SHODH SAGAR®
International Journal for Research Publication and Seminar
ISSN: 2278-6848 | Vol. 15 | Issue 2 | Apr-Jun 2024 | Peer Reviewed & Refereed

# Effect of Marine Phycotoxins on Pediatric Neurological Health

**Srihari Padmanabhan**
Independent Researcher,USA.

**Ojaswin Tharan**
Independent Researcher,USA.

**DOI:** https://doi.org/10.36676/jrps.v15.i2.1551

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Published: 2024-06-30

* Corresponding author

**Abstract**

Phycotoxins are toxic substances which are generated by varieties of algae; marine phycotoxins are particularly dangerous to the neurological welfare of children. The types of marine phycotoxins, their neurotoxic action, and the management of exposure in children are all examined in this paper. It is just in this case that the patient undergoes early evaluation of signs of a chronic illness, and the prevention of the worsening of the condition and its impact on the neurological system. In total, the present research aims at analyzing the short and the long-term effects of the mentioned toxins with the purpose of providing a broad look at the risks they present to neurodevelopment of children.

**Keywords**

marine phycotoxins, paediatric neurology, neurotoxicity, HABs, neurodevelopment, toxins.

## 1. Introduction

### 1.1 Background

Marine phycotoxins are also known as algal toxins, natural products synthesized by algae during the phenomena known as toxic algal blooms. These toxins which mostly originate from toxic seafood are serious threats to the health of human beings especially the kids. The nervous system of children appears to be very sensitive to phycotoxins and this results to neuro developmental problems and perhaps permanent damage. This paper aims to familiarize the reader with the sources, types, and modes of phycotoxin contamination and their adverse effects on children's neurological system particularly and the developing an exigent necessity for the prevention of exposure to phycotoxins and the development of therapeutic methods.

**1.2 Research Objectives and Scope**

1. To assess the metabolites of marine phycotoxins and the different routes by which children come into contact with them.
2. To evaluate the neurotoxic impacts of certain marine toxins – phycotoxins – on the neural tube formation in children.
3. To assess the exogenous parameters that facilitate the occurrence of marine phycotoxins in food products and water sources.
4. To find out early indicators/biomarkers of neurotoxicity due to exposure to phycotoxin from marine sources in children.
5. To discuss possible approaches to controlling children's exposure to marine toxins or to define the most appropriate policy actions to undertake.

This research will be based on many marine phycotoxins, from which attention is to be paid to the neurotoxic effects of the chemicals like saxitoxins, brevetoxins, domoic acid etc. This study will incorporate both primary (ingesion of contaminated seafood) and the secondary routs (through environmental contact) to the pediatric population because of their brain susceptibility during development. The study will also ascertain current developments on marine phycotoxin blooms, influences affecting toxin, public health measurers for protection of children against phycotoxin.

**2. Theoretical Framework**

Research into marine phycotoxins as well as their impact on neurological development in children can only be completed through an environmental toxicology, developmental neurology, and public health research point of view. This theoretical framework will posit plausible conceptual constructs from these fields that will inform the nature, processes and outcomes of marine phycotoxin exposure. It also incorporates the models of environmental risk factors and neurodevelopmental vulnerability and regulatory frameworks already developed to create a framework for the analysis of the impact of phycotoxins on the children.

**2.1 Environmental Toxicology and Marine Phycotoxins**

Marine phycotoxins are secondary metabolites of toxicity functional compounds that are formed by HABs in marine environments (Pulido, 2016). These toxins are saxitoxins, domoic acid and brevetoxins, which enter the marine food chain and contaminate seafood (shellfish) that is harmful to human beings especially the young children. The Environmental Risk Theory also assists in describing how phycotoxins as toxins in the marine ecosystem turn into risks to health by means of the ecological and human significance routes.

Marine phycotoxins are most widespread in regions with water temperature, nutrient enrichment and coastal water inputs that promote HABs occurrences and magnitude. An exposure pathway related to interaction of these toxins with marine organisms is through bioaccumulation of toxins in seafood that gets into the marine food chain in this manner. Risk Assessment Models are essential for estimating the probability of exposure to marine phycotoxins including children who are most likely to suffer from neurotoxicity impacts due to a young brain.

