with faecal material or through contact with a carrier of the causative bacilli; flies probably play an important part in transmission.

Nutrition and Nutritional Diseases

Most Melanesians and Polynesians take a varied diet, which is conducive to good nutrition. They have a variety of crops, and use forest leaves, roots and fruits. Pigs and hens are almost universal, but cattle as a rule are not kept. Polynesians usually live close to the sea and collect a great variety of fish, molluscs and other animal foods. With the advent of white men, there is a tendency to use tinned foods, the effect of which on nutrition cannot yet be assessed. In New Guinea taro, breadfruit, maize, bananas, sweet potatoes, sugar cane, sago, tapioca (manioc) and yams are largely eaten, with small quantities of animal fat and ground nuts. Pig flesh, fish, birds, and shellfish are also eaten, but, in the interior, sparingly.

In the Japanese mandated islands taro and tapioca are staple foods and these, with yams, sweet potatoes, fruits and vegetables form the main articles of diet throughout the south Pacific also. Pigs, though common, are usually only eaten at feasts; eggs of fowls and turtles are used to some extent; milk is not available on most islands. While there is no actual shortage of food, therefore, it is evident that the common diet is ill-balanced. It is said that tropical phagedaenic ulcer is specially common in sago eaters and rare in fish and taro eaters.

The diet of the Pacific islanders, like that of many other primitive peoples, contains too much carbohydrate and too little protein and fat. It is also deficient in vitamins. In New Guinea this deficiency is said to apply particularly to vitamins A and C. Shortage of vitamin B does not, apparently, occur to a serious extent, though in plantation labourers, who usually subsist on a diet which differs from that of village natives, shortage of this vitamin has led to outbreaks of *beriberi*. Infant beriberi has been reported from western Papua and is one of the causes of high infant mortality. *Scurvy*, indicating deficiency of vitamin C, is also seen in Papua and New Guinea, where it was known as 'New Guinea sore mouth', and certain eye conditions which suggest vitamin A deficiency have been noted. Incidentally, in some parts of Papua there is a shortage of iodine in the soil, indicated by the high incidence of *goitre*.

In the Solomons the main deficiency is of vitamin A, which has been held to be, in some part, responsible for the high incidence of

disease of the respiratory organs. Vitamin D is also lacking, but sunlight largely makes up for this. In French Oceania there is vitamin B1 deficiency among Chinese and Annamite labourers. In Nauru, in 1926 and 1936, there were serious outbreaks of infant beriberi, and this still caused, in 1940, eight out of the 34 deaths recorded. This very fatal condition is being prevented by the use of unfermented toddy (prepared from the spathe of the coconut). Unfermented toddy (*ekarawe*) is rich in vitamins A, B and D, and its use is encouraged for expectant mothers and infants. It is the use of the fermented product (*demani*) which is restricted in Nauru, and not of *ekarawe* as has sometimes been stated. Beriberi exists also in the Gilbert and Ellice islands. In Fiji a form of *epidemic dropsy* has been reported, but it is not clear if this is the same as the epidemic dropsy which occurs in India and which is due to poisoning from the use of mustard oil contaminated with the products of *Argemone mexicana*. In Tonga vitamins A and D are deficient, and there is a shortage of calcium, iron and iodine.

Poisonous fish are present in the waters around the Japanese mandated islands, the Ellice islands, Tonga and French Oceania; the fish concerned include the sea-perch, *Ephinephelus merra*, the eel *Anguilla mauritiana*, the parrot-wrasse *Pseudoscarus abacurus* and the sea-bream *Monotaxis grandoculis*. The symptoms of poisoning are nausea, vomiting, diarrhoea, and paralysis of the legs. Death is rare; it occurs only among strangers unfamiliar with local fish. The turtle *Chelonia imbricata* may be poisonous; 52 cases of poisoning, with 9 deaths, have been reported from Wooi on the north coast of New Guinea. Whether there has been a temporary alteration in the species to become poisonous, or whether a particular variety of the species is concerned, is not known. The symptoms of poisoning are characteristic.

It may here be noted that *undulant fever*, usually acquired by drinking milk from cattle or goats infected with the disease, occurs in the Hawaiian islands. It is not reported in the south Pacific except from Australia and New Zealand.

Depopulation

The depopulation which has occurred in many of the Pacific islands is a subject upon which a great deal has been written. Many factors, physical and psychological, appear to have a bearing on this matter; so far as health is concerned, however, it would be difficult to deny importance to the high infant mortality which is the general rule,

resembles superficially in possessing an elongate beak. The largest skull known from any living thecophoran is an Australian one in the Bell Col
Cambridge. It is 285 mm. wide and probably belonged to an animal weighing about 600 lb. The dimensions of an ordinary sized mounted femal
Colombo Museum are—head length 220 mm., head width 185 mm., curved carapace length 960 mm., plastral length 650 mm., bridge length 340 mm
specimens had a straight carapace length of (*a*) 975 mm. and width of 730 mm., (*b*) 840 by 690 mm., (*c*) 795 by 655 mm.

Distribution. The Indo-Pacific, Mediterranean and East Atlantic as far north as England and Norway where it is straggler.

The following European specimens of this race are (*a*) in the British Museum, an adult from Italy, another from the East Atlantic, as well as t
that were half grown, and two spirit specimens from Selsey bill, Sussex, and from Eire; (*b*) in the Whitby Museum; (*c*) in the Dublin National Museum—
1938 from Blacksod bay, West Mayo, April 16, 1945; No. 11—1945, from Galway bay, and 39—1945, from Tralee, County Kerry.

BR
Be

(See fig. 19)

Subfamily Eretmochelinae

Prefrontal scales in two pairs, costal scutes in four pairs, the first separated from the prevertebral by the first vertebral, four enlarged infr
on each side, intergular well developed, dorsal scutes strongly imbricate up to middle age, thereafter juxtaposed, claws two on each limb, choana
1 or 2, pigmentation highly variegated. Pleuro-peripheral vacuities disappear with age (fig. 20).

