Health risks associated with the consumption of sea turtles: A review of chelonitoxism incidents and the presumed responsible phycotoxins

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Abstract

Consuming the meat of some marine turtles can lead to a specific type of seafood poisoning known as chelonitoxism. A recent poisoning event (March 2024) on the Tanzanian island Pemba, resulting in the death of 9 people and hospitalisation of 78 others, underscores the need to obtain an up to date overview and understanding of chelonitoxism. Here, we document a global overview of poisoning incidents resulting from the consumption of sea turtle flesh worldwide. All events combined involved over 2400 victims and 420 fatalities. Incidents were predominantly reported in remote regions (often islands) across the Indo-Pacific region. Reported health effects of consuming poisonous sea turtles include epigastric pain, diarrhea, vomiting, a burning mouth and throat sensation, and dehydration. In addition, ulcerative oeso-gastro-duodenal lesions, which occasionally have resulted in hospitalization and death, have been reported. Lyngbyatoxins have been suggested as (one of) the causative agents, originating from the cyanobacterium *Moorena producens*, growing epiphytically on the seagrass and seaweed consumed by green turtles. However, due to the limited evidence of their involvement, the actual etiology of chelonitoxism remains unresolved and other compounds may be responsible. The data outlined in this review

offer valuable insights to both regulatory bodies and the general public regarding the potential risks linked to consuming sea turtles.

Keywords: food poisoning; chelonitoxicity; cyanobacteria; lyngbyatoxin; bioaccumulation; ecotoxicity

1. Introduction

Sea turtles, intriguing to both scientists and the general public, play vital roles in marine ecosystems, serving as key predators, nutrient suppliers, hosts for parasites, and habitat landscapers, potentially engineering entire ecosystems (Bjorndal and Jackson, 2002; Heithaus, 2013; Lovich et al., 2018; Teelucksingh et al., 2010). Sea turtles play a crucial role as biological transporters, introducing marine nutrients and energy to nutrient-deficient coastal ecosystems, including islands (Bouchard and Bjorndal, 2000). Despite their significance, all of the world's seven sea turtle species are threatened, with 85.7% of sea turtle populations worldwide facing the threat of extinction (IUCN, 2024; Rothamel et al., 2021).

Marine turtles have been subject to exploitation for their meat, eggs, shells, skins, and internal organs, for at least 13,000 years (Des Lauriers, 2006). They commonly nest on sandy beaches, making nesting females and their eggs relatively easy targets for humans and predators (National Research Council, 1990). Additionally, turtles are frequently caught at sea as well, sometimes unintentionally, for example by getting caught in fishing gear. Throughout history, they have held significant importance as a resource for coastal communities around the globe. Estimates indicate that approximately 9 million hawksbill turtles (*Eretmochelys imbricata*), or an average of 60,000 turtles annually, were traded globally for their shells over a span of approximately 150 years (1844–1992) (Miller et al., 2019). However, the unsustainable consumption of sea turtles has

47 contributed to declining populations and the deterioration of the marine environments they inhabit.
48 Despite regulations in several nations around the globe aimed at limiting the capture of sea turtles,
49 these species continue to represent a crucial resource for some communities (Campbell, 2003;
50 Mack et al., 1982; Poti et al., 2021). Reports of ongoing sea turtle consumption (legal and illegal,
51 or effectively unregulated) have emerged from many parts of the world. In 2013, 42 countries and
52 territories still permitted direct take of turtles, resulting in the legal capture of more than 2 million
53 turtles since 1980, however levels had reduced to 60% compared to those in the 1980s (Humber
54 et al., 2014). However, Senko et al. (2022) estimated over 1.1 million marine turtles to be illegally
55 exploited between 1990 and 2020 in 65 countries or territories worldwide, despite existing laws
56 prohibiting their use, with over 44,000 turtles exploited annually over the past decade. In fact, sea
57 turtle consumption is increasing in parts of the southwest Indian Ocean (Rothamel et al., 2021).
58 For example, over 80% of rural households along Madagascar's eastern coastline were estimated
59 to consume an average ± SD of 1.47 ± 0.99 kg of sea turtle meat per year over the past decade
60 (Rothamel et al., 2021). Although turtle hunting in Madagascar is illegal under national law, there
61 are currently no government initiatives to manage this type of fishery (Humber et al., 2010). Turtle
62 fisheries continue to be an important source of finance, protein and cultural identity in these parts
63 of the world (Humber et al., 2014).

64 Coastal communities that consume sea turtles typically make use of the entire animal. Turtle meat
65 is consumed directly, while internal organs such as the kidney and liver are utilized for soup (Mack
66 et al., 1982). Aguirre et al. (2006) reported the fat to be extracted for oil, used as a remedy for
67 respiratory issues, particularly in children, and the blood to be consumed raw to address anemia
68 and asthma. Furthermore, sea turtle eggs are highly prized as an aphrodisiac (Spotila, 2004). Yet,
69 consuming sea turtle meat poses significant risks to both turtle conservation efforts and the health

of local communities. For example, consuming sea turtles can pose health risks due to the potential presence of zoonotic bacteria, such as *Salmonella* spp. (e.g. Aguirre et al., 2006; Draper et al., 2017; O'Grady and Krause, 1999). Additionally, environmental contaminants such as organochlorines and heavy metals, which persist and accumulate in marine ecosystems, may transfer to humans through the consumption of contaminated seafood, including the meat of sea turtles (Aguirre et al., 2006). These compounds tend to reach particularly high concentrations in long-lived organisms like sea turtles, exceeding international food safety standards and posing potential health hazards to those who consume them (Aguirre et al., 2006). Poisoning following ingestion of the flesh of sea turtles, medically known as chelonitoxism or chelonitoxicity, is uncommon but seen especially in coastal areas (Fussy et al., 2007; Yasumoto, 1998). Several incidents of chelonitoxism poisoning have been reported over the last decades, with the last report only dating from March 2024. Insufficient understanding of the poisoning frequently results in misdiagnosis and treatment based solely on symptoms (Gatti et al., 2008). Thus, it is crucial to raise awareness of chelonitoxism, particularly in non-endemic regions. In this review, we provide an exhaustive list of reports listing poisoning, resulting from consuming sea turtles, and discuss potential symptoms as well as the occurrence of poisoning symptoms. Furthermore, we discuss the origin behind the poisoning from an ecological and ecotoxicological point of view.

2. Material and methodology

2.1 Literature search

We queried Web of Science using all possible combinations of the search terms “chelonitoxin”, “chelonitoxism”, “chelonitoxicity”, “poisoning”, “turtle”, “seafood consumption”, from 11th of March to 20th of March 2024. We used the search criteria “all topics” to search Web of Science, returning in total 11 relevant publications. We also used literature that was cited in the acquired

articles for appropriate publications (25 additional articles collected), as well as screened for potential reports in the grey literature. The applied search criteria yielded a total assembly of 36 articles, which we screened for the following information: study location, year of poisoning, sea turtle species consumed, inflicting taxa, number of victims, poisoning symptoms, attack rates, incubation period, recovery period, potential remediation strategies, the identity of the responsible chemicals, and the used methodology to isolate and identify the chemical compound.

2.2 Data analysis

All retrieved studies were collected in one table (Table S1) and information criteria were summarised. Graphs were constructed using the ggplot2 package (v.3.4.0) in R v.4.3.0.