*Table 1 Common Marine Phycotoxins, Sources, and Their Neurological Impact on Pediatrics*

| Phycotoxin | Source | Neurological Impact | Exposure Route |
|---|---|---|---|
| Saxitoxin | Dinoflagellates (Alexandrium spp.) | Paralytic Shellfish Poisoning (PSP) | Contaminated Shellfish |
| Domoic Acid | Diatoms (Pseudo-nitzschia spp.) | Amnesic Shellfish Poisoning (ASP) | Contaminated Fish, Shellfish |
| Brevetoxins | Karenia brevis | Neurotoxic Shellfish Poisoning (NSP) | Inhalation, Contaminated Seafood |
| Ciguatoxins | Gambierdiscus spp. | Ciguatera Fish Poisoning (CFP) | Contaminated Fish |

Minimizing the impact of environmental risks of phycotoxins in the aquatic ecosystem is informed by the Precautionary Principle. This principle presupposes that action should be needed at least when and if some harm might be done although science cannot or will not say so especially as concerns vulnerable populations and individuals like children. This paper clearly shows that there is need to set regulatory measures to reduce levels of phycotoxins in the marine ecosystem, and several countries already have systems of monitoring phycotoxins in seafoods. However, due to inconsistency in the rules governing e-commerce around the globe, fail in enforcement and implementation affords people from one region a lesser chance of being exposed compared to those from another region.

**2.2 Developmental Neurology and Vulnerability of Children**

This is because there are critical years in the development of the human brain, and children are likely to be harmed by neurotoxins more than others. The Developmental Neurotoxicity (DNT) Model serves as one of the ways to explain the link between toxicants and impairments of a child's developing brain by marine phycotoxins. Children are especially at risk for neurotoxic effects since their brains rapidly develop during childhood, and neurotoxins can alter structural and functional organization of the brain leading to cognitive, behavioral and motor problems.

Marine phycotoxins such as saxitoxins and domoic acid have an effect on ion channel and neurotransmitter receptor causing excitotoxicity and neurons lesion (Visciano et al., 2016). These interactions in children especially their growing nervous systems cause long and permanent effects. The Vulnerability-Stress Model reminds that children's exposure to neurotoxins is aggravated by stressors such as economic disadvantage, poor diet, and no or limited medical attention.

*Table 2 Phases of Pediatric Brain Development and Potential Impact of Phycotoxin Exposure*

| Age Range | Brain Developmental Phase | Potential Impact of Phycotoxins |
|---|---|---|
| Prenatal – 2 years | Rapid neuronal proliferation | Disrupted cell division, impaired brain growth |
| 3 – 5 years | Synaptogenesis and myelination | Altered synapse formation, excitotoxic damage |
| 6 – 12 years | Refinement of neural networks | Cognitive impairments, behavioral changes |
| Adolescence | Synaptic pruning, maturation of PFC | Long-term neurological deficits, memory loss |

During these critical development periods phycotoxins can be particularly hazardous and require further investigation into allowable levels of contamination in foodstuffs for children. Some precautionary existing intervals of marine toxins are in fact derived from adult values leaving the young very vulnerable. As the Neurodevelopmental Plasticity Theory has it, the human brain is indeed very flexible during development but at the same time very vulnerable to being damaged systematically and with no possibility of recovery by toxic substances. Therefore, relatively small levels of chronic exposure to phycotoxins may lead to very large impacts on children.