A single monotypic genus.

Genus *Eretmochelys* Fitzinger

Caretta Ritgen, 1828, Nova Acta Acad. Leop. Carol XIV. (Partim. Type *Chelonia imbricata*.)
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Characters same as in subfamily. Beaks compressed, the lower with a feebly denticulate edge; mandibular symphysis as long as one and
depth of narial aperture. A single species.

Eretmochelys imbricata (Linné)

Plate 4 and

Testudo imbricata Linné, 1766. Syst. Nat. ed. 12, p. 350; (American and Asian seas).
Eretmochelys imbricata Fitzinger, 1843. Syst. Rept. p. 30; Deraniyagala, 1930. Spolia Zeylanica XVI, p. 70, pls. XI, XII.
Chelonia pseudo-mydas Lesson, 1834. In Belang. Voy. Ind. Or., Zool. p. 299 (type loc. Atlantic).
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Local names. (Sinhala) *Potku käsbāva, Leli käsbāva* = Shell turtle; *Panā käsbāva* = Comb turtle. (Tamil) *Alunk amei.* (English) *Haw
Caret, Shell turtle.*

The hawksbill turtle derives this name from its compressed bird-like beak and slender head. Until middle age it is easily identifiable b
imbricate scutes (Pl. V) which become juxtaposed and thin after this stage (fig. 19 *c*.).

Scalation. Two pairs of prefrontals, prevertebral separated from the costals, the conspicuous intergular and four pairs of costals establish
This species furnishes the valuable "tortoise shell" of commerce which is thought to lose its translucent lustre if taken off a dead animal, conse
scutes of the living turtle are smeared with coconut oil and heated with a lighted taper whereupon they soften and are readily detached, after which
is released. The prevertebral scute however is not removed as fishermen affirm that to do so would cause its death. Experiments upon a capti
showed that the scutes so removed are completely regenerated within about six months, but in clean sea water this process would have been com
earlier. The scutes attain to commercial value when the animal is about 2½ years old. They are now imbricate with the vertebrals larger than
the carapace outline is cordate. They are partially shed periodically, the process commencing upon the anterior aspect of the plastron. Whe
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about half the length of its posterior edge (fig. 19 *c*) whereas in *Chelonia mydas* they are subequal (fig. 19 *f*).

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BRASS RING TAG
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(See fig. 19)

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(See fig. 20)

(*b*) Adult with yellow interspaces between dark scales, carapace scutes an aggregation of black, orange, reddish brown and yellow blotches, the black predominating with age. Plastron gamboge yellow. Inside of mouth pale pink with dark blotches. Specimens inhabiting atoll lagoons become encrusted with mud and algae, and the scutes thus deprived of sunlight are lighter colored than those from more active water which prevents this.

Osteology. Maxillaries separated by vomer, frontal enters orbit, the alveolar area possesses a premaxillo-maxillary ridge, the fontanels in the choanal passage are visible ventrally. Neural bones 11 to 9, the posterolateral sides of each being much longer than the anterolateral ones; the pleuro-peripheral fontanels disappear with age and the plastron also loses its fontanels more or less completely (fig. 20).

Food. Generally carnivorous but not infrequently subsists entirely upon algae and is also known to feed upon fruit of the red mangrove (Carr). It is mainly a diurnal feeder, for a captive specimen neglected live food at night until lit up by electricity. In seizing a crab it would wait for the victim to move, then grab it from its side, thus avoiding its chelae. An adult's stomach contained a black Ascidian.

Toxicity. The flesh is edible but insipid. At times it is virulently poisonous and has caused numerous deaths. Its toxicity is thought to be due to the diet of the animal at the time; accordingly fisherman chop up its liver and throw it to the crows before cooking its flesh. If the crows refuse it, the animal is discarded. Another test is to mix the raw flesh with slaked lime which turns greenish if the flesh is poisonous.

At Mandaitivu twenty-four persons were poisoned in June, 1921, by eating this turtle and seven of them died after two days. The Medical Officer of Health reported that the symptoms were inflammation of the mouth, a burning sensation in the stomach, weals on the body, nausea and pains in the limbs.

On December 3, 1941, several deaths occurred at Habaraduva in the south. The first symptoms commenced in children within 5 or 6 hours, and in adults between 10 and 12 hours. Some of the former died after 24 hours; the adults recovered. The symptoms were convulsions and coma in children; vomiting, severe abdominal pain and drowsiness in adults.

Reproduction. There are two maximum periods of breeding intensity. In December and January, and in April, May and June; about 115 eggs are laid at a time and are 35 to 37·5 mm. in diameter. The young hatch out in from 50 to 60 days according to the weather; the carapace is 39 to 42 mm. long in the newly hatched.

Variation.

Carapace scute variation in two broods of *Eretmochelys*

(*a*) Bentota brood (mother's carapace vacuities closed, an old specimen) (fig. 20*b*)

L. M.	L. C.	V.	R. C.	R. M.	No. of turtles
13	4	5	4	13	2
13	4	5	4	12	1
12	4	4	4	13	1
12	4	5	4	13	3
12	5	5	4	13	1
12	5	5	4	12	1
12	5	6	4	12	1
12	4	5	4	12	11
11	4	5	4	12	2

(*b*) Karaduva brood (mother's vacuities open, a young adult) (fig. 20*a*)