3. Results and discussion

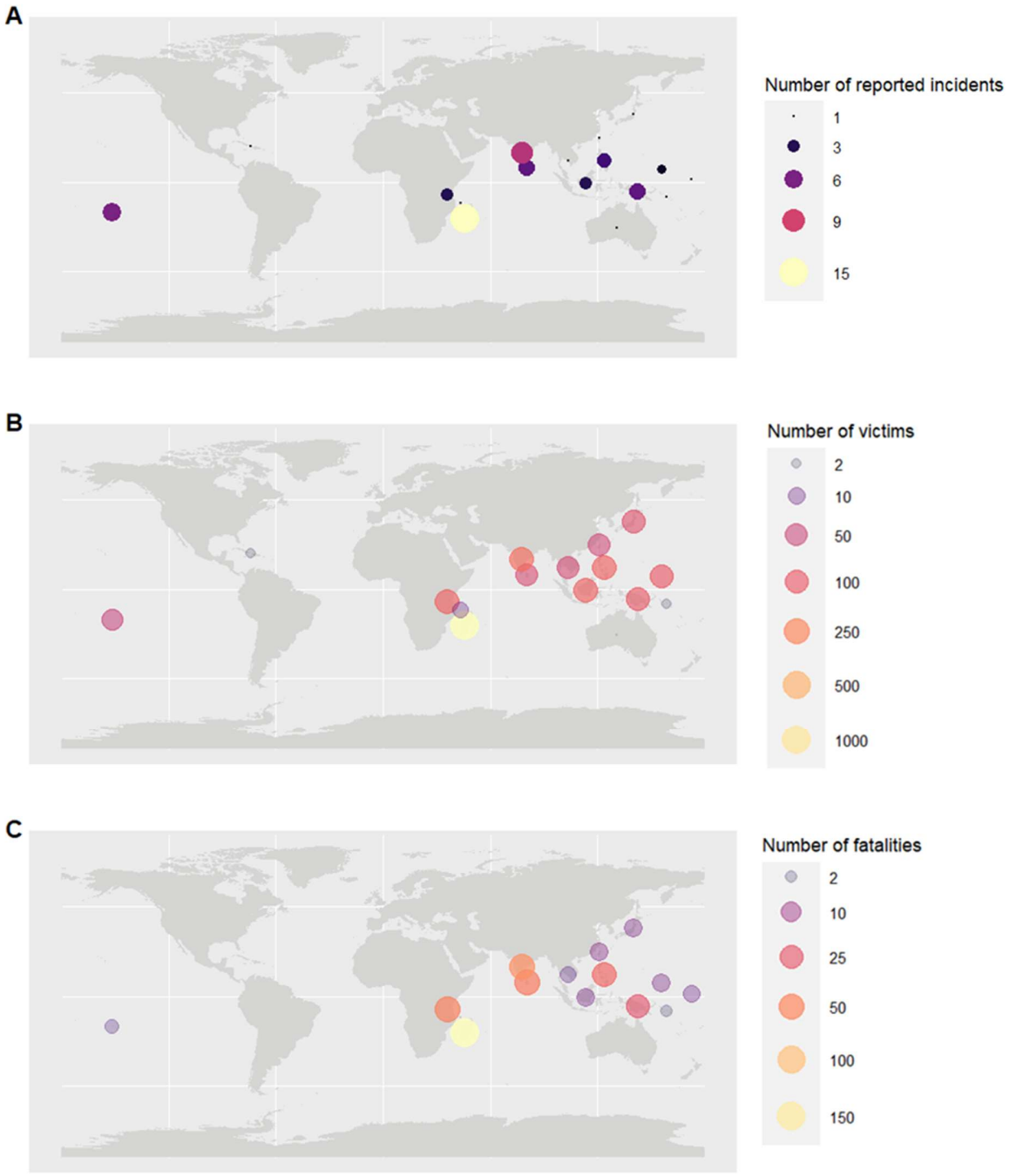
3.1 Historic overview of incidents involving chelonitoxism

Chelonitoxism is less common (and less reported) compared to other forms of seafood poisoning like ciguatera or scombrotoxism, which have received more attention (Brodin, 1992; Fussy et al., 2007). Nevertheless, sea turtle poisoning has been acknowledged for centuries, with cases reported by Europeans dating as far back as the 17th century (Chevallier and Duchesne, 1851). Here, we list 62 reported incidents of chelonitoxism poisoning, following ingestion of the flesh of sea turtles in the Caribbean, and Indian and Pacific Ocean, involving a minimum of 2424 victims presenting illnesses and 420 human fatalities. This type of poisoning has primarily been observed across the Indo-Pacific region, spanning areas such as Zanzibar, Madagascar, India, Japan, Sri Lanka, Taiwan, the Philippines, Indonesia, Papua New Guinea, Australia, the Comoros islands, Kiribati, and Fiji (Suppl. Table 2). Conversely, cases of poisoning from turtle consumption are notably rare/absent in the Caribbean and broader Atlantic region where the turtle species associated with

115 chelonitoxism also live and are consumed (Figure 1). The rarity of chelonitoxism, in comparison
116 with other types of seafood poisoning, is potentially connected to the endangered status of sea
117 turtles, safeguarded by global regulations, or because of religious restrictions in terms of
118 consumption, with sea turtles being revered as taboo species in various ethnic communities
119 (Champetier de Ribes et al., 1998). However, in many cultures, such as within Pacific islands,
120 consumption of sea turtles has or had no such stigma (Adams, 2003; Humber et al., 2014). The
121 reported instances of turtle poisoning have predominantly surfaced within more isolated and
122 impoverished coastal communities (Suppl. Table 2), where a captured sea turtle can serve as a
123 significant food source. In such scenarios, mass poisonings are not uncommon, as the reptile's flesh
124 is often distributed among entire families or villages during communal gatherings (Pavlin et al.,
125 2015). Nevertheless, the number of turtle poisoning cases is likely much higher than reported in
126 this study, but as consumption of sea turtles is illegal in several countries and territories, most cases
127 are rarely reported. Another contributing factor to the scarcity of reports on sea turtle poisoning is
128 that affected individuals often reside in remote geographical areas, mostly islands without any
129 access to healthcare facilities or diagnostic capacity (e.g., Pillai et al., 1962; Ranaivoson et al.,
130 1994; Robinson et al., 1999). Attack rates are few reported in literature. An attack rate is defined
131 here as the proportion of an at-risk population that contracts the poisoning (symptoms) during a
132 specified time interval. Notable exceptions are: 18 % (6/33 consumers) by Deveraturda et al.
133 (2015), 48 % (32/66) by Robinson et al. (1999) and 84% (101/120) in Pavlin et al. (2015).

134 The ingestion of toxic turtle tissue, blood or water used to clean/cook the turtle can also prove fatal
135 to domestic animals like cats, dogs and goats (Pavlin et al., 2015; Ronquillo and Caces Borja,
136 1968; Silas and Fernando, 1984; Singh et al., 2016). In some cases, alongside adult turtles, eggs
137 were also reported to be consumed (Ronquillo and Caces Borja, 1968). Reports, such as Likeman's

(1975) account of a child's death after consuming "unlaid eggs" of a hawksbill turtle in Papua New Guinea, and anecdotes from a Vietnamese fisher describing multiple deaths following hawksbill turtle egg consumption (Aguirre et al., 2006), underscore the hazard. Most turtles are not poisonous, and, despite some attempts, identifying poisonous turtle flesh is impossible without performing chemical analyses. According to Deraniyagala (1939), experienced fishermen used to chop off the liver of *Eretmochelys imbricata* and fed it to crows. If the birds discarded the liver, the animal was considered poisonous. In New Guinea, feeding of turtle meat to dogs and cats for adverse reactions has been reported as well (Bierdrager, 1936).



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148 Figure 1. Geographic distribution of (A) reported chelonitoxism incidents with (B) associated number of poisoned
149 victims (displaying poisoning symptoms) and (C) associated fatalities. The size and color of each bubble indicates the
150 value of the particular variable, with the position on the map indicating location. Date and location of incidents are
151 reported in Supplementary table S2.