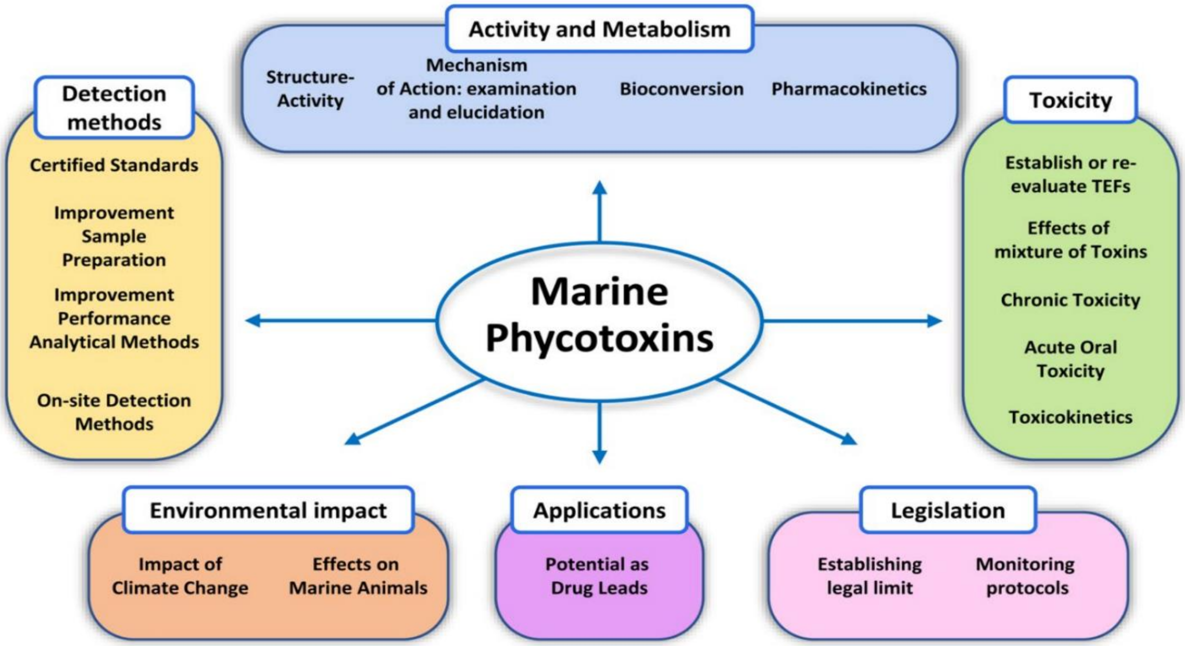


*Figure 1 Current Trends and New Challenges in Marine Phycotoxins (MDPI, 2021)*

**2.3 Public Health and Regulatory Response**

The theoretical framework also uses Public Health Theories concerning the environmental justice and health inequity. Companies producing marine products with high levels of phycotoxins have their production located in the coastal, less developed regions where the health requirements of the population are lower. The SDOH including income, education and health care insurance force that determine the neurotoxic exposures found in this study (Cifuentes et al., 2015). These inequalities have to be redressed by the regulatory agencies for instance the children aspect which is vulnerable in the society should be given the priority in monitoring and establishing programs that control marine toxins.

The One Health Approach is important in the fight against the complexities of human, animal and ecology concerning marine toxins. Since it is possible to prevent the conditions that lead to hazardous algal bloom through other approaches whose effects reduce human interferences such as nutrient loading and over fisking. Second, Risk Communication Models assert that affordances for the publicity of information on the risks accompanying ingestion of phycotoxin are fit for release for consumer, especially those with children.

The presented theoretical framework offers an integrated perspective on the pathways through which marine phycotoxins impact pediatric neurological conditions, the increased susceptibility of children to those toxins, and corresponding public health and legislative actions that should be taken to reduce the identified risks. This work seeds a theoretical foundation with the environment toxicology, developmental neurology, and theories from public health that points to the way toward understanding and meeting the very difficult problem of marine phycotoxins in children.

**3. Types and Sources of Marine Phycotoxins Impacting Pediatric Neurology**

Marine phycotoxins are poisonous substances that are found in different species of marine algae which can pose severe health effects once ingested and are considered worst in children. These toxins are accumulated in the food chain mainly through shell fishes, fishes and other sea food which in turn feed on the algae yielded the toxins. In the field of pediatric neurology, key knowledge about marine phycotoxins is the classification and their sources because the listed toxins affect the BBB and cause severe neurological outcomes, including permanent damage. Consumption of toxins happens trough seafood but aerosolized toxins may be inhaled especially in areas influenced by HABs.

For instance, increased formation of new algal forms of dinoflagellate Alexandrium has led to the production of saxitoxin, the most toxic phycotoxin implicated in paralytic shellfish poisoning (PSP). Saxitoxin inhibits voltage gated sodium channels in neurons thereby interfering with normal conduction of impulses, depending on the level of poisoning, the victim may just experience mild tingling sensation or paralysis of respiratory muscles and failure. As children possess relatively small bodies as well as developing nervous systems, they are almost three and five times susceptible to saxitoxin in comparison with adults even to low levels of exposure. Ingestion of tainted shellfish results in neurological manifestations of a serious nature and may warrant an early evaluation by a medical practitioner (Vilariño et al., 2015).