12	4	5	4	12	18

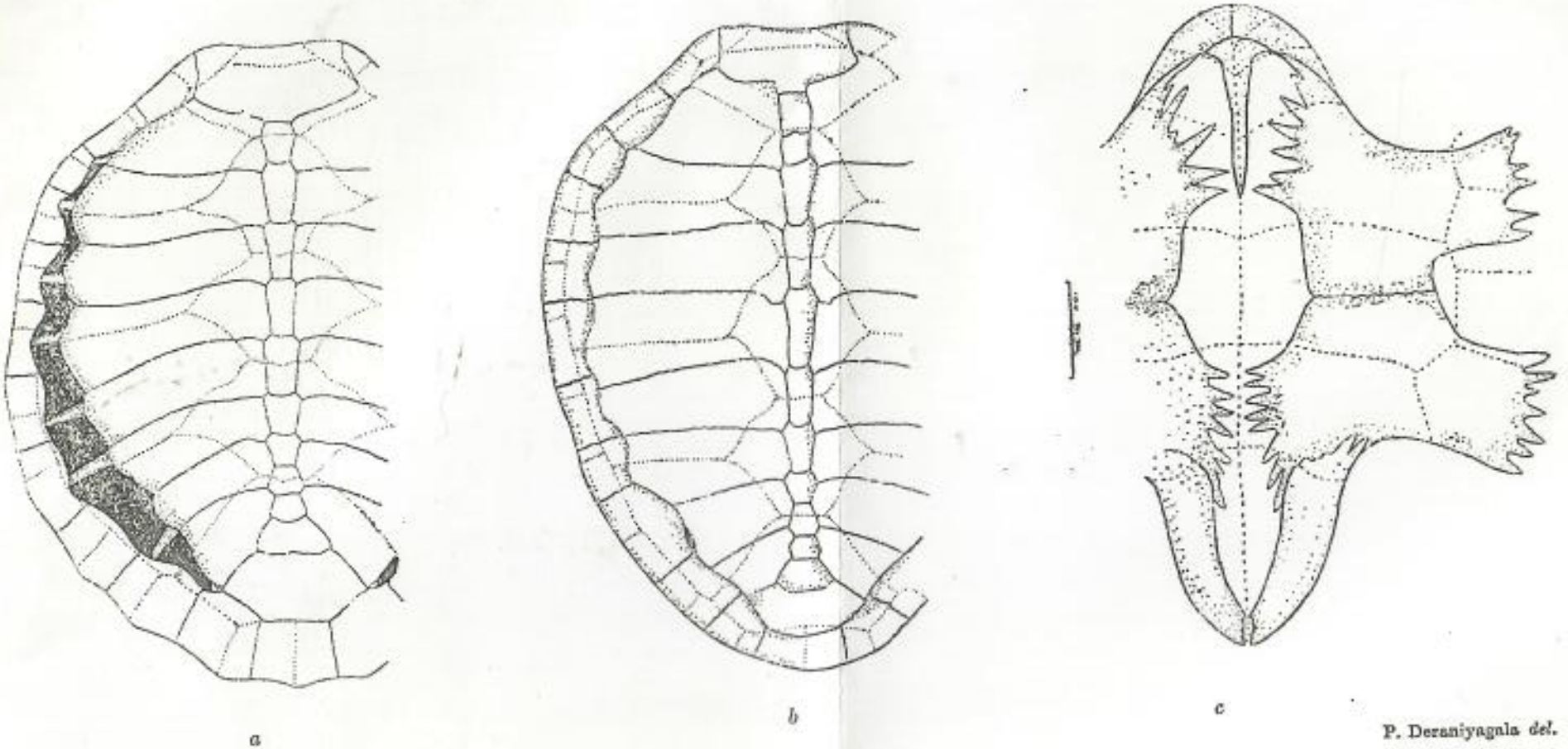

Fig. 20. Corselet ossification in *Eretmochelys imbricata*.

a young adult. *b* old Bentota female × 1/7. *c* plastron of young adult female × 1/6. *Black* = vacuities.

Out of twenty-six specimens three had the frontal scute fused to the frontoparietal. In the Bentota brood there were eight types of variation from the normal and out of twenty-two young only eleven were normal, whereas in the Karaduva brood all eighteen young examined were normal. This suggests that the offspring of old females are more variable than those of young ones.

Dimensions:

TABLE

Dimensions of *Eretmochelys* in mm. and gm.

Turtle	1	2	3	4	5	6	7	8	9	10	11
Head length ..	18	22	23	45	75	95	100	145	185	185	
Carapace length ..	30	52	58	152	285	360	400	600	737	850	750
Plastral length ..	—	42	49	120	209	265	300	470	—	650	—
Plastral width ..	—	—	—	—	—	—	—	—	—	650	595
Bridge length ..	17	—	30	67	119	148	180	300	235	350	
Frontal length ..				9	16	17	21	25	27		
Frontoparietal length ..				14	18	23	25	31	35		
Parietal length ..				9	15	28	27	39	55		
Weight in gm. ..							12700 (28 lb.)	36288 (80 lb.)	44452 (98 lb.)	53524 (118 lb.)	
Remarks ..	just hatched	43 days old	54 days old	Mus. sp.	Mounted sp.	Mus. sp. in spirits	Udappuva sp.	Talaimannar ♀	Bentota ♀	Karaduva ♀	

Distribution. The hawksbill, like the green turtle, is found in all tropical and subtropical oceans and with other marine Thecophoroidea keeps comparatively closer to land than the Athecoidea. It seldom strays into lagoons in Ceylon although common in the muddy lagoons of the Aldabra and Cosmoledo atolls in the Indian Ocean (Hornell). The northern range of the turtle in the Indo-Pacific appears to be the Hainan region of China; it is common among the Riu Kiu islands (Stejneger 1907, p. 511) and also occurs at the Pescadores (Horikawa 1930, p. 23) while in the South it is known from East Australia along the more northern part of the Great Barrier Reef.

So eagerly is it hunted for its valuable scutes that its numbers are greatly depleted and although in Bennett's day (1843) Government farmed the right to capture these turtles, today it is too scarce to do so profitably.

