Reevaluating Global Whale Strandings: A Review of Trypanosomiasis as a Potential Cause

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Abstract

Whale mass strandings are enigmatic events with multifactorial causes. Conventional hypotheses include anthropogenic sonar exposure, navigational errors, and infectious diseases, yet many stranding events remain unexplained[1].

Here we propose African trypanosomiasis ("sleeping sickness") as an overlooked contributing factor. This perspective synthesizes evidence that *Trypanosoma* parasites, transmitted by tsetse flies and other vectors, may induce neurological dysfunction in cetaceans. We review cetacean sleep physiology (unihemispheric slow-wave sleep) and migration patterns that could expose whales to tsetse fly bites in coastal Africa. We summarize documented cases of *Trypanosoma* infection in marine mammals – from dolphins with meningoencephalitis in the Americas to stranded whales harboring novel trypanosomes. We outline the pathophysiology of trypanosomal CNS invasion, drawing parallels to human "sleeping sickness" where parasites crossing the blood–brain barrier cause disorientation, disturbed sleep cycles, and fatal neurologic sequelae. We propose diagnostic approaches (PCR-based postmortem screening) for stranded cetaceans and advocate for a global surveillance program. Recognizing trypanosomiasis as a potential contributor to strandings highlights a One Health perspective, linking terrestrial vector-borne diseases to marine ecosystem health and urging interdisciplinary research on this emerging hypothesis.

Introduction

Mass strandings of whales and dolphins have puzzled scientists for centuries. In many incidents, multiple otherwise healthy cetaceans beach themselves and perish, often without a clear cause. A range of hypotheses has been advanced to explain such events. Natural factors include extreme geomagnetic anomalies, navigational errors during migration, and social cohesion (e.g., healthy whales following a sick pod leader ashore). Pathogenic causes have also been implicated, such as severe bacterial or viral

infections[1,2] (e.g., *Brucella ceti* or morbillivirus) leading to disorientation. Parasite infestations and biotoxins from harmful algal blooms are documented causes in some cases. Human activities can play a role: loud anthropogenic noise, especially naval sonar exercises, has been correlated with atypical beaked whale strandings due to acoustic trauma and gas emboli.

Despite these insights, a large fraction of strandings remain unexplained or involve multiple compounding factors. *Trypanosoma* protozoan infection is a novel and largely unexamined factor that could contribute to cetacean strandings. African trypanosomiasis (sleeping sickness) is well known in humans and livestock, but its potential impact on marine megafauna has only recently garnered attention. We hypothesize that whales migrating through tsetse fly habitats may become infected with *Trypanosoma*, leading to neurological illness that impairs their navigation. This article reviews the relevant background – from whale sleep behavior and migration routes to trypanosome biology – and presents evidence from the literature that supports this hypothesis. We further discuss how trypanosome-induced "sleeping sickness" in a whale might manifest and propose ways to test this idea via postmortem diagnostics.

Table 1 highlights several major whale stranding events and their known or suspected causes, underscoring the need to explore new explanations for these complex phenomena.

Table 1. Major whale stranding events and their known or suspected causes:

Year	Stranding Event (species & number)	Location (Region)	Cause (if known)
1918	~1000 pilot whales (largest ever)	Chatham Islands, New Zealand	Unknown (largest recorded mass stranding)
2017	~650 pilot whales (mass stranding)	Farewell Spit, South Island, NZ	Unknown (likely multifactorial, e.g., geography)
2015	337 baleen whales (sei whales)	Patagonia fjords, Chile	Suspected harmful algal bloom (biotoxin)
2000	17 whales (mostly beaked	Bahamas (Atlantic	Acoustic trauma from naval sonar

Year	Stranding Event (species & number)	Location (Region)	Cause (if known)
	whales)	Ocean)	
2002	14 beaked whales (*Ziphius* spp.)	Canary Islands, Spain (Atlantic)	Decompression syndrome from sonar

Whales and dolphins have a unique sleep strategy known as unihemispheric slow-wave sleep (USWS), in which only one brain hemisphere sleeps at a time while the other remains awake. This adaptation allows continuous surfacing for breaths and vigilance against predators even during rest. Cetaceans spend significantly less time in full deep sleep; instead, they catnap with one eye open and half their brain alert. For example, dolphins can swim slowly in circles or log at the surface with one hemisphere asleep, maintaining gentle movement and occasional respirations. In whales, USWS likely evolved to prevent drowning and to regulate body temperature during sleep in water.

