



The cyanotoxins occurrence in water and fresh foods: human health implications

La presencia de cianotoxinas en aguas y alimentos frescos: implicaciones para la salud humana

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Abstract

The cyanotoxins occurrence in water, and their incorporation into the food chain have caused numerous reports of health damage. This study presents a systematic-critical review on the presence and implications of cyanotoxins in water and fresh foods, considering their potential impact on human health. The applied methodology corresponds to a critical-narrative analysis of research published in institutional repositories and high-impact databases (PubMed, Crossref, Google Scholar, Scopus) considering the last 10 years of documentary validity. The results demonstrate a recognition of the impact of human activities and climate change on the increasing incidence of cyanotoxins on human well-being, with negative implications, related to gastrointestinal symptoms, liver conditions and damage to the nervous system, the impact being relevant. of microcystins and cylindrospermopsins. Emphasis is placed on the need to obtain precise data on the toxicological charge in both water, biomass, and fresh foods, to establish the pertinent restrictions in order to provide health guarantees. Governments must take measures to prevent the risk associated with the presence of cyanotoxins, with training and capacity building for research and management being necessary in vulnerable contexts. Mitigation of the impacts of cyanotoxins must be treated from a communication and instructional point of view. It is important to develop awareness campaigns to improve perception of this emerging risk, which often compromises the lives of human beings.

Keywords

cyanotoxins, foods, health damages, toxicity, water

Resumen

La presencia de cianotoxinas en las aguas y su incorporación a la cadena trófica, han causado numerosos reportes de daños a la salud. Este estudio presenta una revisión sistemático-crítica sobre la presencia e implicaciones de las cianotoxinas en aguas y alimentos frescos, considerando su impacto potencial para la salud humana. La metodología aplicada corresponde a un análisis crítico-narrativo de investigaciones publicadas en repositorios institucionales y bases de datos de alto impacto (PubMed, Crossref, Google Académico, Scopus) considerando los últimos 10 años de vigencia documental. Los resultados demuestran un reconocimiento del impacto de las actividades humanas y el cambio climático en la incidencia cada vez mayor de las cianotoxinas en el bienestar humano, con implicaciones negativas, relacionadas con síntomas gastrointestinales, afecciones hepáticas y daños al sistema nervioso, siendo relevante el impacto de las microcistinas y cilindrospermopsinas. Se hace énfasis en la necesidad de obtener datos precisos de la carga toxicológica tanto en agua, biomasa, como en alimentos frescos, para establecer las restricciones pertinentes en función de dar garantías de salud. Los gobiernos deberán tomar medidas para prevenir el riesgo asociado a la presencia de cianotoxinas, siendo necesarias en aquellos contextos vulnerables, la capacitación, y la formación de capacidades para la investigación y la gestión. La mitigación de los impactos de las cianotoxinas debe ser tratada desde el punto de vista comunicacional e instructivo. Es importante desarrollar campañas de sensibilización para mejorar la percepción sobre este riesgo emergente, que en muchas ocasiones compromete la vida de los seres humanos.

Palabras clave

agua, alimentos, cianotoxinas, daños a la salud, toxicidad.

1. Introducción

Since their origin, cyanobacteria have had an impact on life, either due to their significance in generating the oxygenic atmosphere of Earth, forming the basis of the diet of various peoples, or their ability to produce toxins that affect the ecology of water bodies where they massively develop, with socio-economic implications [1].

Health issues caused by the effects of cyanotoxins result from changes in the environment of cyanobacteria, which induce alterations in their organic composition, stimulating the production of highly harmful molecules capable of affecting living organisms, with serious environmental implications and economic repercussions for societies [2]. To date, more than 50 genera of cyanobacteria with toxic potential have been described, making it relevant to understand their dynamics in ecosystems, triggering factors, their presence in water and food, and the consequences of

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exposure to different concentrations of these cyanotoxins, as all of these are associated with risks to human life [3]. In this regard, the One Health approach considers their impact on the human trophic web and their entry routes, necessitating the responsible handling of potable water and fresh food with high toxic loads [4]. Responsible production and consumption of food are essential to ensure the health of humans and animals, as well as the long-term health of the environment. Without good practices along the food value chain, food can become a major vehicle for the transmission of microbiological and chemical hazards [5].