The homing instincts of this turtle are probably stronger than in most animals, for Bennett states that individuals stripped of their scutes continued to visit the same locality each following year although their scutes were removed on each occasion.

Evidence of these repeated visits was obtained in 1794 when the Dutch Commandant of the south-eastern coastal part of Ceylon marked several turtles with brass rings. One of these rings was recovered by a renter who brought it to Bennett in 1826 and affirmed that to his certain knowledge this turtle had revisited the cove of Amaidhuva for those thirty-two successive years. Bennett (p. 275) replaced the ring upon the animal and liberated it.

Ceylon, India, Seychelle, Maldive Islands, Singapore, Malay Archipelago, Philippines, China, Formosa, New Guinea, Australia, Samoa, Arabia, Africa, Tohuantepec, Gautamala, Bahamas, West Indies, Brazil, Florida, and along the North American coast as far north as Buzzard's bay.

Subfamily Cheloniinae

Color?

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12-2-82 Not listed in Catalog. I need to look through the card catalog

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CEYLON NATIONAL MUSEUMS PUBLICATION

A COLORED ATLAS OF SOME VERTEBRATES FROM CEYLON

(Illustrated by the Author)

VOLUME TWO

TETRAPOD REPTILIA

By

P. E. P. DERANIYAGALA

Director of National Museums, Ceylon

1953

pecimen from California, referred to *Chelonia virgata* by ..., exhibits the same generical affinities. A further study of ...em, together with a comparison of specimens from the various localities where they are said to occur, will undoubtedly reveal some curious results. We subjoin the following references:

Syn.—*Chelonia virgata*, Schw. Prodr. Monogr. Chelon. in Arch. Koenigsb. I, 1812, 291 & 411.—Cuv. Règn. anim. 2d ed. II, 1829, 14.—Guér. Iconogr. du Règn. anim. 1834, Rept. Tab. 1, fig. 4.—Dum. & Bibr. Erpét. gén. II, 1835, 541.—Gray, Catal. Tort. Croc. & Amphisb. Brit. Mus. 1844, 54; &, Catal. Shield. Rept. Brit. Mus. I, 1855, 74.—Cantor, Catal. Rept. Malay. Penins. 1847, 11.—Agass. Contr. Nat. Hist. U. S. Amer. I, 1857, 379.

It is easier to conceive how a sea-turtle might, from the eastern coast of Asia, reach the Red Sea, than its passage from the same coast to California, or *vice versâ*.

Genus CARETTA, Merr.

Gen. Char.—Head small, anteriorly compressed and tapering forwards; snout declivous and protruding; jaws robust, with a blunt and even margin, which is nearly horizontal to the tips. Two pairs of frontal plates; a vertex plate and one pair of parietals; a middle occipital, rather large; two pairs of latero-occipitals, and one pair of postoccipitals. Three postoculars. Mental shields none. Side of lower jaw with an elongated plate. Carapax cordate, ovate, covered with thirteen imbricated shields; marginal shields twenty-five, constituting posteriorly a serrated edge. Plastron, with six middle pairs of shields, and four lateral ones; several postaxillars. Two claws to either flipper.

Syn.—*Caretta*, Merr. Tent. Syst. Amph. 1820, 17.—Fitz. Neue Class. Rept. 1826, ...—Bonap. Amph. Europ. &c. 1839, 12.—Gray, Catal. Tort. Croc. & Amphisb. Brit. ...us. 1844, 53; &, Catal. Shield. Rept. Brit. Mus. I, 1855, 73.
Chélonées imbriquées, Dum. & Bibr. Erpét. gén. II, 1835, 547.
Cheloniae imbricatae, Cantor, Catal. Rept. Malay. Penins. 1847, 12.
Eretmochelys, Fitz. Syst. Rept. 1, 1843, 30.—Agass. Contr. Nat. Hist. U. S. Amer. ..., 1857, 380.

Observ.—Although the name *Caretta* was framed as early as 1820, this genus was really distinguished and characterized as a natural group, by Duméril and Bibron, fifteen years later, and not by Fitzinger, who wrote eight years after the second volume of the "Erpétologie générale" was published, and who, moreover, never characterized the genus.

A better name than *Caretta* could not have been selected to designate this genus, viewed in the same light as *Caouana* for the Loggerhead; and, it having priority over its competitor, *Eretmochelys*, there is no plausible reason for rejecting it. To say that its present limits are not those originally ascribed to it by Merrem, is mere trifling. Was the Shell Tortoise, *Caretta imbricata*, not included in it by Merrem himself? How many genera of the older writers have met with the same fate, and yet have been universally adopted, although in a restricted sense.

Hence, we cannot perceive why the name *Eretmochelys* should "now be retained," and on what ground "no one has a right to change it hereafter."*

There are several well-marked species of Carets distributed over the warm temperate and torrid zones of both hemispheres. The typical one, and, perhaps, the most ancient on scientific record, is that of the West Indies, or *Caretta imbricata*, Merr. The East Indian species, *Caretta squamosa*, must have been known to navigators and traders before the discovery of America by Columbus; but its history is interwoven with that of *C. imbricata*, to such an extent as to make it a difficult task to divide the various synonyms between the two. We dare say most of the writers of the eighteenth century have spoken of the two indiscriminately, whether they drew their descriptions or observations from specimens or simply quoted their predecessors. At any rate, if the specimens were before them, they never questioned the identity of the two species, hence, never instituted a series of critical comparisons, owing, perhaps, to the fact, that the materials at their command were in too fragmentary a condition.

The Carets of the Polynesian Sea constitute likewise a peculiar species, distinct both from *C. imbricata* and *C. squamosa*. Furthermore, we should not be surprised at hearing of the existence of more than one species in the South Pacific Ocean. The specimens brought home by the U. S. Exploring Expedition seem to foretell that such is

* Contrib. to the Nat. Hist. of the U. S. of Amer. I, 1857, 380.