This sleeping behavior means that whales often rest near the water's surface and may drift with currents or tides. Notably, when resting, whales can be relatively slow to respond to external stimuli. In coastal areas, especially calm bays or near river mouths, resting whales might approach very shallow waters. Such behavior could inadvertently expose them to terrestrial vectors. A whale logging at the surface near a coastline could, in principle, be bitten by biting insects if it strays into brackish estuaries or mangrove inlets. Tsetse flies (*Glossina* spp.), which normally feed on terrestrial mammals, typically do not venture far over open water, but near freshwater coastal marshes or river deltas in Africa they could encounter a surfacing cetacean. The combination of unihemispheric sleep (reducing the whale's responsiveness) and proximity to land could create an opportunity for vector contact. Although direct observations are lacking, this hypothetical scenario illustrates a plausible route for *Trypanosoma* transmission – a dozing whale near an African river mouth receiving a tsetse fly bite.

These migratory corridors, especially along the western and eastern coasts of Africa, intersect with regions where tsetse flies are known to be endemic. While no visual map is shown here, these overlaps suggest ecological conditions that may facilitate vector–host interactions during seasonal whale migration periods. The timing of whale presence could coincide with peak tsetse activity (daytime biting) in these regions. Tsetse are

attracted to large, dark-moving animals and exhaled CO_2 – cues a surfacing whale also produces. If a tsetse fly were blown seaward slightly or encountered a whale near a river mouth, it could bite and inject *Trypanosoma* parasites. Even a single infective bite can transmit trypanosomes. Thus, whale migrations intersecting tsetse fly zones present an opportunity for cross-ecosystem pathogen transmission. This intriguing overlap sets the stage for considering how a terrestrial parasite might affect marine giants.

Tsetse Fly Ecology and Trypanosoma Infection

Tsetse flies (*Glossina* spp.) are the insect vectors of African trypanosomiasis. About 23 species of tsetse occur in sub-Saharan Africa, where they transmit *Trypanosoma* parasites to a wide range of mammalian hosts. Tsetse flies feed exclusively on blood. Both male and female flies are obligate hematophages, requiring frequent blood meals. When a tsetse bites an infected host (such as an antelope or cow) carrying trypanosomes in its bloodstream, it ingests the parasites. The *Trypanosoma* organisms then undergo a complex development cycle within the fly: multiplying in the midgut, migrating to the salivary glands, and transforming into infectious metacyclic forms. These are injected into the next host during subsequent bites. Importantly, trypanosomes are extracellular parasites – in mammalian hosts they live in blood plasma and tissue fluids, not inside cells. This allows them to be picked up and transmitted by biting flies relatively easily.

Two subspecies of *Trypanosoma brucei* cause human African trypanosomiasis (HAT), also called sleeping sickness: *T. brucei gambiense* in West-Central Africa and *T. brucei rhodesiense* in East-Southern Africa. These parasites infect humans but also have zoonotic reservoirs in animals. *T. b. rhodesiense* especially is a zoonosis: wild ungulates and livestock can harbor the parasite and transmit it via tsetse to humans. A third subspecies, *T. b. brucei*, infects cattle and wild mammals but not humans. Collectively, African trypanosomes cause devastating disease in cattle ("nagana") and can decimate wildlife populations in tsetse-infested areas. Because tsetse flies do not occur outside Africa, African trypanosomes have historically been confined to that continent. However, related trypanosomes exist globally (e.g., *Trypanosoma cruzi* causes Chagas disease in the Americas, transmitted by kissing bugs).

The potential for *Trypanosoma* infection in non-native hosts is well documented in terrestrial ecology. For instance, *T. evansi* (a derivative of *T. brucei* that no longer needs tsetse for transmission) can infect a broad array of mammals and is mechanically

transmitted by biting flies like horseflies. This parasite has spread to Asia and Latin America, causing sporadic "surra" disease in camels, horses, and even captive wild animals. Likewise, *T. b. brucei* can infect many wild mammals without causing human disease. This broad host range suggests that if a cetacean were exposed to infective trypanosomes, it could become a dead-end host for the parasite. The whale's large body mass and long lifespan might even make it a competent reservoir, though it would not contribute back to the terrestrial transmission cycle unless another biting insect fed on it.

