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Captain Cook on poison fish

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Abstract—On his second voyage of discovery, Captain James Cook charted much of the South Pacific. The journey was long, from 1772 to 1775. During the exploration, the geographic, ethnographic, and scientific variety provided no shortage of work for the accompanying naturalists, astronomers, navigators, and painters. Culinary discoveries included new species of fish, many of which were sketched, dressed, and ultimately eaten. The examined journals and correspondence document clinical poisonings after ingestion of two different species of fish. The clinical findings are described and likely represent ciguatera and tetrodotoxin poisonings. Mechanisms of these toxin's actions are discussed in light of more recent studies.

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In 1772 the British Admiralty ordered Captain James Cook to find lands thought to be south of Australia. Cook was qualified: Two years prior, he claimed New South Wales, and having charted much of Eastern Australia, New Zealand, and Tahiti, Cook was familiar with South Pacific conditions and customs.¹ Commanding *HMS Resolution*, Cook sailed further south than ever before. Ice floes and foul weather prevented the claiming or sighting Antarctica. Nevertheless, over the ensuing years (1772 to 1775), Cook mapped and described much of the South Pacific (figure 1).

The journey required constant procurement of fresh provisions, anti-scorbotics, and water.^{2,3} The crew, used to sauerkraut, oatmeal, broth, and malt, anticipated fish and fresh foods.^{1,2} Fish were caught and, if novel, described by the father and son naturalists, Johann and Georg Forster.^{4,5} In this article, accounts of fish poisoning evident from Cook's journal, Georg Foster's diary, and correspondence from John Anderson, surgeon's mate, are examined and discussed in light of current literature with an aim to make speculative diagnoses.^{1,4,6} The fish poisonings are briefly described separately.⁷⁻⁹

Bad fish: The known and the unknown. Cook and crew knew of fish ingestions that resulted in poisonings. Anderson writes of West Indian fish poisonings but failed to reference an example.⁶ Forster or Cook was Anderson's probable source: Both knew of the 1606 poisoning of De Quiros after eating Caribbean sea bream (*Pagrus*) (De Quiros describes probable ciguatera poisoning).^{1,5} Furthermore, Forster knew pufferfish might be poisonous.⁴ In Kaempfer's *History of Japan* (1727), pufferfish are

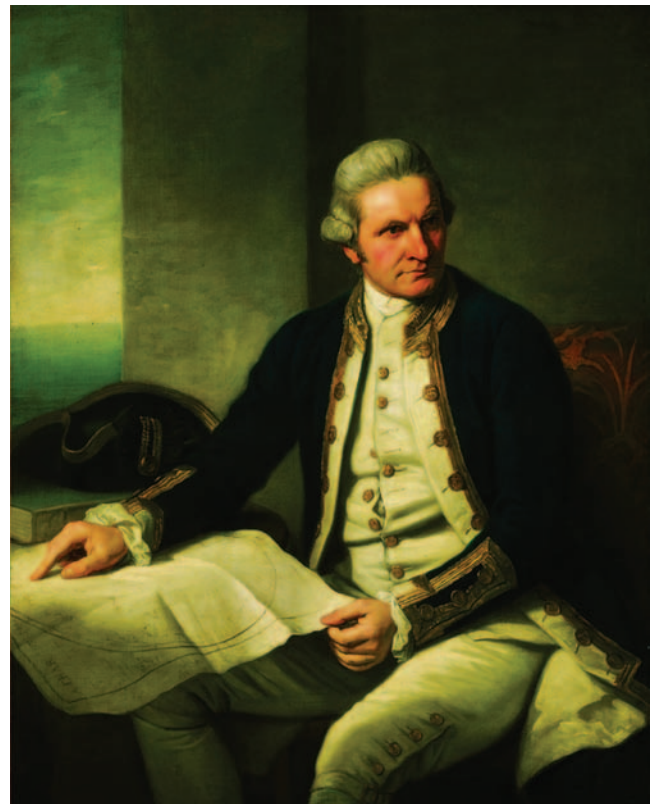


Figure 1. Captain James Cook. Portrait by Nathaniel Dance, 1775 to 1776. (Courtesy of and copyrighted by the National Maritime Museum, London, UK.)

said to be poisonous, but whether Anderson, Cook, and Forster were familiar of Kaempfer's work is not clear.⁷ Kaempfer's work did not detail clinical manifestations of pufferfish poisoning. There is little to

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suggest that Cook and his crew knew what might happen if a poisonous fish were ingested. They would soon find out.