...tion of things. Future investigators alone will be competent ...cide the question rightfully, should they enter the field well ...epared for conducting a series of observations upon all the speci-mens which an Antarctic cruise is likely to place before their eyes.

The "Note-book" of the Expedition, under the head of Broken Bay, Southeast Australia, states that a small specimen of the Caret genus had been observed at that place, and that "it might prove distinct from the Feejees species."

The various species of the genus *Caretta* yield the Tortoise-shell of commerce, which is of various qualities, affecting its market price.* This fact alone would seem to point at a diversity of species. Their flesh is, generally speaking, of an inferior quality, and unpalatable, to Europeans, especially in the East and West Indies. Indeed, in the West Indies, it is spoken of not only as unpalatable but as possessing highly cathartic properties. In the South Pacific Ocean, however, we are informed that "it was tried repeatedly, and not found at all inferior" to that of the true Cheloniae.

1. CARETTA IMBRICATA, Merr.

SPEC. CHAR.—Carapax subcordiform, rather elevated; dorsal region shelving; periphery deeply emarginated posteriorly. Vertebral shields ridged along their middle: anterior one triangular; the remaining four rhomboid. Middle occipital plate much broader than long. Middle postorbital smaller than the other two. Skin of the neck without horny plates. Ground color yellow or fawn, marmorated with brown.

SYN.—*Testudo imbricata*, LINN. Syst. Nat. ed. X^a, I, 1758, 197; &, ed. XII^a, I, 1766, 350.—WALL. Chelonogr. 1782, 46 & 110.—SCHN. Allg. Naturg. Schildk. II, 1783, 309; &, Beytr. Naturg. Schildk. in *Leipz. Magaz. z. Naturk.* 1786, 258.—GMEL. in *Linn.* Syst. Nat. ed. XIII^a, I, III, 1788, 1036.—LACEP. Quadr. ovip. I, 1788, 105. Tab. II.—SCHŒPFF, Hist. Testud. 1792, 83. Tab. XVIII A, & XVIII B.—DONND. Zool. Beytr. III, 1798, 3.—CUV. Tabl. élém. d'hist. nat. 1798, 288; Règn. anim. II, 1817, 13; 2d ed. II, 1829, 13; &, ed. illustr. Rept. 19.—LATR. Hist. nat. Rept. I, 1802, 50. Pl. I, fig. 2.—SHAW, Gen. Zool. III, I, 1802, 89. Tab. XXVI & XXVII.
Testudo caretta, RAY, Synops. meth. Anim. Quadr. & Serp. gen. 1693, 258.

* Annals and Magazine of Natural History. Second Series. Vol. IV, 1849, 297.

Testudo caretta, KNORR, Delic. nat. II, 1767, 124. Tab. XXX.—CATESB. Nat. Hist. Carol. II. 1771, 39. Tab. XXXIX.—BONNAT. Encycl. méth. Erpét. 1789, 21. Pl. IV, fig. 1.—DAUD. Hist. nat. Rept. II, 1803, 39. Pl. XVII, fig. 2.
Chelonia imbricata, SCHW. Prodr. Monogr. Chelon. in Arch. Koenigsb. I, 1812, 291 & 408.—MAXIMIL. Beytr. Naturg. Bras. I, 1825, 24.—GRAVENH. Delic. Mus. Zool. Vratisl. I, 1829, 6. Tab. I, fig. 2.—WAGL. Naturl. Syst. Amph. 1830, 133.—GRAY, Synops. Rept. in *Griff.* Anim. Kingd. IX, 1831, 62.—DUM. & BIBR. Erpét. gén. II, 1835, 518.—TEMM. & SCHLEG. in *Sieb.* Faun. Japon. Erpét. 1838, 13. Tab. V, figs. 1 & 2.—HOLBR. N. Amer. Herp. II, 1842, 29. Pl. VII.—TSCH. Faun. Peruan. Herp. 1845, 22.—BELL, Hist. Brit. Rept. 1849, 1 (viguette).
Caretta imbricata, MERR. Tent. Syst. Amph. 1820, 19.—GRAY, Catal. Tort. Croc. & Amphisb. Brit. Mus. 1844, 53; &, Catal. Shield. Rept. Brit. Mus. I, 1855, 74.
Chelonia multiscutata, KUHL, Beytr. Zool. & vergl. Anat. 1820, 78.
Eretmochelys imbricata, FITZ. Syst. Rept. I, 1843, 30.—AGASS. Contr. nat. Hist. U. S. Amer. I, 1857, 381.
Testudo marina americana, SEBA, Thes. nat. I, 1734. Tab. LXXX, fig. 9.
Testudinis marinae pullus, SEBA, Thes. nat. I, 1734. Tab. LXXIX, fig. 6.
La Tortue Caret, DUTERT. Hist. gén. Antil. II, 1662, 229.—BONNAT. Encycl. meth. Erpét. 1789, 21.
Scaled tortoise-shell, GREW, Mus. Reg. Soc. 1681, 38. Tab. III, fig. 4.
Le Caret, LABAT, Voy. aux Isles de l'Amér. I, 1722, 304.—LACEP. Quadr. ovip. I, 1788, 105. Tab. II.—CUV. Règn. anim. II, 1817, 13; 2d ed. II, 1829, 13; &, ed. illustr. Rept. 19.—BOSC, in Nouv. dict. d'hist. nat. XXXIV, 1819, 255.
La tuilée, DAUD. Dict. Encycl.
The Hawk's-bill Turtle, BROWNE, Civil and Nat. Hist. of Jamaica, 1756, 465.—CATESB. Nat. Hist. Carol. II, 1771, 39. Tab. XXXIX.
La Chelonée Caret, LESS. in Bélang. Voy. Ind. Orient. Zool. 1834, 300.