152

3.2 Involved sea turtle species and patterns of sea turtle exploitation

Among the few reported cases, most are associated with the ingestion of hawksbills (*E. imbricata*) turtles, while green turtles (*Chelonia mydas*) are implicated less frequently (Suppl. Table 2). In 59 % of the chelonitoxic incidents (n=36 incidents), a hawksbill turtle was implicated as the responsible agent, while green turtles were reported to be involved in 23 % of the cases (n=14), or the identity of the inflicting species was unknown or not reported (n=10) in 16 % of the cases (Figure 2). Additionally, there have been unconfirmed instances of poisoning following the consumption of loggerhead (*Caretta caretta*), flatback (*Natator depressus*), olive ridley (*Lepidochelys olivacea*), and leatherback (*Dermochelys coriacea*) turtles in peer reviewed literature (Brodin, 1992; Limpus, 1987). The poisonous potential of these species can be put into question due to the absence of any observations in the last three decades.

These figures can be partially explained by sea turtle consumption patterns reported in other studies. Humber et al. (2014) found that >95% of legal marine turtle exploitation was comprised of green (89%) and hawksbill turtles (8%). Similarly, in the analysis of Senko et al. (2022), green turtles (56%) were the most illegally exploited species between 1990-2020 globally, followed by hawksbills (39%), and loggerheads (3%). Despite hawksbills being consumed by humans less frequently over time, the number of chelonitoxic incidents involving hawksbills is more than double compared to those involving green turtles, regardless of the period in which the incident took place. Due to small size of the collected dataset, we cannot identify any trends statistically in chelonitoxic incidents related to the consumption of a specific species.

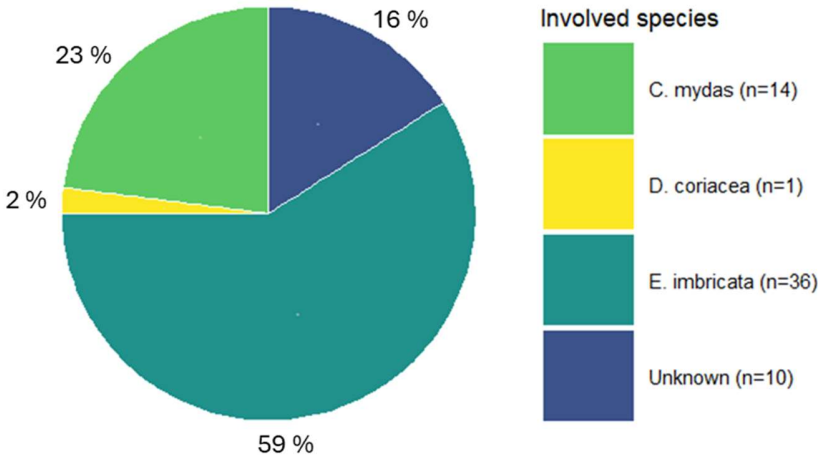

Figure 2. Pie chart illustrating the involvement of sea turtle species in chelonitoxism incidents, relative to total number of incidents.

When all data sources are taken into account, nearly 70% of all reported chelonitoxicity incidents occurred in only five countries: Madagascar (25.4%), India (13.6%), French Polynesia (10.2%), Sri Lanka, and Papua New Guinea (each 8.5%; Figure 1; Table S2). Neither fisheries yields, nor the presence or absence of national legislation, seem to influence the incidence of chelonitoxicity. For example, nearly 75% of illegal exploitation (i.e., number of turtles exploited) between 1990 and 2020 occurred in five countries: Haiti (31%), Tanzania (20%), Honduras (10%), Indonesia (7%), and Mexico (6%), according to Senko et al. (2022). The two most recent poisoning incidents (Pemba Island, Tanzania in 2021 and 2024) likely resulted from an illegally captured sea turtle (Table S2). The permitted take, estimated between 1980 and 2013, is also found to be concentrated in three countries: Papua New Guinea (15,217 turtles $year^{-1}$; 36.1%), Nicaragua (9413 turtles $year^{-1}$; 22.3%) and Australia (6638 turtles $year^{-1}$; 15.7%), accounting for almost three-quarters of

187 legal exploitation efforts in this period (Humber et al., 2014). Hence, these findings suggest that
188 the incidence of chelonitoxism is not influenced by fisheries yields or the presence or absence of
189 national legislation. However, as few incidents are reported and/or documented, this observation
190 could be an artifact caused by the limited data availability.

191 **3.3 Poisoning symptoms, incubation time, and treatment**

192 Detailed clinical descriptions of patients diagnosed with chelonitoxicity have been infrequent, with
193 most information being anecdotal, relying on testimonials – sometimes conflicting – gathered from
194 local fishermen's accounts (Brodin, 1992; Fussy et al., 2007). The reporting authors may have
195 either overestimated or underestimated the total number of cases. First, it is important to
196 acknowledge that the consumption of sea turtles is illegal in several countries, and the fear of legal
197 repercussions may discourage some victims from seeking medical assistance, limiting the
198 reliability of the data collected. Second, the number of suspected cases might exaggerate the scale
199 of an outbreak if individuals without symptoms seek medical attention due to concerns arising
200 from reports of a chelonitoxic outbreak and neighboring deaths. Another contributing factor to the
201 scarcity of reliable data on sea turtle poisoning is that affected individuals often reside in remote
202 geographical areas lacking access to healthcare facilities (Champetier de Ribes et al., 1997;
203 Ranaivoson et al., 1994; Richmond, 2011; Robinson et al., 1999).

204 Nevertheless, the clinical manifestations of chelonitoxism are notably distinct from other types of
205 seafood poisonings, demonstrating a relatively consistent pattern (Brodin, 1992; Fussy et al., 2007;
206 Isbister and Kiernan, 2005). However, Mbaé et al. (2016) suggest a strong similarity between the
207 symptoms and incubation period of consuming fish containing toxins, such as Ciguatera fish
208 poisoning. In human cases, symptoms of sea turtle poisoning primarily affect the upper digestive

tract, manifesting as nausea, vomiting, epigastric pain, and occasionally diarrhea (Suppl. Table 2, Table 2). Additionally, affected individuals may experience general symptoms such as dizziness, malaise, and sweating. Typically, recovery occurs within a week without further complications (Bagnis and Bourligueux, 1972). Nevertheless, severe cases may progress to glossitis, dysphagia, drowsiness, and multiorgan failure, characterized by tubular nephropathy, liver cytolysis, and respiratory distress, ultimately leading to coma and high mortality rates, or neurological complications in survivors (Aguirre et al., 2006).

Fussy et al. (2007) delineate three grades of chelonitoxism based on symptom severity. Grade I entails gastrointestinal signs (e.g., vomiting, epigastric pain and, occasionally, diarrhea, and mouth pain). Other symptoms possibly occurring at onset include vertigo (dizziness), malaise, sweating, sore throat and chest pain. Most patients do not develop further symptoms. However, some patients reach grade II (moderate poisoning), in which pathognomonic oral and pharyngeal involvement occurs, accompanied by a burning mouth and throat sensation, and alongside dysphagia, excessive salivation (hypersialorrhea) and glossitis (Fussy et al., 2007; Kinugasa and Suzuki, 1940; Romeyn and Haneveld, 1956). Ulcerative oeso-gastro-duodenal lesions have also been reported (e.g. Silas and Fernando, 1984; Singh et al., 2016). Being the only report to investigate physical symptoms of chelonitoxism in domestic animals, Silas and Fernando (1984) reported on a dog with ulceration in the mouth, which was unable to consume food after ingestion of some of the cooked turtle flesh leftovers. During this phase (grade II) , neurological symptoms, which serve as the most reliable indicators of severity, may manifest with alternating episodes of drowsiness and full consciousness (or psychomotor agitation). Grade III symptoms encompasses coma and multiorgan failure (tubular nephropathy, liver cytolysis, respiratory distress). Mortality is prevalent among individuals who reach this grade, particularly among children (Suppl. Table 2). Case fatality rates

(CFR, i.e., proportion of people who die from a specified disease among all individuals diagnosed), if reported, differ drastically among the studies. For example, Ventura et al. (2015) and Pavlin et al. (2015) report a low CFR value of 6%, while Taylor (1921) reported a CFR of 42 % with 14 deaths of 33 reported cases of chelonitoxism. Survivors who recover from comatose states frequently exhibit intricate central and/or peripheral neurological sequelae (such as hemiplegia, tetraplegia, dementia, sensory-motor deficits, or cerebellar syndrome; Fussy et al., 2007). However, most studies report the observed symptoms anecdotally, lacking quantitative data on incidence, though there are a few exceptions, as detailed in Table 1.