Foodborne diseases are caused by the consumption of contaminated food and encompass a wide group of diseases caused by enteric pathogens, parasites, chemical contaminants, and biological toxins. These diseases reduce societal productivity, impose substantial pressure on the healthcare system, and reduce economic output due to decreased consumer confidence, food losses, and disruption of access to domestic and export markets, affecting trade and tourism, and threatening food security [6].

Especially populations that do not properly manage the risks caused by constant interaction with cyanotoxins; do not have or do not apply regulations, do not control established maximum permissible limits, or do not even pay attention to this issue, will have a latent cause of natural health damage in these toxic events [1]. This study focuses on systematizing scientific literature on the health impacts of cyanobacteria due to the presence of cyanotoxins, specifically in freshwater bodies and contaminated fresh foods, all being publicly accessible resources essential for sustaining life, development, and social interaction. However, it is important to consider that the ubiquity of these microorganisms makes any ecosystem and food vulnerable.

Moreover, this work allows a recap of key aspects related to the presence of cyanotoxins, including management initiatives during risk events, to safeguard the integrity of living beings, with an emphasis on human health; focusing on documenting triggering factors, present toxins, associated cyanobacteria species, and the main health impacts on humans after exposure to toxicological elements, such as a conflicting experience due to chronic exposure, and the presence and/or accumulation through the food chain.

2. Materials and Methods

The methodological management implemented for the development of this study is based on a narrative review of research published in high-impact institutional repositories, as well as relevant academic publications on the topic. To obtain the documents, a general technical search was conducted using scientific databases of interest: Crossref and Google Scholar, with specific descriptors; in addition to targeted searches on PubMed, Scielo, and Scopus,

considering authors previously identified for their contributions to the topic. The publication limits consider the last 10 years of documentary validity, which was delimited with the intention of obtaining a completely updated source of information on the implications of the presence of cyanotoxins in water and fresh food. The descriptors or keywords considered were: cyanotoxins, toxic cyanobacteria, including health risks as a complementary phrase.

Publications in Spanish, English, and Portuguese were included, corresponding to indexed articles, theses, books, and scientific reports. Once the information was recapitulated, a semi-structured analysis of the contributions of the research was carried out, regarding the impact of cyanotoxins on human health, and the management of drinking water and food with toxicological loads, considering the One Health approach.

3. Cyanotoxins and Their Conflict Relationships in Societies

The proliferation of cyanobacteria in water and food is largely due to anthropogenic effects such as eutrophication and even the presence of industrial and agricultural pollutants, as well as issues related to domestic sanitation [7], without minimizing the effects of climate change. Urban and sociodemographic expansion promotes a considerable environmental impact with a direct climate repercussion by overwhelming the structural sanitation of large cities [8]. The discharge of wastewater that has not found new territorial fractions for proper treatment, especially in the mass production of food, directly tied to the need to meet consumption demands, simply increases the indiscriminate elevation of harmful elements for health with high loads of chemicals such as nitrogen and phosphorus [9]. This leads to secondary events such as the development of phytoplankton blooms, which extend to reservoirs that supply water, later used for the operational development of various activities, notably agroindustry [4]. The situation becomes a vicious cycle produced by the lack of good practices and the failure of control mechanisms and management models.

The impact of cyanobacteria blooms and the presence of cyanotoxins has been demonstrated [10], in most cases being associated with a high occurrence of specific health problems, driven by conflictive organisms that in certain proportions can cause gastrointestinal, dermatological, and even neurological system disorders as one of their main consequences [11].

According to Almeida [12] certain species of cyanobacteria in the animal kingdom can produce cyanotoxins, although they are not sufficient causes to affect the health of living beings if interacting with a tolerable load of toxins; however, once present, they constitute an imminent danger. Even so, industrial activities facing all these natural ecosystem conditions intensify the production of



cyanotoxins, causing liver damage, cytotoxicity, and even neurotoxicity in part of the population [13] and in many cases, primary healthcare is not culturally prepared to face the situation, associating it with other toxic agents; this is more noticeable in places where risk perception is null or low.

In the case of exposure, it not only includes the consumption of intoxicated animals through the trophic chain or contaminated water, but there are also cases where the development of recreational activities in water bodies is an influential contact factor for the impact of cyanotoxins on health[2], [14].