OBSERV.—The above specific characters are derived from the "Erpétologie générale." The synonymy is given for the reasons already stated, that the history of this species is interwoven with that of *Caretta squamosa*.

LOC.—Atlantic Ocean: West Indies especially.

In the Zoology of Bélanger's "Voyage aux Indes Orientales," pp. 301 & 302, Lesson mentions, as occurring in the Atlantic Ocean:

1. *Chelonia pseudocaretta* (LA Chélonée faux Caret), and,
2. *Chelonia bicarinata* (LA Chélonée à sternum bicaréné).

But his descriptions of the same are so inaccurate as to leave us in doubt regarding the true affinities of these Turtles.

UNITED STATES

EXPLORING EXPEDITION.

DURING THE YEARS

1838, 1839, 1840, 1841, 1842.

UNDER THE COMMAND OF

CHARLES WILKES, U.S.N.

HERPETOLOGY.

BY

CHARLES GIRARD,

DOCTOR IN MEDICINE AND SURGERY; CORRESPONDING MEMBER OF THE BOSTON SOCIETY OF NATURAL HISTORY; THE ACADEMY OF NATURAL SCIENCES OF PHILADELPHIA; THE LYCEUM OF NATURAL HISTORY OF NEW YORK; THE ELLIOT SOCIETY OF NATURAL HISTORY OF CHARLESTON, S. C.; THE CALIFORNIA ACADEMY OF NATURAL SCIENCES, SAN FRANCISCO; THE "SOCIETE HELVETIQUE DES SCIENCES NATURELLES;" THE "NATURFORSCHENDE GESELLSCHAFT IN ZURICH;" AND THE "SOCIETE DES SCIENCES NATURELLES DE NEUCHATEL, (SWITZERLAND,)" ETC. ETC.

WITH A FOLIO ATLAS.

PHILADELPHIA:
J. B. LIPPINCOTT & CO.
1858.

The Gilbert and Ellice Islands Colony

SAILING DIRECTIONS
FOR THE
LINE ISLANDS

Captain E.V. Ward M.B.E., F.R.I.N.
Master Mariner.

1974

Training Ship
"Teraaka",
October 1974

Fish

All the islands abound in game fish, reef fish, crabs, coconut crabs, rock lobsters and turtles. Most game fish including tuna, kingfish, wahoo, trevally, marlin and sailfish can be eaten with impunity as can crabs and rock lobsters. Very large barracuda and some species of shark are sometimes poisonous. On reef fish local advice should be sought, but as this is not available at the uninhabited islands any brightly coloured bottom fish should be treated with caution. The imperor cod, or red schnapper is certainly very poisonous in Washington, Fanning, Christmas, and Malden. In 1968 a yachts crew were taken seriously ill at Malden after eating the emperor cod and some died. Eels, puffer fish, and hawksbill turtles should never be eaten without local advice.

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PLEASE ADDRESS REPLY TO
THE SECRETARY-GENERAL

Dr. G. Balazs
Wildlife Biologist
NMFS Honolulu Laboratory
P.O. Box 3830
Honolulu
Hawaii 96812

29 August 1983

Dear George,

Thanks for your letter of 10 August with the copy of your letter to Simisi Fakahau regarding the reported case of poisoning from eating Hawksbill turtle in Tonga. I appreciate being kept informed and will include a brief note along the lines requested in the next Fisheries Newsletter.

Regards.

Barry

B.R. Smith
Fisheries Adviser
for Secretary-General

BRS/Ab

MARINE TURTLES IN THE REPUBLIC OF SEYCHELLES:

a Report on Their Status and Management

World Wildlife Fund Project 1809

Jeanne A. Mortimer, PhD.

November 1983

Table 13. The symptoms of turtle meat poisoning.

Severe vomiting and diarrhea

Hot sensations in abdomen; Cold sensations in extremities

Mucus membrane of mouth and throat is red, swollen and sore

Tongue coated in white membrane

Heavy salivation and difficulty in swallowing

Foul breath

Boils

High fever; Chills

Loss of hair

Peeling of skin over most of body

Affects liver and kidneys

Frequent urination of highly colored urine

Dizziness

Blurred vision

Headache

Sleepiness followed by coma

Death in 28% of cases

Note: Those who do survive often take many months to fully recover.

Table 12. Reported cases of poisonous turtle meat.

Date	Location	No. of Cases	Deaths	Species of Turtle	Reference
1697	Windward Is., Caribbean	2	0	Hawksbill	Chevallier and Duchesne (1851)[1]
1840	Colombo, Sri Lanka	28	18	Spp?	Tennent (1861)[1]
1888	Sri Lanka	12	12	Hawksbill	Deraniyagala (1939)[1]
1912	Queensland, Australia	1	0	Hawksbill	Banfield (1913)[1]
1917	Philippines	33	14	Spp?	Taylor (1921)[1]
1921	Sri Lanka	24	7	Hawksbill	Loveridge (1945)[1]
1933	Netherland Indies	2	1	Spp?	Kariadi (1933)[1]
1935	New Guinea	100	9	Hawksbill	Bierdrager (1936)[1]
1935	West Java	4	1	Spp?	Siegenbeck van Heukelom (1936)[1]
1939	Taiwan	57	7	Spp?	Kinugasa and Suzuki (1940)[1]
1949	Gilbert Islands	"a group"	5	Hawksbill	Cooper (1964)[1]
1954	Philippines	14	14	Hawksbill	Ronquillo and Caces-Borja (1968)
1954	New Guinea	6	2	Hawksbill	Romeyn and Haneveld(1956)[1]
1956	Solomon Islands	2 +	2 +	Hawksbill	Vaughan (1981)
1961	Kerala, India	130	18	Hawksbill	Pillai et al. (1962)[1]
1965	Papua New Guinea	5	5	Spp?	Likeman (1975)
1966-68	Japan	4	0	Hawksbill	Hashimoto et al. (1969)
1974	Papua New Guinea	21	3	Hawksbill	Likeman (1975)
		450 +	118 +		

[1] cited in Halstead (1978)

APPENDIX 3

POISONOUS TURTLE MEAT

Many Seychellois refuse to eat the meat of hawksbill turtles, claiming that it is sometimes poisonous. Other people discount this belief as unfounded. In fact, however, in recent history there are many documented cases of poisoning caused by hawksbill meat (Table 12). Throughout the tropical regions of the world where hawksbills occur, their meat is commonly shunned by the indigenous peoples. The highest recorded incidence of poisoning seems to occur in the Indo-Pacific region (Table 12).