Table 1. Occurrence of symptoms among probable cases in several described cases of food-poisoning after sea turtle consumption. Number of patients with the respective symptom are reported in the table, with percentages noted between brackets. NR = not reported; * Number of respondents varies as not all patients responded to all questions (several of the patients were infants in this study).

	Madagascar, December, 1994	**Madagascar, December, 1994**	**Madagascar, October, 1995**	**Micronesia, October 2010**	**India, August 2012**	**Comoros, December 2012**	**Madagascar, May 2014**
Number of victims	N = 60	N = 32	N = 95	N = 87-94*	N = 7	N = 8	N = 76
General symptoms							
Itching	NR	NR	NR	NR	1 (14)	5 (63)	NR
Asthenia	13 (22)	7 (22)	30 (32)	NR	2 (29)	4 (50)	13 (17)
Paleness	1 (2)	3 (1)	NR	NR	NR	NR	NR
Rash	NR	NR	NR	NR	NR	2 (25)	NR
Fever	8 (13)	4 (13)	20 (21)	40 (43)	1 (14)	1 (13)	NR
Dehydration, thirst	NR	NR	NR	66 (71)	NR	NR	3 (4)
Myalgia	NR	NR	NR	NR	NR	2 (25)	NR
Digestive symptoms							
Vomiting	23 (38)	12 (38)	32 (34)	29 (31)	4 (57)	3 (38)	33 (43)
Abdominal pain	NR	NR	29 (30)	26 (28)	3 (43)	3 (38)	27 (36)
Stomatitis	20 (34)	11 (34)	11 (12)	72 (78)	NR	NR	NR
Mouth/throat burn	NR	NR	NR	47 (54)	5 (71)	1 (13)	1 (1)
Mouth ulcers	19 (32)	9 (28)	21 (22)	NR	4 (57)	NR	1 (1)
Sore throat	NR	NR	NR	76 (84)	NR	1 (13)	1 (1)
Itching of the month/throat	NR	NR	NR	NR	NR	2 (25)	9 (12)
Dysphagia	30 (50)	16 (50)	48 (51)	NR	NR	1 (13)	11 (14)
Constipation	NR	NR	NR	20 (21)	NR	NR	NR
Diarrhea	2 (4)	3 (1)	12 (13)	NR	NR	1 (13)	9 (12)

Respiratory symptoms							
Shortness of breath	NR	NR	NR	NR	NR	2 (25)	NR
Dry, tickling cough	NR	NR	NR	35 (38)	NR	NR	5 (7)
Thoracic pain	NR	NR	NR	NR	NR	NR	2 (3)
Neurological & cardiological symptoms							
Vertigo	11 (19)	6 (19)	NR	NR	4 (57)	1 (13)	12 (16)
Nausea	NR	12 (38)	NR	NR	NR	2 (25)	17 (22)
Paresthesia	19 (31)	NR	30 (32)	NR	NR	1 (13)	3 (4)
Headache, migraine	11 (19)	6 (19)	NR	NR	NR	NR	25 (33)
Somnolence/Lethargy	NR	7 (22)	20 (21)	NR	NR	NR	2 (3)
Erectile dysfunction	NR	NR	NR	NR	NR	1 (13)	NR
Tachycardia	NR	6 (19)	NR	NR	NR	NR	NR
Coma	2 (4)	NR	11 (12)	NR	1 (14)	NR	1 (1)

First symptoms are usually visible within 24 hours of consumption, and can occur up to usually 7 days after consumption, with a few exceptions known (Figure 3). The onset period is typically characterized by the appearance of grade I symptoms, i.e. the gastrointestinal signs. Fussy et al. (2007) report the appearance of digestive symptoms such as nausea and vomiting within three hours. The following day, three persons, who had experienced these symptoms, decided to eat the leftover flesh at even larger quantities than the day before, and all three developed more severe symptoms (grade II) and required medical evacuation to the hospital in Tahiti. Similarly, Ranaivoson et al. (1994) described the epidemic curve associated with the chelonitoxism incident in December 1994, Madagascar. In this case, 22 % of the people who got sick, presented first symptoms within 24 hours after consumption of the turtle, while 64.5 % of the people that consumed turtle meat showed signs of poisoning within 72 hours (Figure 3). They also described the occurrence of one case in which a person only displayed the first signs of poisoning after 20 days of consumption. Champetier de Ribes et al. (1998) described an onset of typically 12 to 24 hours after the implicated meal, ranging from 3 hours to over 10 days. Deveraturda et al. (2015) noted an incubation period ranging between one and 45 hours after consumption (median of 4

hours), while Mbaé et al. (2016) reported an average incubation period of 1.6 days (ranging from 0 to 6 days; Figure 3).