Fresh fish. The first poisoning occurred in July 1774, while off the coast of Mallicollo (Vanuatu). The lieutenants, their messmates, several midshipmen, and the carpenter ate three freshly caught red fish that resembled *Sparus pagrus* or *Sparus erithrynus* (a bream or snapper).^{1,5,6} The accounts from Cook, Forster, and Anderson are similar. The men presented with “uneasiness” and pain in the mouth and throat. An upset stomach and nausea followed along with lassitude, retching, and weakness.^{1,5,6} Anderson describes the progression:

... a flushing heat and violent pains in the face and head, with a giddiness and increase in weakness; also a pain, or as they expressed it, a burning heat in the mouth and throat. Some had the mouth affected ... they imagined their teeth were loose ... a considerable spitting attended this symptom. The pulse ... was rather slow and low ... the pain and heat of the head extended to the arms, hands and legs ... patient(s) continued in this manner all night ... towards the morning, the pains, especially those in the legs and arms, but more particularly about the knees was severer than before. These would sometimes remit and frequently shift, or be more violent in one place than in another. Sometimes the pain would remove suddenly from the legs and fix in the head; the palms of the hands were hot; and the fingers, legs and toes, felt often as if benumbed: nay the whole limbs became in some measure paralytic, the sick person being unable to walk unless supported ... although there appeared to be no swelling in the face, it might be observed to have a sort of shining or gloss upon it; and the patient sometime imagined his nose was grown to a great size. ...⁶

Other symptoms included headache, thirst, proctalgia, and “a disagreeable sensation on rubbing his skin in any part of his body.”⁶ Animals on board that ate the fish fared poorly. “Guts and bones” fed to the dogs left some with paralytic rear legs, priapism, and “groaning.” A dog given a tobacco juice emetic died 16 hours later, as did a hog and a favorite shoulder-perching parakeet from the “Friendly Isles.”^{1,5,6} Over the next 10 days, the afflicted men recovered.¹

A solar eclipse, the butcher dies, and a fish is purchased. The second poisoning occurred 2 months later. On September 7, 1774, *HMS Resolution* was anchored off an island in New Caledonia. It was an eventful day. The butcher died after falling down one of *Resolution's* hatches. Onshore, some of the officers took measurements of a solar eclipse. Crew were busy buying food and chasing women who “[encouraged] the proposals of our seamen,” and a clerk traded a piece of Tahitian cloth for a big-headed fish killed by a “native with a spear.”^{1,4} Forster thought it a new species and sketched it (figure 2); he wrote, “It was of the genus, by Lineaus named *Tetraodon*. ... several species are reckoned poisonous.”⁴



Figure 2. *Tetraodon lagocephalus scleratus*, “Silverstripe blaasop,” Georg Foster, September 7, 1774, Poemanghee, New Caledonia. (Courtesy of and copyright by the Natural History Museum, London, UK; image digitally adjusted to enhance contrast via Photoshop 7.0 [Adobe, Seattle, WA].)

Damn the torpedos. Georg Forster warned Cook of the *Tetraodon's* poison nature; he was rebuffed: “[Cook] had eaten this identical sort of fish ... during his former voyage, without the least bad consequences.”⁴ The fish was preserved, and the liver served the following day.^{1,4,6} The butcher’s unexpected fall does not appear related to fish preparation or paralysis. The consequences were nearly disastrous. Cook writes:

Without the least suspicion of its being of a poisonous quality we had ordered it for supper ... only the Liver and Roe was dressed of which the two Mr. Fosters and my self did but just taste. About 3 or 4 o'clock in the morning we were seized with an extraordinary weakness in all our limbs attended with a numbness or sensation like to that of caused by exposing ones hands or feet to a fire after having been pinched much by frost, I almost lost the sense of feeling nor could I distinguish between light and heavy bodies, a quart pot full of water and a feather was the same in my hand. We each of us took a vomit ... [and] a sweat which gave great relief ... one of the pigs which had eat the entrails was found dead.¹

Anderson’s account differs only with regard to a dog that later died after eating a similar fish.⁶ Georg Foster describes its effects on his father, Cook and himself:

The liver of this fish was served up, which was very large and oily ... at 3 o'clock in the morning my father awaking found himself extremely giddy, and his hands and feet ... benumbed ... he was scarcely able to stand. ... [Captain Cook] found himself unable to walk without holding; I was in the same situation ... and crawled into the cabin. The blood had left our cheeks, all our limbs were benumbed, and without sensation, and a great degree of langour and oppression had taken place. ...

Emetics and sudorifics (sweat inducers) were given.

In the morning we got up, giddy and heavy; however, I found myself well enough to pass the whole morning sketching. ... Towards noon my father endeavored to converse with some of the natives who came into the ship. At sight of the fish, which was hung under the half deck, they made signs that it occasion pain in

the stomach, and by leaning their heads upon one hand, and shutting their eyes, expressed that it caused sleep and drowsiness, and death . . . we offered it to them, but they refused it with the strongest marks of aversion . . . I was severely punished . . . by a returning fit of dizziness. . . .⁴

The next day, Forster was unable to walk for more than 5 minutes at a time. He had intermittent dizziness and was “unfit for researches . . . deprived the power of thinking, judging and remembering, as well as the perfect use of our external senses.”⁴ It took the men 11 days for all but “trifling pains and a little weakness” to completely resolve.⁶

Clinical presentations: Were they different?