Hawksbill turtles whose meat is poisonous are encountered on only the rarest of occassions. Nevertheless, the symptoms that occur when such meat is consumed are virulent, and death has resulted in about 28% of the cases reported (Table 13).

U.S. DEPARTMENT OF COMMERCE
National Oceanic and Atmospheric Administration
NATIONAL MARINE FISHERIES SERVICE
Southwest Fisheries Center
Honolulu Laboratory
P. O. Box 3830
Honolulu, Hawaii 96812

October 26, 1983 F/SWC2:GHB

Mr. Lata 'i Muli
Medical Assistant
Nomuka
Ha'apai
Tonga

Dear Mr. Muli:

Mr. Tevita Lātū recently sent me information on the hawksbill poisoning that occurred in March of this year at Nomuka Island. Your name was listed as the medical officer that attended the case. I am therefore writing to you in an effort to obtain some additional facts. It would be greatly appreciated if you would answer the following questions to the best of your knowledge.

1. Has hawksbill poisoning ever occurred before at Nomuka Island, or anywhere else in Tonga?

2. How many people total ate the hawksbill? I understand that 18 people became very sick and 2 died. Were there people that ate portions of the turtle but did not become sick? Could the sickness or deaths be attributed to eating a particular part of the turtle, such as the viscera?

3. What were the approximate ages of the two people that died? Were they male or female?

4. What were the symptoms of the poisoning?

5. How soon after eating the hawksbill did the people become sick? How soon did the deaths occur?

6. How was the hawksbill cooked?

7. Approximately how many hawksbills, and other turtles, are captured and eaten each year by the people of Nomuka Island?

8. Do hawksbills, or any other sea turtles, lay their eggs at Nomuka?

Thank you for any help that you can give to this inquiry. I have enclosed a color poster showing the different kinds of sea turtles. This may be of use to you in your work. I look forward to hearing from you at your earliest convenience.

Sincerely,

George H. Balazs

George H. Balazs
Wildlife Biologist

becoming increasingly big business. Shell ornaments are popular, especially among people of Bengali origin.

Near the jetty at Katchal island where I utilized the stopover period of a inter-island ferryboat to take a quick plunge in the sea, I excitedly beheld the first colony of garden eels (troglodyte eels) that I had seen, on a sloping sandy bed in about 15 ft of water. As I approached them, they simultaneously retreated tail-first into their sandy burrows, swaying like stalks of vegetation in the gentle swell. Dr. Hans Kaus has recorded the presence of troglodyte eels from deeper water off Great Nicobar island, but whether or not these were of the same species as the ones I saw, I am unable to confirm.

Close to the town of Wandoor in South Andaman, the intertidal fauna is particularly rich. Large chitons cling to spray-moistened rocks; sea cucumbers of at least five species are found in the shallows. A small pale white octopus crawled over rocks exposed by the tide.

South of the hamlet of Pulo Babi on Great Nicobar island, I twice observed avian predators — perhaps Nicobar Serpent Eagles — snatch up octopi from a reef exposed at low tide. In one instance the bird was forced to drop its prey after partaking of a bite or two, because of the mollusc's weight. Despite having a chunk missing from its mantle, I found the octopus to be alive and active after its fall.

I was fortunate enough to see civet cats (Paradoxurus tytleri) on two occasions: Once at day break on uninhabited Tarmugli island at a distance of ten feet as it leisurely climbed to the top of a tall tree, and another individual at dusk as it searched for titbits among crevices in the exposed reef on Rutland Island, much as I had observed wild pig do in Little Andaman. On both occasions the civets displayed a degree of apparent unconcern about the proximity of a human being that was startling to me.

It is to be hoped that the rapidly expanding population in the Andamans and Nicobars and the influx of refugees and settlers, with the resultant need for living space and resources like timber, will not result in the undermining of its irreplaceable forest wealth or cause the disappearance of the the surviving negrito tribes and of their culture.
Satish Bhaskar.

TURTLE MEAT KILLS THREE

Tuticorin, June 17, 1980 (UNI)

Turtle meat took three lives — one directly and two indirectly — today. Two suckling infants aged six months and one year died after their mothers had taken turtle meat, and a seven-year old girl who took the meat also died today, official sources said.

Seventy nine people (all fisherfolk) including several women of the fisherman colony of Tresputam who had taken turtle meat on Sunday (June 15, 1980) were treated at the Government headquarters hospital here. 57 were treated as out-patients and 22 were still in hospital.

From the 'Indian Express' Wednesday, June 18, 1980.