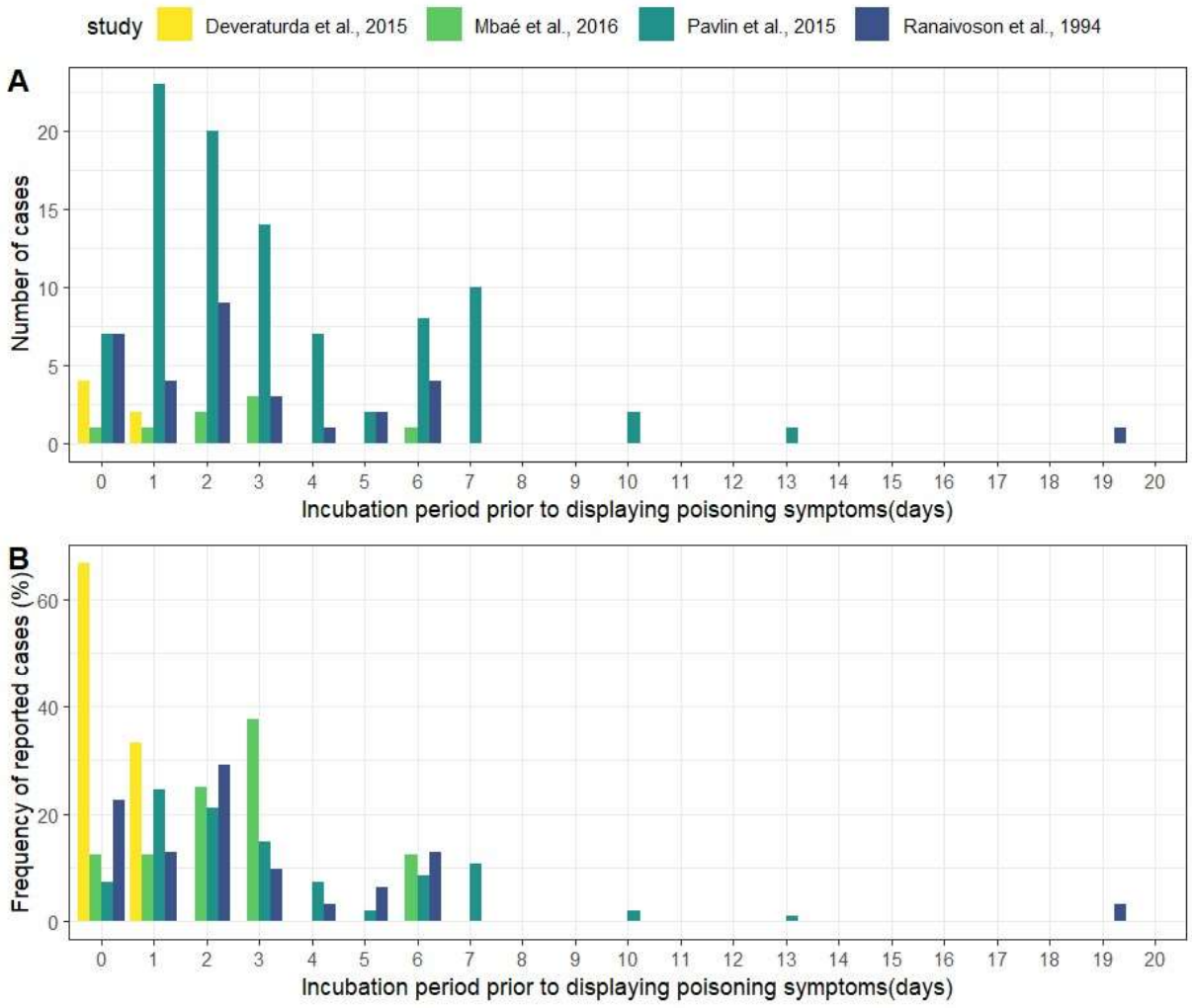

Figure 3. Date of illness onset after sea turtle consumption as reported in Deveraturda et al. (2015), Mbaé et al. (2016), Pavlin et al. (2015) and Ranaivoson et al. (1994) in (A) absolute and (B) relative number of cases.

The duration of symptoms ranges from a few hours to over 4 weeks (Champetier de Ribes et al., 1998). The majority of grade I patients do not experience any additional effects and typically recover within a week after poisoning (Bagnis and Bourligueux, 1972; Fussy et al., 2007). For example, Singh et al. (2016) reported how six villagers with symptoms of vomiting, epigastric pain

and burning pain in the throat, mouth, and lips recovered completely between 3 and 7 days. Most patients with moderate forms (grade II) recover completely within about 3 weeks if no aggravation takes place (meaning patients do not reach grade III). In contrast, high mortality percentages are observed among patients who reach stage III, and even if they awake from their comatose condition, often neurological sequelae exist (Fussy et al., 2007).

Chandrasiri et al. (1988) describe the autopsy results of the two lethal cases in the Talpe incident, 1985, which revealed mostly changes in the lungs: a gross thickening of the alveolar septa with interstitial oedema with hemorrhage and microthrombi in the lumen of pulmonary arterioles. The brain showed interstitial oedema while myocardial fibers showed diffuse degeneration and necrosis. A possible mode of action could thus have been an alveolar capillary block induced hypoxia, resulting in cardio and pulmonary failure in these cases. Silas and Fernando (1984), as well as Dewdney (1967), also attributed death to respiratory depression in other cases. Singh et al. (2016) on the other hand described how an autopsy revealed ulceration and congestion with edema of the mucosa throughout almost every part of the gastrointestinal tract, alongside congestion of kidneys and brain throughout the entire cortex. Sub-pericardial patchy hemorrhages were observed in the heart, and by consequence, forensic reports concluded that the cause of death was severe neurotoxicity followed by sudden cardiac failure.

The chelonian most frequently mentioned in the literature and seemingly accountable for the highest mortality due to ingestion of its tissue is the hawksbill. Ingestion of this species has been associated with the gravest poisoning symptoms as well, as reported by Robinson et al. (1999). These authors conducted a survey on knowledge, attitudes, and practices regarding seafood poisoning in the Tuléar Province of Madagascar. This survey included inquiries into the clinical

291 signs associated with consumption of various turtle species by local inhabitants. While individuals
292 attributed vomiting and diarrhea solely to poisoning from leatherback turtle consumption, they
293 associated asthenia, fever, headaches, vertigo, and abdominal pain, in addition to vomiting and
294 diarrhea, with chelonitoxism resulting from consuming green turtles. In contrast, the most severe
295 symptoms were linked to the consumption of hawksbill turtles, with villagers additionally
296 reporting stomatitis, mouth ulcers, itchiness, conjunctivitis, coma, and death. Most reports indicate
297 that all organs are potentially toxic regardless of preparation (boiling, cooking), suggesting a
298 thermoresistant toxin (e.g. in Champetier de Ribes et al., 1999; Mbaé et al., 2016). Ingestion of
299 the turtle fat caused more severe intoxication symptoms in adults (Rasamimanana et al., 2017a,b).
300 Ranavaison et al. (1994) found a significant difference in type/piece of meat consumed and
301 subjects who consumed eggs were often more ill compared to those who did not (57% versus 23%,
302 p=0.03).

303 It has also been observed that the toxic effects are dose-dependent, as symptoms tend to be most
304 severe in individuals who consume larger quantities of flesh (e.g. Fussy et al., 2007; Mbaé et al.,
305 2016; Pavlin et al., 2015; Ventura et al., 2015). For example, Mbaé et al. (2016) found a lower
306 attack rate with those who had consumed 1 to 5 pieces of turtle meat (25%), while those who had
307 consumed more than 5 pieces were 3 times more likely to develop symptoms (attack rate of 75%).
308 However, it is important to note that this study did not clarify how a "piece" was defined or
309 standardized, making comparison not straightforward.

310 It is noteworthy that breast-fed children are also affected by sea turtle poisoning, despite not
311 consuming the turtle flesh itself, as reported by several studies (Ariyananda and Fernando, 1987;
312 Buden, 2011; Kirschner and Jacobitz, 2011; Mbaé et al., 2016; Ranaivoson et al., 1994;

313 Rasamimanana et al., 2017a,b). The responsible toxins can therefore pass into breast milk.
314 Transplacental contamination might have occurred in the study case of Fussy et al. (2007), where
315 the fetus perished during the early stages of the severe phase of poisoning, when the mother's
316 metabolic condition itself was still stable.

317 ## 3.4 Biotoxin origin

318 ### *3.4.1 Previous attempts and challenges to identify the causative toxin(s)*

319 The process by which individual turtles become poisonous remains ambiguous as the specific
320 toxins responsible for sea turtle poisoning remain unknown (Aguirre et al., 2006). Limpus (1987)
321 concluded that the freshness of contaminated turtle meat seems unrelated to its toxicity, and neither
322 washing nor cooking removes the toxic component. Ronquillo and Caces Borja (1968) report how,
323 following the 1954 incident in the Philippines, left over turtle meat was salted and sent to the
324 laboratory to be fed to rats, but failed to give proof of poisoning as the rats did not display any
325 symptoms. Interestingly, Chevellier and Duchesne (1851) noted that, if the flesh of a hawksbill
326 turtle has been salted, the purgative effects of the poison would be lost.

327 It is currently widely believed that one or possibly multiple toxins stem from prey items consumed
328 by the turtles, resulting in an accumulation of toxic compounds within the food chain (e.g., Aguirre
329 et al., 2006). However, the specific turtle tissues where these toxins accumulate have yet to be
330 pinpointed. Moreover, as with many turtle meat poisoning outbreaks, most studies were not able
331 to identify the responsible toxin directly via laboratory confirmation due to insufficient laboratory
332 capacity (e.g. Deveraturda et al., 2015) or because of the absence of leftover samples at the time
333 of investigation (e.g. Mbaé et al., 2016). Only two out of the 33 collected studies reporting
334 poisoning incidents have attempted to identify the responsible toxin. Noteworthy, Pavlin et al.

(2015) report how, after a poisoning incident, samples of patient sera, scutes (shell plates) and bones of the implicated hawksbill (as no turtle flesh was left), the internal organs from a deceased dog, and algae from the neighbouring reef (presumed to be *Moorena producens*), were tested for the presence of various marine toxins, including lyngbyatoxin A, debromoaplysiatoxin, okadaic acid, gymnodimine, pectenotoxin 2, and dinophysistoxins. All samples tested were found to be below the limit of detection for all toxins examined.