The *Sparus pagrus* and *Tetraodon* poisonings differ subtly. Clinically, the *Tetraodon* poisoning caused vomiting, sweats, sensory loss, dizziness, and motor paralysis. Although Forster describes a mental lassitude, more discrete CNS effects were not evident. The snapper (*Sparus pagrus*) also caused motor paralysis, yet the ingestion resulted in dysesthesias, shifting pains, spitting, numbness, tenesmus, priapism, and delirium. Current knowledge suggests involvement of central autonomic, motor, and neurotransmitter systems in both poisonings; however, the *Sparus pagrus* poisoning may have more involvement with pain systems and possibly c-fibers. The clinical presentations and fish descriptions likely implicate two different poisons.

Cook and his crew make little of the similarities or differences between the two poisonings. The fact that Cook previously ate a fish that resembled the *Tetraodon*, and that the crew consumed more *Sparus pagrus* after the poisoning suggest that although aware of the fish poisoning, the linking of specific fish species to the poisonings did not occur. Alternatively, the Islanders demonstrated *Tetraodon* should not be eaten but could be sold.

Possible toxins? The *Tetraodon* name and illustration, particularly the beak-like mouth, suggest a relative of the box, toad or pufferfish. Different bacteria living in *Tetraodon* liver, gonads, intestines, and skin are known to synthesize tetrodotoxin (TTX).¹⁰ The toxin resists heat, is not damaged by freezing, and, when ingested, acts as an extremely potent voltage-sensitive sodium channel blocker.¹⁰⁻¹³ Clinical poisonings mainly affect peripheral nerves, resulting in motor paralysis, though central pathways may also be disrupted, leading to confusion, coma, and diabetes insipidus.¹²⁻¹⁴ As pufferfish remains a sushi delicacy and occasionally an unwitting meal, clinical poisonings still occur, and with transport of fresh and frozen produce, in geographic distributions far beyond catch location.

Wandering, severe thermal, dysesthetic, and arthralgic pains, combined with a particular sense of teeth loosening hours after eating tropical reef fish like *Sparus pagrus* remains a classic presentation of ciguatera poisoning.¹⁵ Coral algae, particularly *Gam-*

bierdiscus toxicus, produce marine toxins such as ciguatoxin (CTX), maitotoxin (MTX), and scaritoxin (CTX-4A). CTX and CTX4A change voltage-sensitive sodium channels, permitting sodium influx (ironically opposite to the effect of TTX, which antagonizes sodium influx), the net effect being weakness and, in the most severe cases, paralysis. MTX and CTX4A both may increase acetylcholine and norepinephrine release.^{11,14} Forster was nearly correct in speculating that fish ingested poison vegetables.⁵ In fact, pelagic reef dwellers, snapper included, eat small algae-eating fish, effectively bioconcentrating these coral algae toxins. These toxins, like TTX, resist breakdown with heat or freezing.¹⁴

Ciguatera poisoning occurs with small concentrations and combinations of multiple toxins. In Vanuatu, July remains the most likely month to develop ciguatera.¹⁶ Anderson noted similar fish caught and eaten a month after their poisoning, in August, resulted in no ill effects. Unlike TTX, ciguatera toxins are known fish contaminants with possible seasonal and geographic variability. However, as for pufferfish, CTX-containing fish can be exported to distant locations and lead to clinical poisonings.

The accounts of fish poisoning generated from the *Resolution's* voyage are uniquely detailed and were widely published in Europe on their return. Their historical impact is probably marginal, antiquarian trivia at best, at worse fuel for speculation of “what if Cook ate more of the liver?” However, scientifically they represent good case narratives, communicated to the Royal Society and published a year after their voyage concluded.

Describing phenomena is often the first step in a scientific endeavor. Since Cook's voyage, our accumulated knowledge of fish poisonings suggests Cook and his crew had distinct ciguatera and TTX poisonings. Their clinical descriptions from 1774 are little changed when compared with contemporary presentations; the key historical detail remains a recent ingestion of fish. Perhaps more interesting for the neurologist is that Cook and his crew likely had toxins that target the same voltage-sensitive sodium channels but result in antagonistic functions: TTX blocks sodium influx, CTX promotes it. Whether this subtle difference in molecular targeting explains the different clinical presentations is unclear, particularly as clinical ciguatera poisoning may involve differing toxins.

Cook's second voyage was a triumph of navigation and health. During the 3-year voyage, Cook lost four men: Two drowned, one fell, and the fourth had a chronic illness.² Not one died of scurvy or fish poisoning, despite likely TTX and ciguatera poisonings. Neurologists should consider fish poisonings when a history of recent fish consumption and progressive motor and neurologic compromise are seen.

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