Another great phycotoxin is domoic acid that is generated by diatoms including Pseudo-nitzschia. This is related to amnesic shellfish poisoning and what it does is to bring about dysfunction in the hippocampus which functions in areas to do with memory and learning within the brain. Domoic acid fixates into the glutamate receptor which is an excitatory receptor and cause excitotoxicity and neuronal deaths. For pediatric population, the residual neurological impact of domoic acid is more dangerous indeed minor cases also leads to learning disability and memory loss. In contrast to PSP for which signs and symptoms are more evident and abrupt, domoic acid may induce both primary and secondary toxic encephalopathy, and both may manifest within the acute phase.

Brevetoxins associated with red tides causing harm by Karenia brevis end up in neurotoxic shellfish poisoning (NSP). Such toxins impact on both peripheral and central nervous system and its manifestations include nausea, vomiting and paresthesia. As a neurotoxin, brevetoxins can also be aerosolized and inhaled making coastal communities including children vulnerable during red tide events. Inhalation exposure causes respiratory distress and worsening of neurological status in people with epilepsy. While the neurotoxicity of brevetoxins especially to pediatric patients has not been fully established, there is high possibility that children are highly vulnerable to environmental toxins.

*Table 3 Key Marine Phycotoxins, Sources, and Pediatric Neurological Impact*

| Phycotoxin | Source | Neurological Impact on Pediatrics | Exposure Route |
|---|---|---|---|
| Saxitoxin | *Alexandrium* spp. | Paralysis, respiratory failure, death | Contaminated shellfish |
| Domoic Acid | *Pseudo-nitzschia* spp. | Memory loss, learning disabilities, excitotoxicity | Contaminated shellfish |
| Brevetoxins | *Karenia brevis* | Respiratory distress, neurological impairment | Inhalation, contaminated seafood |
| Ciguatoxins | *Gambierdiscus* spp. | Sensory disturbances, paralysis, long-term neurological deficits | Contaminated tropical fish |

Gambierdiscus species are responsible for ciguatoxins that lead to CFP through ingestion of tropical reef fish. This neurotoxin presents gastrointestinal symptoms through to more serious neurological manifestations including: sensory changes (cold allodynia); signs of muscle paralysis; and cardiovascular manifestations (Pulido, 2014). Therefore, ciguatoxins are most toxic to children mainly because they influence the cells of the nervous system and have potential irreversible neurological effects. CFP is the most widespread type of seafood poisoning throughout the world and remains a constant threat to people in tropical and subtropical areas.