3.4.2 Limited evidence for lyngbyatoxins as causative agents in poisoning incidents

In the study by Yasumoto (1998), lyngbyatoxin A was isolated from the 'flesh' of a green turtle responsible for a fatality in Madagascar by liquid chromatography–mass spectrometry (LC–MS). This toxin likely originated from a cyanobacterium of the genus *Moorena,* which was deduced to be ingested unintentionally by the turtle while grazing. The study did not provide additional details, such as the methodology used and the toxin concentrations obtained. Nevertheless, 7 out of the 33 studies reporting chelonitoxism incidents, mention lyngbyatoxins as the potential causative agent afterwards, despite not having performed any laboratory testing, and/or report the presence of the cyanobacteria at the site of the incident. *Moorena producens* Engene and Tronholm 2019 (previously in literature as *Lyngbya majuscula* and/or *Lyngbya sordida,* and later, *Moorea producens* Engene et al. *nom. inval.*), is a benthic filamentous cyanobacterium distributed in tropical and temperate regions globally, including India, Sri Lanka, Philippines, Madagascar, Micronesia, Polynesia (Curren et al., 2002; Tronholm and Engene, 2019). This species thrives on solid or sandy substrates, rocks, mangroves, and coral reefs, at depths ranging from 0.3 to 30 meters (Curren et al., 2022; Engene et al., 2012). Furthermore, *M. producens* has been observed to epiphytically grow on other macroalgae and seagrasses (Ganesan et al., 2015; Yasumoto, 1998), including on those cultivated in Kenyan seaweed farms (Wakibia et al., 2006). Blooms of *M.*

producens are common in tropical regions and are documented to possess toxicity attributed to secondary bioactive metabolites (Curren et al., 2022). The pharmaceutical potential of *M. producens* has been well documented with a total of 231 natural products being identified by Curren et al. (2022). The top three groups of identified secondary metabolites were malyngamides (13.0%), microcolins (7.7%) and dolastatins (5.6%) with various cytotoxic effects. Yet, most studies on chelonitoxicity have focused on lyngbyatoxin A, the primary lyngbyatoxin variant, which was found to directly induce dermatitis via patch testing in humans and inflammation in tissues (Grauer and Arnold, 1961; Osborne et al., 2001; Osborne and Shaw, 2008), which may have led to the observed sore throats, mouth pain, and oral ulcers (Table 1, Suppl. Table 2). Moreover, lyngbyatoxin A induced contractions of isolated rabbit aorta rings when exposed to concentrations of 1 µM (Robinson et al., 1991). Jiang et al. (2014) identified three additional analogues of lyngbyatoxin which displayed varying toxicities against L1210 murine lymphocytic leukemia cells. Among them, 12-epi-lyngbyatoxin A exhibited the highest toxicity, with an IC50 value of 20.4 nM. Meanwhile, 2-oxo-3(R)-hydroxy-lyngbyatoxin A and 2-oxo-3(R)-hydroxy-13-N-desmethyl-lyngbyatoxin A showed lower cytotoxicity, with IC50 values of 98 and 32 nM, respectively. Lyngbyatoxins B and C, described by Aimi et al. (1990), elicited a positive irritant response when tested on mice ears. While there is so far no evidence of lyngbyatoxin's neurotoxicity, significant quantities might contribute to the neurological symptoms observed in the fatal poisoning cases. *Moorena* spp. have been found to bloom in feeding areas of green turtles that were involved in chelonitoxism incidents (e.g. Pavlin et al., 2015; Yasumoto, 1998), as well as elsewhere. In Shoalwater Bay, Queensland, Australia, and Florida, USA, for example, has *M. producens* been documented in the feeding grounds of green turtles (Arthur et al., 2006; Capper et al., 2013). According to their research, 51% of observed turtles were found to consume *M.*

381 *producens*. The authors suggested the turtles ingest the cyanobacterium while feeding on the
382 seagrass and red macroalgae to which *M. producens* is attached. Lyngbyatoxin A was detected in
383 a range of tissues sampled from live green turtles, demonstrating trophic transfer of toxin from the
384 cyanobacterium to a herbivorous vertebrate (Arthur et al., 2008). Mortality in humans following
385 the direct consumption of *M. producens* has been reported on minimum two occasions (Sims &
386 Zandee van Rilland, 1981; Marshall & Vogt, 1998). Indirect human consumption of "*Lyngbya*",
387 growing epiphytically on the edible endemic Hawaiian alga *Gracilaria coronopifolia,* was also
388 linked to a poisoning incident from 1994, as a result from ingesting the red alga (Nagai et al., 1996;
389 Ito & Nagai, 2000). Similar to the observations made from chelonitoxicity incidents, the direct
390 consumption of the cyanobacteria (associated with consuming seaweed) has been associated with
391 an excruciating burning sensation on the patient's lips, anterior part of the oral cavity, and the
392 anterior portion of the tongue. Twenty-four hours after consumption, the mucous membranes
393 appeared scalded and swollen and the patient became free of discomfort after 3 days (Sims &
394 Zandee van Rilland, 1981).

395 Lyngbyatoxin A is slightly lipophilic, with an experimentally obtained mean log $K_{octanol/water}$ of 1.53
396 ± 0.02, indicating the toxin to be 33.9 times more soluble in octanol than in water (Stafford et al.,
397 1992). However, the established log K_{ow} is smaller than the threshold value of 4 (OSPAR, 2003),
398 indicating that lyngbyatoxin A has a low potential for bioaccumulation and therefore is not
399 expected to accumulate to significant levels in aquatic organisms or through intermediate trophic
400 levels. Nevertheless, the sequestration of lyngbyatoxin A in some invertebrates has been
401 demonstrated. For example, a high concentration of this toxin was observed in the body of the sea
402 hare *Stylocheilus striatus* (3.94 $mg.kg^{-1}$) compared to its secretions (ink 0.12 $mg.kg^{-1}$, fecal matter
403 0.56 $mg.kg^{-1}$; eggs 0.05 $mg.kg^{-1}$) after consumption of *Moorena* (Capper et al., 2005). In other

404 sea hare species, toxin concentrations were significantly higher in their secretions or in certain
405 parts of the body, suggesting strategies for actively sequestering *Moorena* secondary metabolites
406 in these sea hares, probably providing a defensive role to these organisms (Capper et al., 2005).
407 While some invertebrate grazers appear to be indifferent to extracts of *Moorena*, some reef fish
408 are more likely to be deterred (Capper et al., 2006). Implications for bioaccumulation or
409 biomagnification of lyngbyatoxin A through the marine food web warrant further investigation.
410 The relatively low toxicity of lyngbyatoxins (see also discussion below), their low potential for
411 bioaccumulation, their association with some but not all clinical signs, and the lack of detection in
412 poisoning incidents, do not offer persuasive arguments to support the involvement of
413 lyngbyatoxins in the phenomenon.

414 ### *3.4.3 Sea turtle exposure and sensitivity to biotoxins*

415 Some studies (e.g. Fussy et al., 2007) argue it is less probable that phycotoxins play a role in
416 poisoning by *E. imbricata*, as it is predominantly a carnivorous species, primarily feeding on
417 sponges (Meylan, 1998; Léon and Bjorndal, 2002). Yet, symbiotic sponge-cyanobacteria have
418 been described in literature (Konstantinou et al., 2021; Mutalipassi et al., 2021), which are also
419 producers of potentially toxic secondary metabolites, such as alkaloids. Yet, sponges (and other
420 filter-feeding organisms) could also potentially take up toxins from the water column directly.
421 Finally, sponges themselves are also producers of an arsenal of secondary metabolites, even in
422 those species that are not associated with cyanobacteria. The toxic properties of several sponge
423 species have been demonstrated already on a plethora of marine organisms (e.g., Lee et al., 2021;
424 Proksch, 1994). *E. imbricata* was considered to be primarily a sponge-feeding specialist and
425 secondarily an omnivorous species, as it has been reported to also feed on sea grasses and algae
426 occasionally (Limpus, 2009). However, Bell (2012) observed hawksbills, foraging on reefs of the

427 Northern Section of the Great Barrier Reef Marine Park (Australia), to be primarily algivorous
428 (72.7% of all ingested food items could be attributed to macroalgae from the three main macroalgal
429 taxonomic divisions) and secondarily omnivorous. As a result, algal toxins cannot be ruled out as
430 the origin of the chelonitoxism phenomenon, especially as the clinical picture of chelonitoxism
431 caused by the different turtle species is identical.