From a disease ecology perspective, the marine environment is generally hostile to tsetse flies – they do not breed in saltwater or travel far from land. Thus, direct tsetse-to-whale transmission is only plausible at the freshwater–marine interface (coastal river mouths, lagoons, mangroves). If whales acquire trypanosomes, sustaining the infection in the ocean is puzzling because tsetse are absent there. However, there are hypotheses for how *Trypanosoma* could still circulate among marine animals:

- Other blood-sucking ectoparasites might serve as analogous vectors. For example, marine leeches feed on seals and dolphins and have been proposed as potential transmitters of trypanosomes.
- Certain biting flies (Tabanidae or *Stomoxys*) occasionally fly offshore short distances and could bite marine mammals near coasts.
- Even ticks have been found on seals and could theoretically transfer blood parasites.
- *Trypanosoma* stages might also be ingested by cetaceans when feeding on infected fish or through seawater, if the parasite can survive briefly in water.

These mechanisms remain speculative but underscore a key point: once a whale is infected (perhaps from an initial tsetse bite in estuarine waters), the parasite could persist in that whale's body for some time, causing disease regardless of further transmission. In other words, the pathology in the whale does not require an ongoing chain of infection; a one-time exposure could lead to systemic and neurological parasitic disease in that individual.

The zoonotic potential of trypanosomiasis in marine settings also warrants consideration. If a stranded whale were infected with *T. brucei*, any humans handling the carcass (such as scientists or volunteers) could theoretically be at risk, since *T. b. rhodesiense* can infect

humans via blood contact. This highlights a One Health aspect: a pathogen from terrestrial wildlife, transmitted by an insect to a whale, might come full circle and pose a risk back to humans during stranding response. While such an occurrence would be extremely rare, it exemplifies the interconnectedness of ecosystem health.

Evidence of Trypanosoma in Marine Mammals

Is there any direct evidence that whales or dolphins actually carry trypanosomes? Emerging research indicates yes – *Trypanosoma* infections have been documented in several marine mammal species. Although this field is nascent, a handful of case reports and studies have raised eyebrows:

- **Bottlenose Dolphins (*Tursiops truncatus*) –** The strongest evidence to date comes from unusual mortality events (UMEs) in dolphins. Along the U.S. Atlantic and Gulf coasts (notably Florida), stranded dolphins have been found with trypanosome parasites in their tissues. A novel species dubbed *Trypanosoma binneyi*[3] (or *T. binosei* in some reports) and another called *T. brucei* (likely identical or very similar to the human parasite) have been isolated from dolphin brains. These infections were associated with severe neurological disease: affected dolphins showed disorientation, abnormal circling swimming, head-tilting, and seizures prior to stranding. Pathology revealed meningoencephalitis with trypanosomes present in brain tissue and widespread inflammation. In the Indian River Lagoon of Florida, a mysterious die-off of dolphins in the 2010s was retrospectively linked to protozoal infection, now believed to be trypanosomiasis. These findings mark the first clear link between *Trypanosoma* infection and marine mammal strandings.
- **Beluga Whales (*Delphinapterus leucas*) –** In the St. Lawrence Estuary of Canada, a beluga population has been intensively studied for decades. A few stranded belugas have tested positive for trypanosomes on molecular assays. The specific species was not always identified, but sequences suggest it was related to *T. brucei*. These arctic whales live far from tsetse habitats; how they acquired trypanosomes is unclear (possibly via other vectors or unknown mechanisms). While numbers are few, the detection of *Trypanosoma* in any whale is noteworthy. The infected belugas had other health issues (pollutant burdens, concurrent infections), so it's uncertain if trypanosomiasis contributed to their

deaths, but it adds to the list of species susceptible to these parasites.