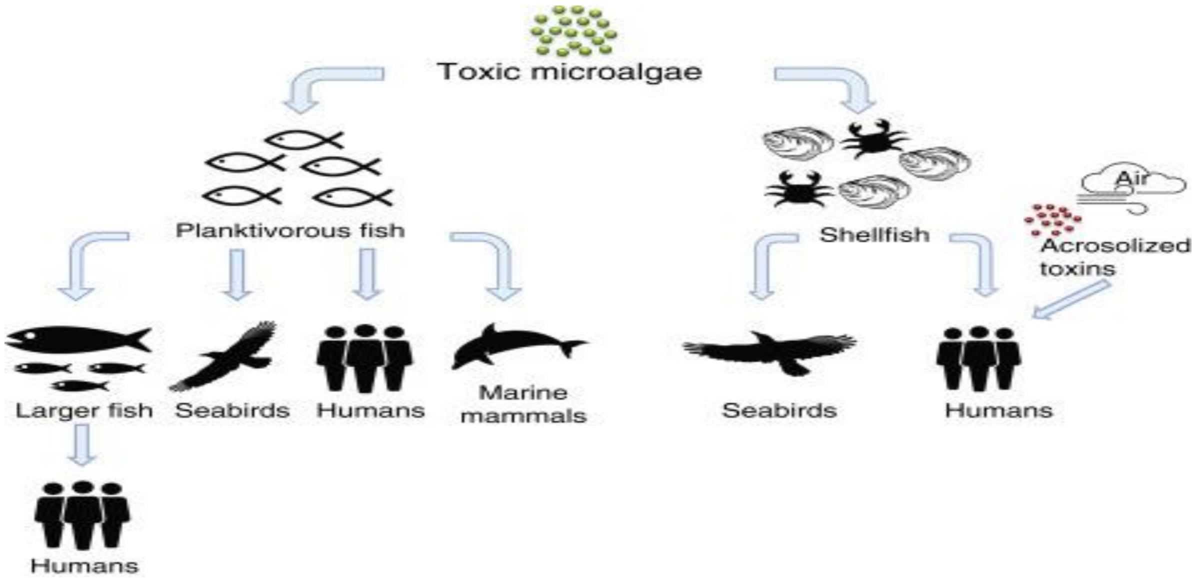


*Figure 2 Marine-Based Toxins and Their Health Risk ( ScienceDirect, 2021)*

Sources of these marine phycotoxins are well known though the specific sources differ from one zone to another and are commonly related to HAB. HABs are evident today due to such things as climatic change, eutrophication and so on, which affect the coastal regions. These advanced toxigenic algal blooms widened the area that phycotoxins occur and increased the frequency of toxin incident. This is more so in children who reside in coastal regions where fish forms part of their staple diet and other children in areas where fishing is done for household consumption. Screening for seafood safety and the management of such risks are important tenets of ensuring that vulnerable children are protected from these neurotoxins.

**4. Mechanisms of Neurotoxicity in Pediatric Populations**

Neurotoxic effects in children exposed to marine phycotoxins are functional complex and diverse in pediatric populations. The toxins originating from hazardous algae known as phycotoxins are capable to disrupt the normal neurological development and also functioning. Since the development of children's nervous systems is still in progress, they are pronounced targets for the toxic impact of these agents (Zhu et al., 2024). Marine toxins usually known as phycotoxins including saxitoxin, domoic acid, brevetoxins and ciguatoxins which affects different neurological system, both peripheral and central having negative impacts.

One of the major processes of neurotoxicity is the effect of marine phycotoxins channel function in neurons. For example, saxitoxin which is the main toxin associated with the paralytic shellfish poisoning (PSP) acts a sodium channel inhibitor. In normal neurons, sodium channels are involved where they give the action potentials that transmit impulses across neurons. It binds to these channels in such a way that sodium ions can be excluded from the neuron and that propagation of action potentials can be stopped. Therefore, nerve impulses are abolished, and the common signs are tingling, numbness, paralysis and in the worst cases; respiratory failure. While receiving low dosages, young patients with relatively small statures and developing nervous systems can develop severe lethal toxicological reactions. The inhibition of sodium channels is especially problematic to children because young peoples' nervous systems are most vulnerable to the changes in electrical conductance, which can ultimately result in various developmental problems.

One of the ways that phycotoxin induces neurotoxicity is as follows; they act as antagonizers of the neurotransmitter receptors such as excitement of glutamate receptors that includes NMDA receptors by toxins for instance domoic acids. Domoic acid, implicated in amnesic shellfish poisoning (ASP), is an antagonist of both kainate and AMPA receptors as well as an agonist of the metabotropic glutamate receptors. Glutamate is the most broadly used and important excitatory neurotransmitter of the CNS and proved to be involved in synaptic plasticity, learning, and memory. However, if the glutamate receptors are over excited, for instance when given domoic acid, the cells in the brain end up being over stimulated to the extent of damaging themselves. This excitotoxicity kills the neurons in the areas that contain many neuron cells in the brain for example the hippocampus area important in memory. This is the case because in populations of pediatric age the developing brain is more susceptible to excitotoxicity. In general, domoic acid is neurotoxic and has been shown to cause complications to the developing nervous system as well as inflict long term memory, learning and behavioral deficits when the subject is exposed to large concentrations for several hours or was exposed continuously.

Neurotoxic shellfish poisoning is the result of eating shellfish containing the toxins called brevetoxins; these interfere with normal neuronal signaling via voltage-gated sodium channels. Thus, opening of these channels is not illustrated in case of saxitoxin, but in cases with brevetoxins the sodium channels remained opened, hence sodium ions continue to flow into neurons in a persistent manner (Lee et al., 2024). This kind of depolarization of the neuron continues for quite a long time, which leads to the release of neurotransmitters in an uncontrolled way, thereby resulting in neuronal hypeactivity. In paediatrics, brevetoxin over-stimulatory effects on neurons can cause symptoms as mild as gastroenteritis and respiratory problems, to severe neurotoxic signs, including convulsions. Bretoxins can aerosolize during HABs and these toxins may be inhaled by children with residence in coastal areas. Pet exposure to brevetoxin-containing aerosols may cause respiratory illness and exacerbation of neurological disease after brevetoxin exposure, especially asthma or epilepsy in children.