432 Toxic harmful algal blooms (HABs) negatively affect human and wildlife health. Air breathing
433 vertebrates can inhale these aerosolized biotoxins, but ingesting food containing the toxin appears
434 to be the most significant means of exposure (Amaya et al., 2018; Foley et al., 2019; Landsberg et
435 al., 2009). Harmful algal toxins have been previously documented in the diet and tissues of several
436 sea turtle species, for example, in Australia (Arthur et al., 2006, Arthur et al., 2008, Takahashi et
437 al., 2008), El Salvador, (Amaya et al., 2018), Mexico (Trejo et al., 2016), and the USA (Fauquier
438 et al., 2013, Foley et al., 2019; Perrault et al., 2020; Walker et al., 2018), as well as in sea turtle
439 plasma (e.g. Perrault et al., 2014, 2016, 2017; Walsh et al., 2010). Most of HAB toxin
440 concentrations (i.e., brevetoxins, dinophysistoxins, domoic acid, lyngbyatoxins, okadaic acid, and
441 saxitoxins (STXs)) measured in sea turtle tissues are usually low (e.g. Capper et al., 2013). In only
442 few cases, excessive doses of phycotoxins can be linked to the death of these animals (e.g. 3 out
443 of 13 green turtles in Capper et al., 2013) or to adverse effects within sea turtle populations such
444 as changes in other physiological parameters (e.g. Perrault et al., 2014, 2016, 2017). For instance,
445 in Morobe Lagoon, Papua New Guinea, fish and turtle fatalities have been linked to red tides
446 caused by armored dinoflagellates, *Pyrodinium bahamense* (MacLean, 1975). Between 2013 and
447 2017, residents of Limón Province in Costa Rica witnessed mass deaths of Caribbean sharpnose
448 puffer fish (*Canthigaster rostrata*) coinciding with green turtle strandings, which exhibited
449 abnormal neurological symptoms (Barrientos et al., 2019). Biotoxin analyses suggested that the

turtles were exposed to and adversely affected by saxitoxins from opportunistically scavenging on *C. rostrata* during fish mortality incidents (which were confirmed via stomach analyses). Concentrations of STXs in stranded fish (whole body, 16.6–47.5 µg STX-eq/g; mean of 29.0 µg STX-eq/g) exceeded human seafood safety threshold (0.8 µg STX-eq/g) significantly, while also being quantified in ingested puffer fish within the turtle (mean of 0.78 µg STX-eq/g), as well as in the serum, lung, kidney and brain tissues of the turtle itself (approximately 0.04 µg STX-eq/g). Based on the average STX concentration and lethal doses derived from other taxa, the authors concluded that ingestion of 3–9 fish per kg body weight would provide enough toxin to reach the LD50 range in sea turtles. Barrientos et al. (2019) confirmed that the number of fish ingested by turtles easily exceeded this dose as the stomachs of necropsied turtles contained the remains of dozens of specimens.

The consumption of turtles affected by the mentioned phycotoxins (i.e., brevetoxins, dinophysistoxins, domoic acid, okadaic acid, and saxitoxins) has not been associated with any human poisoning cases yet. Comparing the symptoms associated with exposure to these phycotoxins (Table 2) does not indicate that any specific toxin can be directly linked to the symptoms observed in cases of sea turtle consumption. Comparing toxicity values (acute oral and peritoneal LD50 values for mice) for these biotoxins, suggests a lower toxicity of lyngbyatoxin A in mice, and thus potentially other vertebrates, such as chelonians, as well (Table 2). Based on EC50 values obtained from standard oral and intraperitoneal toxicity tests in mice, saxitoxin appears to be the most toxic of all phycotoxins that have been identified in sea turtles so far (Table 2). A maximum limit value of at 800 µg/kg in marine seafood for STX toxins has been set by the European Regulation No. 853/2004, corresponding with most limits established in countries outside the EU as well (Australia, USA, Canada; Sanseverino et al., 2017). To our knowledge,

there are no specific guidelines and regulations for lyngbyatoxins in seafood both in Europe and in other countries and territories around the globe. The responsibility of lyngbyatoxins in the sea turtle poisoning incidents cannot be fully confirmed, and other toxins may also be involved.

Table 2.Summary of observed symptoms in mammals after exposure to harmful algal toxins that have been identified in sea turtles. Acute toxicity in mice is summarized as lethal dose (LD50) values. Occurrence of symptoms, mechanisms of action, hydrophilic or lipophilic characteristics, as well as European Union (EU) maximum regulatory limits, are reported.

Toxin	Oral toxicity in mice, LD50 (µg/kg bw)	Intraperitoneal toxicity in mice, LD50 (µg/kg bw)	Symptoms in mammals	Primary mechanism(s) of action	Hydrophilicity	EU Regulatory limit in marine seafood (µg/kg bw)	References
Brevetoxin 2	520	/	Paraesthesia, dizziness, nausea, vomiting, ataxia, muscle weakness, asthenia as well as seizures and coma	Neurotoxic; respiratory irritant	Lipophilic	Not regulated	Poli et al., 1986
Brevetoxin 3	6600	875	Paraesthesia, dizziness, nausea, vomiting, ataxia, muscle weakness, asthenia as well as seizures and coma	Neurotoxic; respiratory irritant	Lipophilic	Not regulated	Costas et al., 2023; Poli et al., 1986
Dinophysistoxin-1	897	150.4	Gastrointestinal (diarrhea, nausea, vomiting, body weight loss, reduced food consumption)	Gastrotoxic	Lipophilic	160	Suzuki and Okada, 2018
Domoic acid	/	3600	Severe headaches, loss of balance or dizziness, vision disturbances, memory loss, cardiac arrhythmias, unstable blood pressure, hiccoughs, bronchial hypersecretion; involuntary chewing, grimacing, myoclonia, convulsions; coma	Neurotoxic	Hydrophilic	20000	Grimmelt et ak., 1990
Lyngbyatoxin A	2000	300	Gastrointestinal and dermatologic symptoms (dermatitis), eye irritation, asthma	Dermatoxic, Gastrotoxic	Lipophilic	Not regulated	Cardellina et al., 1979
Okadaic acid	1069	192, 210, 225	Gastrointestinal (diarrhea, nausea, vomiting, body weight loss, reduced food consumption)	Gastrotoxic	Lipophilic	160	Dickey et al., 1990; National Center for Biotechnology Information, 2024; Park et al., 2023; Tachibana et al., 1981; Tubaro et al., 2003
Saxitoxin	263	8	Gastrointestinal (nausea, vomiting) and neurological (cranial nerve dysfunction, a floating sensation, headache, muscle weakness, paresthesia and vertigo), respiratory failure and death can occur from paralysis	Neurotoxic	Hydrophilic	800	Cheymol and Toan, 1969