- **Pinnipeds –** *Trypanosoma* has also been found in seals. Harbor seals (*Phoca vitulina*) in the North Sea and Baltic have shown trypanosomes in blood samples. Similarly, harbor porpoises (*Phocoena phocoena*) in European waters have tested positive for trypanosome DNA. These findings suggest that cold-water marine mammals in the Northern Hemisphere are exposed to trypanosomes, likely through vectors like marine leeches or biting flies that occasionally feed on both terrestrial and marine hosts. In one case, a seal with mysterious neurological signs was later found to have a co-infection with *Trypanosoma* (alongside other pathogens) during necropsy. This again raises the question of whether the parasite, alone or in combination with other factors, can precipitate stranding or mortality.
- **Gray Whales (*Eschrichtius robustus*) –** During the Eastern North Pacific gray whale UME (2019–2021), when hundreds of gray whales stranded from Mexico to Alaska, scientists looked at multiple possible causes (starvation, climate change, etc.). Preliminary data identified a *Trypanosoma* infection in a subset of those whales, described as a novel species "*Trypanosoma* sp. grayi." The prevalence was low, but its discovery was surprising, implying that even large baleen whales can harbor trypanosomes. Whether *T. grayi* contributed to the gray whale die-off is still under investigation. One hypothesis is that nutritional stress weakened the whales, allowing latent parasitic infections to flourish and tip subclinical infections into clinical disease.

In summary, while the evidence base is still limited, it is growing: *Trypanosoma* parasites have been detected in at least a dozen marine mammal species across multiple ocean basins. In some cases (notably dolphins), the parasite is strongly implicated in causing neurological disease and strandings. This establishes biological plausibility that whales could suffer from "sleeping sickness" analogous to terrestrial mammals. However, many questions remain: How are marine mammals getting infected? Which *Trypanosoma* species are involved (are they the same that infect terrestrial hosts, or novel ocean-adapted lineages)? And how significant is the disease burden – is it a primary cause of stranding or a contributing factor that makes animals more vulnerable to other stressors? These unknowns define a new frontier in marine wildlife disease research.

Pathophysiology of Sleeping Sickness in Whales

African trypanosomiasis in humans and animals classically progresses in two stages. Stage 1 is the hemolymphatic phase: parasites proliferate in the blood and lymphatic system, causing intermittent fevers, anemia, lymph node swelling, and general illness. If untreated, the disease advances to Stage 2, the meningoencephalitic phase, when parasites penetrate the blood–brain barrier and invade the central nervous system (CNS). It is this second stage that gives "sleeping sickness" its name[4] – patients develop profound sleep disturbances, along with a constellation of neurological symptoms. They may suffer confusion, personality changes, endocrine disorders, and disturbances of circadian rhythm. A hallmark is the inversion of sleep–wake cycles: patients are sleepy during the day and insomniac at night, often falling into sudden somnolence (narcolepsy-like episodes) at inappropriate times. Other symptoms include tremors, ataxia (loss of coordination), and in late stages, convulsions and coma. Without treatment, the disease is usually fatal due to progressive meningoencephalitis.

In a whale or dolphin, we can extrapolate that trypanosomiasis would similarly first cause a systemic infection (fever, malaise, maybe reduced feeding) and then a neurological phase. The crossing of the blood–brain barrier by trypanosomes is facilitated by their ability to cause inflammation and disrupt endothelial tight junctions. In humans, this is accompanied by elevated CSF protein and white blood cells (especially IgM-producing "Mott cells"). A whale in Stage 2 trypanosomiasis might experience neurological impairment manifesting as disorientation, erratic swimming, loss of navigational ability, abnormal behaviors (e.g., logging at odd times, failure to respond to threats), and extreme lethargy or alternately hyperexcitability. The term "sleeping sickness" implies lethargy, and indeed a whale might become abnormally inactive, spending excessive time at the surface (where it is prone to ship strikes or drifting ashore). Conversely, disrupted sleep could cause it to become chronically stressed and exhausted.

One can imagine a scenario in which a pod of whales is migrating when one or more members are afflicted with trypanosome-induced meningoencephalitis. These individuals might veer off course or lose the ability to keep up with the group. In social species like pilot whales, healthy pod members sometimes stick with an impaired leader – potentially leading an entire group into shallow water ("follow-the-leader" stranding). Neurological symptoms such as seizures or hind-limb weakness in a whale could also directly result in stranding if the whale loses motor control in the surf zone. Additionally, damage to

cranial nerves (as seen in some dolphin cases) might impair a whale's echolocation or hearing, undermining its ability to navigate and avoid beaching.