It appears likely that the turtle in question was a Hawksbill seaturtle (Eretmochelys imbricata), a species which has been indicated recurrently as causing deaths in India and Sri Lanka. The following instances have been recorded:

On 6th and 7th August 1977, nine persons — two adults and seven children of varying ages — died in the village of Manappad, southern Tamil Nadu from eating, on 3rd August, the meat of a seaturtle whose head was described as being somewhat aquiline, and as resembling a parrots beak. The seaturtle was also known locally as "Natchely Ammai" which means "turtle with a mouse-like head" and had a yellow plastron whereas that of the seaturtle species that was usually consumed (in all likelihood, the green turtle, (Chelonia mydas) was always white. On this occasion, some of the fishermen's advice against consumption of the meat, on the basis of it being of an occasionally poisonous variety, went unheeded. In 1970, deaths occurred at the village of Periathalai, 7 miles from Manappad, from the consumption of turtle meat.

In 1972, about 20 persons died in Thazai village from Hawksbill meat poisoning. (Valliappan and Pushparaj, 1973).

Deraniyagala (1953) cites instances of deaths in Sri Lanka in June 1921 at Mandaitivu (24 persons) and on December 3, 1941 at Habaraduva "Its toxicity is though to be due to the diet of the animal at the time; accordingly fishermen chop its liver and throw it to the crows before cooking its flesh. If the crows refuse it, the animal is discarded. Another test is to mix the raw flesh with slaked lime which turns greenish if the flesh is poisonous".

Valliappan and Pushparaj cite additional tests hat some Tuticorin fishermen employ: the turtles lood drips off quickly if the meat is nonpoisonous nd thickens on the knife blade if poisonous. A drop f blood on the skin itches and the spot becomes nflamed if the meat is poisonous.

Among symptoms of Hawksbill meat poisoning are:

eurological symptoms like vertigo, twitching of the uscles leading to convulsions, coma and finally death. ceration throughout the buccal cavity, severe itching nsation in and sloughing of the upper layers of the ngue. A sensation of obstruction in the chest, spiratory failure followed by cardiac failure.

In the absence of knowledge of the exact type of poison involved, patients were given high doses of tetracycline, massive doses of vitamin C and corticosteroids and were put on plenty of fluids and diuretics. Where treatment was started before the collapsing stage, cases responded very satisfactorily to the administration of 'Siquil' as an antiemetic, "Anthisan" tablets for food allergy and "Terramycin" injection for the infection. In all cases where death occurred, one to four days elapsed from the time the meat was consumed.

The above data were kindly supplied by Berchmann Moraes and Dr. B.V. Balaji of Manappad, and by Drs. S.C. Thanupillai, G.C.I.M. and Dr. Ramasubramaniam of Udangudi.

S.B.

* * *

MUGGER (Crocodylus palustris) RELEASES IN ANDHRA PRADESH & TAMIL NADU

Andhra Pradesh

On 7th April 1980, the Andhra Pradesh Crocodile Conservation project released 33 mugger crocodiles (11 males and 22 females hatched in June 1977) into the Kinnerasani reservoir situated within the Kinnerasani Wildlife Sanctuary. This sanctuary is located 300 km north-east of Hyderabad. The released crocodiles all ranged from 1 to 1.3m in size. Follow up monitoring survey of the released crocodiles was carried out in August 1980. Some have shown a upstream movement of over 15 km during this monsoon time.

During previous surveys in this reservoir only a few (less than five) resident muggers were reported. No breeding has taken place in past years. Since, the released muggers are all of Gir (Gujarat) origin and are a very slow growing strain (1.2m in three years!) it was decided not to mix them up with the resident Andhra Pradesh wild breeding stock occuring in the Krishna and Godavari rivers and some other tributary rivers. The remaining 58 Gir muggers of 1977 origin are being released in Pakhal Wildlife sanctuary and again in Kinnerasani sanctuary. These releases are planned for the coming winter (November 1980 to February 1981).

Tamil Nadu

The second large scale mugger release by the Tamil Nadu Crocodile Conservation Project was carried out at Hoggenakal in May 1980. (The first release was

HAMADRYAD: NEWSLETTER OF THE MADRAS SNAKE PARK TRUST

6:No.1 January 1981

Editor's Note

During the course of a survey or study there are invariably details of interest that are not compatible with tables and charts, graphs and scientific terminology. A wild scramble in the mud after a crocodile results in an erudite paper on captive growth, and a cobra who scared the pants off you becomes The Incidence of Parasites in South-east Indian Elapids. This is of-course most unfair, so we asked Satish Bhaskar to put together some homely dope on his 8 month Andaman-Nicobar trip last year. This the poor man has done. Satish is the author of "The Status of Sea Turtles in the Eastern Indian Ocean", presented at the International Sea Turtle Symposium in Washington D.C. in November '79.

We spent five weeks in the States last year, visiting various crocodilian "facilities", as the Americans call them. Our three days at Rockefeller Refuge with Ted Joanen were fascinating. We flew over the Refuge for 3½ hours in a helicopter with Ted, doing a fair bit of hovering and circling. Fortunately our great respect for him prevented us from being sick. In the Everglades with Jim Kushlan's team we watched an alligator being radio tracked and a pair of surprised eyes pop up beside the airboat. It was the first time we had seen telemetry in action and felt like country cousins come to town.

There was a large gathering of crocodile biologists at the meetings in Milwaukee (SSAR) and Gainesville (IUCN Crocodile Group). Our last stop was in South Carolina with Heyward Clamp, a professional snake hunter and an encyclopedia on south-eastern snakes. One of Heyward's methods of catching diamondbacks is to scatter tin sheeting around the countryside. These are solemnly turned over every few weeks, the ritual being called "checking the tin". We drove with him to Georgia (his snake hooks carefully fitted into the pickup truck's rifle rack) and met the locally famous Okefenokee Joe, an ex country-singer who now works for the Okefenokee Park and is also an avid snake collector. There was an earnest snake hunt the next day which I passed up; but did manage to disgrace myself by sitting a few feet away from a pigmy rattlesnake with Joe's wife, and failing to see or catch it.

Satish Bhaskar is now involved in a comprehensive survey of sea turtle nesting areas in India and is currently in the Andaman Islands (again)