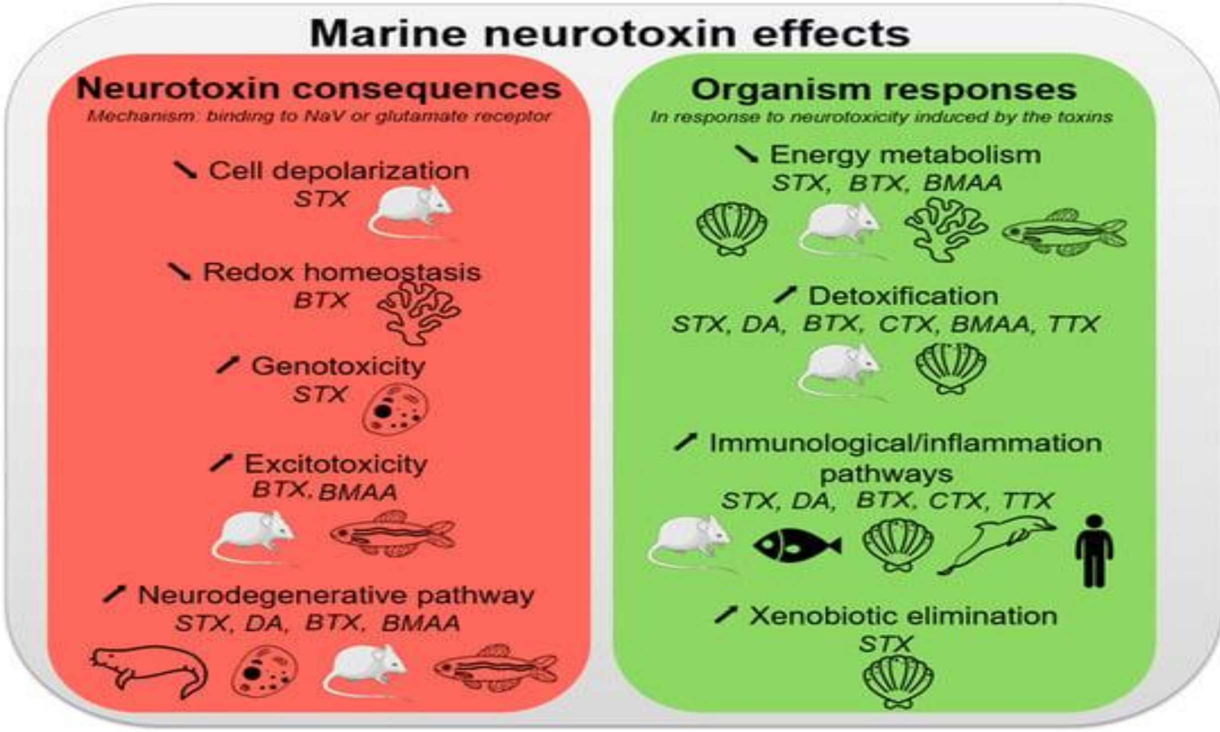


*Figure 3 Marine Neurotoxins effects on Environmental and Human Health (MDPI, 2021)*

Ciguatoxins which induce ciguatera fish poisoning are yet another method of neurotoxicity populist in pediatric patients. These toxins, obtained from Gambierdiscus species, work by depressing the threshold for action potential in neurons making nerves more sensitive. Such changes can cause abnormally sensitized touch and movement sensations — featuring a key sign of neuropathic neuropathy, cold allodynia, where subjects report painful hotness in response to cool temperatures. In children, ciguatoxin exposure can result in pathological alterations of sensory systems and motor coordination and may result in delayed neurological sequelae. In addition, ciguatoxins have a long half-life and thus children affected by these toxins would continue to relapse from neurological poisoning for quite some time even after the exposure had taken place (Mafra et al., 2023). This persistence presents a particular problem in children, for whom the nervous system is still vulnerable and capable of being easily influenced by pathological changes.