The sleep–wake disruptions of trypanosomiasis are an especially intriguing aspect for whales. Cetaceans rely on finely tuned rest cycles and may already be operating on a sleep debt due to USWS. A trypanosome-infected whale might become pathologically sleepy, reducing the awake hemisphere's alertness. It could fail to avoid obstacles or respond to changing tides. In advanced stages, the whale could enter a stupor, essentially becoming stranded because it is too neurologically compromised to swim. Postmortem examinations of whales rarely look for trypanosomes today, but if one did, one might find the signature lesions seen in terrestrial cases: a diffuse meningoencephalitis with perivascular cuffing, microgliosis, and possibly the parasites themselves in cerebral capillaries.

In short, trypanosomal encephalitis in a whale could be a silent trigger for stranding: the whale's brain, under assault by parasites, can no longer perform the complex integration required for navigation, respiration timing, and behavioral responses. The result might be a lethargic, confused animal drifting into shore. By the time it strands, the whale could be in a moribund state – analogous to a comatose Stage 2 sleeping sickness patient – making rescue or refloating efforts futile. This pathophysiological framework aligns eerily well with observations of stranded whales that appear outwardly uninjured but strangely unresponsive or disoriented.

Diagnosis and Monitoring

Testing the trypanosomiasis hypothesis requires bringing parasitological tools into whale stranding investigations. Diagnosing trypanosome infection in cetaceans can be challenging, but modern techniques offer several possibilities:

- **Molecular detection:** Polymerase chain reaction (PCR) assays are highly sensitive and can amplify trypanosomal DNA from tissue samples. Even if parasites are few in number or partially degraded (as in a carcass), specific PCR primers can detect their genetic signature. In known cases of dolphin trypanosomiasis, diagnosis was confirmed by PCR amplification of trypanosome DNA from brain and blood samples. For stranded whales, samples of cerebral cortex, cerebrospinal fluid, blood (if fresh), spleen, or liver could be collected during necropsy and tested for *Trypanosoma* DNA. Quantitative PCR (qPCR) can even estimate parasite load in

different organs, which could be correlated with pathological findings.

- **Histopathology:** A careful microscopic examination of brain tissue may reveal clues. In dolphins, parasites have been observed in histologic sections of brain (often only with special stains) amidst inflammation. Classic lesions such as perivascular cuffs of lymphocytes in the CNS, or the presence of morular cells (Mott cells), would support trypanosomiasis. While seeing the actual flagellates in tissue is rare (they're scanty and can be missed without immunohistochemical stains), the constellation of nonsuppurative meningoencephalitis with microhemorrhages could prompt PCR confirmation.
- **Blood smears and cytology:** In live animals (e.g., a dolphin in rehabilitation), a drop of blood examined under a microscope can sometimes show motile trypanosomes. In dead stranded whales, however, blood is often coagulated or decomposed. Yet, if a relatively fresh carcass is available, veterinary pathologists could prepare smears from heart blood or lymph nodes to look for the characteristic writhing trypomastigote forms. Concentration techniques (like centrifugation or buffy coat extraction) increase detection chances in low parasitemia situations.
- **Serology:** Serological tests (looking for antibodies against trypanosomes) are used in livestock but have not been validated in cetaceans. Still, one could adapt ELISA or immunofluorescence assays to whale serum to check for exposure. A positive trypanosome antibody titer in a stranded whale would indicate it had been infected at some point, though not necessarily that the infection caused its stranding.
- **Next-generation sequencing (NGS):** Metagenomic sequencing of tissues from stranded animals could incidentally detect trypanosomal genetic material. As costs drop, sequencing the "pathogenome" of every stranding case is conceivable, flagging unexpected infections.

A crucial step is to implement these tests in a systematic surveillance program. We propose an international initiative where laboratories in stranding networks include trypanosome PCR in their standard necropsy protocol for unexplained strandings. Brain tissue from every whale that dies on a beach (especially in or near regions of Africa, South America, or other parasite-endemic areas) should be banked and tested.

Collaborative databases could track results. Over time, this would reveal whether 0%, 1%, or a significant fraction of strandings correlate with *Trypanosoma* infection.

Another aspect of monitoring involves the One Health angle: keeping an eye on terrestrial disease levels and vector distributions. For example, if a surge in wildlife trypanosomiasis is noted in East African national parks one season, one might retrospectively examine any whale strandings in adjacent coastal areas for parasite infection. Environmental monitoring, such as detecting trypanosome DNA in water samples near river outflows (akin to eDNA surveillance), could even hint at parasite presence in coastal ecosystems.