3.4.4 Suggested approach to identify the responsible toxin(s)

One of the main challenges in identifying the causative compounds is that only a fraction of marine toxins have been identified, and standards are available for only a few of them (Gerssen et al., 2019). Current food safety regulations now recommend incorporating *in vitro* bioassays to detect compounds based on their effects, enabling broad screening of samples and serving as valuable tools for identifying new and emerging contaminants. For instance, a general assay that measures the reduction of MTT (3-[4,5-dimethylthiazol-2-yl]-2,5 diphenyl tetrazolium bromide) by neuroblastoma cells appears effective for detecting marine biotoxins like okadaic acid, saxitoxins, ciguatoxins, and tetrodotoxins in shellfish (Gerssen et al., 2019; Okumura et al., 2005). In some cases, cell-based bioassays can be even more sensitive than chemical analysis. For example, with ciguatoxins, the neuro-2a assay can detect levels considered relevant for human exposure, while LC-MS methods often lack this sensitivity (Caillaud et al., 2010). Since 'new' compounds are unlikely to be included in current libraries, they are typically missed by MS-based methods. Thus, using effect-based bioassays can provide a first screening against unknown analogues, which may exhibit unforeseen adverse effects. In addition to their sensitivity, *in vitro* bioassays help prevent a tunnel vision on screening of known or specific agents causing intoxication or observed effects. They are usually also cost-effective, highly sensitive, and suitable for testing large numbers of samples efficiently (Gerssen et al., 2019). Samples testing positive should be followed up by more dedicated specific bioassays and chemical analysis to reveal the identity and nature of the toxins.

Extraction and fractionation methods should be as broad as possible to avoid matrix effects in either the applied bioassays, MS-based methods, or both (Gerssen et al., 2019). If the extraction or fractionation is too specific, there is a higher risk of missing the compound of interest. Untargeted MS data collected can be analyzed using chemometric models (either unsupervised or

504 supervised) to identify relevant m/z values and to determine the elemental composition based on
505 accurate mass data and isotopic distribution in the spectra (Gerssen et al., 2019). The obtained data
506 can then be searched against large databases such as ChemSpider or PubChem. Additionally, MS
507 fragmentation data can help narrow down potential matches in these databases. *In silico*
508 fragmentation tools, like MetFrag, could also be used with MS fragmentation data to further
509 tentatively identify the bioactive compound (Gerssen et al., 2019).

510 Once a candidate compound is tentatively identified, the simplest approach is to obtain the pure
511 compound (if available) and use it as a reference standard in the used bioassays to verify the results.
512 If the pure compound is not commercially available, a complete purification and identification
513 using preparative LC and nuclear magnetic resonance (NMR) may be required.

514 ### 3.5 Remediation

515 Silas and Fernando (1984) noted that there is often a complete lack of knowledge about turtle
516 poisoning and its effects among local medical practitioners. In two private clinics at Udangudi
517 (India), the doctors who suspected that the observed symptoms (January, 1961) could have been
518 caused by a neurotoxin, suggested mild purging to eliminate toxins, and administered charcoal
519 tablets to detoxicate and tetracycline as antibiotic and vitamins (Silas and Fernando, 1984). There
520 is no antidote for sea turtle poisoning; treatment is solely supportive and symptomatic, with
521 intensive care if necessary (Fussy et al., 2007; Silas and Fernando, 1984). In the last decade
522 however, physicians and veterinarians have begun to use intravenous lipid emulsion (ILE), i.e.
523 bringing a lipid emulsion directly into the bloodstream, in the treatment of various acute
524 intoxications (Perrault et al., 2021; Rothschild et al., 2010). ILE are now commonly administered
525 to eliminate lipophilic toxicants from their sites of action, drive the offending chemical from target

526 tissues into the newly formed 'lipid sink' (Perrault et al., 2021; Rothschild et al., 2010). The
527 successful use of ILE to treat neurotoxicity has been documented in numerous studies in domestic
528 animals and humans in a variety of toxicosis incidents (e.g. DeGroot, 2014; Gwaltney-Brandt et
529 al., 2012; Heggem-Perry B et al., 2016; Rothschild et al., 2010), as well as for green turtles exposed
530 to domoic acid, as well as loggerheads, Kemp's ridleys and green turtles experiencing
531 brevetoxicosis (Perrault et al., 2021).

532 Given that any turtle or turtle eggs could potentially be toxic, we advise discontinuing the
533 consumption of turtle products in those regions where chelonitoxic incidents have occurred,
534 especially because of the potential thermoresistant nature of possible causative toxins. During
535 cases of poisoning by marine animals or episodes of chelonitoxism, breastfeeding must be
536 suspended immediately. Moreover, it is crucial to follow up on cases of intoxication in humans
537 and animals. Clear instances of intoxication after consumption of sea turtle tissue should be
538 reported to the relevant authorities, and subsequent studies should be initiated using a combination
539 of effect-based assays and targeted or untargeted chemical analysis, as suggested above. As sea
540 turtles are endangered globally, initiatives aimed at safeguarding human health through reduced
541 turtle capture also contribute to the preservation of these threatened and endangered species. For
542 example, Ventura et al. (2015) describe how after the poisoning incident, a community assembly
543 was organised by the Municipal Health Office and Department of Health to educate villagers on
544 the law prohibiting the killing and consumption of sea turtles and it's dangers. Yet, many
545 inhabitants of remote islands rely on fishing as their main means of sustenance. Despite being
546 aware that capturing, harming, and trading sea turtles is forbidden and carries legal consequences,
547 they persist in these activities due to the local demand for this delicacy (Ventura et al., 2015).
548 Similarly, despite awareness of these health risks, consumption habits often persist (Friant et al.,

549 2015). To avoid consumption practices, engaging social actors/stewards in the local communities
550 through regulatory authorities and other stakeholders, could be more effective. Aquaculture could
551 offer advantages in food quality and safety compared to wild catch through controlled
552 environments and management practices. A historical practice of meat production from the green
553 sea turtle indeed persists in several regions worldwide, such as Grand Cayman Island, Réunion,
554 and the Ogasawara Islands of Japan (Magnino et al., 2009). With high production costs and limited
555 economic viability, there has also been controversy related to animal health challenges and welfare,
556 potential facilitation of illegal trade, and potential disease transmission to wild populations and the
557 society (e.g. Arena et al., 2014; D'Cruze et al., 2015; Warwick et al., 2013). Due to being resource
558 intensive, its value as an alternative for traditional turtle consumption by impoverished
559 communities or as an alternative conservation effort (as compared to bycatch reduction, habitat
560 protection, reduced consumption) has been regarded as inefficient and even a questionable practice
561 by conservationists (D'Cruze et al., 2015). It is noteworthy that the exportation and international
562 trade of sea turtle meat is strictly prohibited under the Convention on International Trade in
563 Endangered Species of Wild Fauna and Flora (CITES) Treaty. Entering into force in 1975, 59
564 countries had become signatories to CITES by 1980, a number that increased to 184 up to now.
565 Despite considerable debate, marine turtle species have been listed on the IUCN Red List of
566 Threatened Species since 1982 (Humber et al., 2014; IUCN, 2024). Consequently, consumption
567 of sea turtle meat is confined to the local populace and is commercially available within domestic
568 markets.

569 ## 4. Conclusion

570 The term chelonitoxism describes intoxication resulting from the consumption of sea turtle flesh.
571 The health data reviewed here offers valuable insights for healthcare providers and the public,
572 highlighting the potential risks associated with eating sea turtles. Given past mortality rates linked
573 to turtle poisoning, it is advisable especially for nursing mothers and children to refrain from
574 consuming any sea turtle products. Lyngbyatoxins, originating from the cyanobacterium *Moorena*
575 *producens*, have been suggested to be (one of) the causative agents. However, due to limited
576 evidence, the involvement of other compounds needs further investigation. A potential remediation
577 for sea turtle poisoning victims is the use of an intravenous lipid emulsion treatment, which has
578 already been successfully applied to cure sea turtles suffering from brevetoxicosis as well.
579 Disseminating this information through public health campaigns could both improve public health
580 and aid in sea turtle conservation efforts by reducing human consumption of these endangered
581 species in regions where consumption is frequent and toxicosis has been reported.

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587

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