The studies show that there is a mechanism known as blood-brain barrier or BBB, which regulates the amount of neurotoxicity by preventing deleterious substances from affecting the brain in pediatric patients. In young children the barrier between the blood and the brain, the BBB, is not completely set up and is therefore less able to keep out toxins such as marine phycotoxins. Therefore, the load of these neurotoxic compounds in the brains of pediatric patients is considerably higher than that admitted to adults because of the opening factor, which, in its turn, results in considerably worsening neurological outcomes. First, the raised permeability of the BBB in children, the relatively small size, and developing neurological connections increase the neurotoxicity of marine phycotoxins, which means early exposure to the toxins is safer for kids.

Another worrying aspect of neurotoxicity in pediatric populations is the question of interactions with other toxic agents. Cumulative effect of low doses of marine phycotoxins, absorbed on regular basis during early stages of development of the organism, appear to intensify the adverse consequences on the developing brain. this is the case with neurotoxicants such as domoic acid and the brevetoxins where sub lethal effects lead to low level, chronic damages with respect to learning ability, motor coordination and behavior. These toxins could have developmental delay, learning disability and other neurological complications implications on children who come from such backgrounds especially that many from the coastal area have a steady take of seafood.

**5. Therapeutic Approaches and Risk Mitigation Strategies for Phycotoxin Exposure**

This is recover/management therapeutic option in children involved either short term/pharmacological intervention or long term/risk management of neurotoxic consequences of phycotoxin exposure. Because phycotoxins show a wide variety of actions, sodium channel blockade, excitotoxicity, and sustained neuronal activity are suitable strategies for therapeutic intervention.

For initial treatment, the re-administration of antitoxins or drugs that help stabilize the neural ion channels is essential. The first is saxitoxin, a presynthetic-ABA, which affects the sodium channel and paralyzes the respiratory muscles; there is no specific treatment, but patients require support with respiration (Daguer et al., 2018). In cases of toxins where excitotoxicity is said to be the main mechanism, such as domoic acid, use of Glutamate antagonists may be of value due to be preventive of overstimulation of the glutamate receptors. It was revealed that intravenous fluids and management of electrolyte imbalance may help to lower the toxin levels in these children and thereby reduce possible neurological complications.

Measures against risks of exposure to phycotoxins should be put in place especially in the regions where Episodes of HABs are experienced. Marine toxins in seafood can be prevented by monitoring devices such as public health surveillance systems in giving advisories. Incorporation of early warning systems, particularly in the HAB affected coasts in combination with awareness creation regarding likely adverse effects of consumption of contaminated seafood. Studies have shown that children must be informed or educated on safe consumption of seafood especially the vulnerable pediatrics in the coastal regions.

At a policy level, it is recommended that; enhanced policies to be enacted by these governments especially in regions where climate change and nutrient runoff are contributing factors to HABs occurrence. Studies suggesting the production of other neurotherapeutics including those which could reduce neurodevelopmental effects of phycotoxin in children should be carried out to avoid long term complications on the development of children.

**6. Future Direction**

There is potential for future investigations to identify new neuroprotective treatments for marine phycotoxins and pediatric neurology to be researched also the low dose chronic effects of marine phycotoxins (Otero et al., 2022). The use of newer generational screening and real time monitoring tools for detecting phycotoxins in marine ecosystems presents a realizable way of minimizing exposure to the toxins. They concluded that future research should be aimed at identifying whether dangerous algal blooms are directly linked to climate change and, therefore, whether there may be risks in the future. Moreover, timely incorporation of educational programs in the coastal areas, and various collaborations important for aspects of regulation policies will strengthen the overall prevention as well as intervention.

**7. Conclusion**

Many marine phycotoxins have been shown to impact pediatric neurological health in the acute and chronic sense and neurological development. The overview of this paper has focused on the types of phycotoxins, neurotoxic effects they cause, and the methods of managing exposure to such toxins. According to the sources, there is need to maintain very high standards of preventive action such as surveillance and public health interferences so that children are protected from such environments. Thus, in identifying the biological and environmental correlates of exposure to phycotoxins, this investigation supports the further importance of intervention initiatives in management of the threat.

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