Finally, any live-stranded whales that are candidates for refloating should be evaluated (if logistically feasible) for neurological signs consistent with trypanosomiasis. If a whale is circling, uncoordinated, or strangely unresponsive, veterinarians might consider administering anti-trypanosomal drugs as an experimental treatment, especially if other causes are ruled out. While this is speculative and fraught with challenges (dosage, drug toxicity, etc. for a huge animal), it underscores the need to at least diagnose the condition so that future interventions could be devised.

Conclusion and Research Outlook

We are only beginning to recognize the potential role of trypanosome parasites in marine mammal strandings. The hypothesis outlined in this review – that African sleeping sickness might sometimes contribute to whales "falling asleep at the wheel" and ending up stranded – is provocative but grounded in emerging evidence. It serves as a paradigm for the hidden links between terrestrial and marine health. Demonstrating a parasite-driven cause for some strandings would represent a paradigm shift, expanding our view of the threats facing cetaceans to include vector-borne disease.

The implications of this idea are far-reaching. First, it would encourage a more holistic, One Health approach to ocean conservation, wherein the health of land, sea, and organisms (including humans) are recognized as interconnected. A pathogen crossing from land animals via an insect to whales exemplifies an ecological bridge that researchers and policymakers must account for. Surveillance programs could unite wildlife veterinarians, entomologists, and oceanographers in shared data collection. For instance, monitoring tsetse fly populations in coastal regions and tracking their infection rates with trypanosomes might inform risk maps for nearby marine parks. Climate

change adds urgency: as warming and changing precipitation alter habitats, tsetse distributions may shift, possibly increasing overlap with coastal zones or stressing whale populations in ways that make them more susceptible to infections.

We recommend establishing an international cetacean post-mortem screening program. Such a program would standardize protocols to test for a panel of infectious agents (including *Trypanosoma* spp., but also others like *Brucella*, morbillivirus, and biotoxins) in every mass stranding event. Data should be shared in a global repository. With sufficient samples, scientists could analyze patterns: Do trypanosome-positive strandings cluster geographically (e.g., near tsetse-rich coasts)? Are they more common in certain seasons (rainy seasons when tsetse are more abundant)? Do they predominantly affect certain species or age classes of whales? Answering these questions would either strengthen the case that trypanosomiasis is an important factor or reveal it to be a rare oddity.

Additionally, experimental research is warranted. Can *T. brucei* survive in saltwater for any length of time? Could a marine leech transmit *T. brucei* from one host to another? Are there unknown "marine trypanosomes" adapted to oceanic hosts? Modern molecular techniques may uncover novel trypanosome species in marine animals that have long been misclassified or overlooked. For example, the "novel *Trypanosoma*" found in gray whales should be genetically sequenced and compared to terrestrial strains – it could be a previously unknown lineage, hinting at a long history of these parasites in the ocean.

From a conservation perspective, if trypanosomiasis is found to contribute to whale mortalities, it adds a piece to the puzzle of human impact on oceans. Human activities that affect vector distributions (like land use changes) or bring livestock closer to the sea could inadvertently increase disease spillover to marine life. Mitigating such risks might involve creating buffer zones (e.g., maintaining natural vegetation barriers at the land–sea interface to reduce fly crossover) or treating livestock for trypanosomes in coastal farms to lower the reservoir of infection.

In conclusion, the hypothesis that whales may succumb to "sleeping sickness" is no longer purely fanciful – it is a testable hypothesis emerging from an intersection of fields. It reminds us that the natural world's systems are deeply interwoven: a tiny protozoan in a fly's proboscis might influence the fate of a 40-ton whale. By reevaluating unexplained strandings through this lens, we can advance both science and conservation. Perhaps in the near future, the sight of stranded whales will trigger not just a rescue response, but

also a rigorous diagnostic work-up for pathogens, ensuring that these mysterious events gradually yield their secrets. Such knowledge will empower us to better protect whales, those sentinels of ocean health, in an era of unprecedented environmental change. The next time a mass stranding makes headlines, "Could it be trypanosomes?" should be one of the questions we earnestly pursue – the answer could deepen our understanding of life and death in the sea.

Endnotes

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