Evidently, states that do not properly manage and address the liquid effluents of water bodies contribute to the increase of toxicological loads [15] through eutrophication and, consequently, the occurrence of cyanobacteria blooms [8]. In Latin America, for example, phytoplankton blooms in reservoirs are a recurring problem that influences the effective management of drinking water, even altering its properties by not using effective technology to eliminate cyanobacteria and cyanotoxins, a situation that worsens after its supply [13].

4. Cyanotoxins: Types and Effects on Human Health

Cyanobacteria toxins are harmful components to living organisms; they comprise a heterogeneous group of chemical compounds with high toxicological loads and multiple metabolic representations [8]. According to the findings of Menescal [15] these substances can develop in all phases of cell growth, as they are released when cyanobacteria cells break (lysis), remaining in the water for many weeks, depending also on environmental conditions. Although the productive occurrences of these toxic substances have not been clarified by science, a plausible hypothesis is their role in herbivore protection, where not all cyanobacteria blooms are toxic [16]; however, they can be harmful.

Regarding toxicological classification, cyanotoxins can be: 1) hepatotoxins, capable of causing liver injuries due to morphological and functional alterations in hepatocytes, promoting autophagy and cell proliferation depending on the amount and duration of exposure; 2) neurotoxins, which cause lethal acute intoxication and interfere with nerve impulse connections, potentially causing muscle paralysis and subsequently respiratory issues; and 3) dermatotoxins, generally not lethal but manifesting with high irritation and inflammatory process alterations in the body [12].

Therefore, understanding the applied action of hepatotoxins and neurotoxins revolves around intoxication, where science strives to effectively treat these compounds to avoid health consequences [17]. Dermatotoxins are also important cases of intoxication in swimmers in coastal waters, highlighting debromoaplysiatoxins and lyngbyatoxin-a

[10]. Additionally, there is a segment called lipopolysaccharide (LPS), which plays a significant role in toxicology [11]. Lipopolysaccharide (LPS) or endotoxin is the major component of the outer membrane of Gram-negative bacteria, playing an important role in activating the immune system by constituting the most important surface antigen of these bacteria. LPS is composed of a lipid and a glycosidic region with separate and/or synergistic functions, making this molecule one of the most complex virulence factors to understand [18].

Animal experiments are noteworthy for validating the behavior of the mechanism of action of cyanotoxins; in this regard, there are multiple deviations that demonstrate direct risks to humans as they are completely different matters [14]. Moreover, there is a latent health risk that transcends into alterations attributable to algal blooms, whether from drinking water sources, consumption of contaminated food, direct interaction in coasts or rivers usually used for recreation, as well as other characteristic factors such as age or pathological history [19].

Cyanotoxins have been studied for their impact on drinking and recreational waters. Paineñilú [8] in his report shows that recreational exposure is linked to mild and self-limiting symptoms that do not require medical consultation; in contrast, Miglione et al. [20] clarifies that the nonspecific symptomatology of these situations can lead to a misdiagnosis, with cases reported in his study frequently directing to gastrointestinal incidents, irritative factors in the eyes or respiratory tract, and dermatological and pulmonary symptoms, where scientific evidence shows that the most severe are hepatic types.

Recapping health effects, historically, the first case of cyanotoxin intoxication was from interacting with contaminated waters of the Ohio River in the United States in 1931. Subsequently, in 1959, a recreational exposure occurred in Saskatchewan, Canada, noted as one of the 10 provinces among the 13 federal entities of this country that demonstrate its incidence since the last century [14].

In Latin America, the first acute microcystin intoxication with demonstrated hepatotoxicity in individuals was due to water contamination, affecting 116 people, associated with recreational exposure within the Uruguay River reservoir in 2007 and later on the beaches of the Río de la Plata in Montevideo in 2015 [14]. Other intermediate events have been recorded.

Thus, direct exposure occurs in areas with higher population density, which in most cases manifests as health alterations, either acute or chronic [9], always due to accidental ingestion of resources such as contaminated water and food, skin contact, and even indirect inhalation of cyanotoxins that can remain suspended in tiny aerosol droplets [21]. Therefore, determining the incidence of these components



in people with prolonged contact is a precedent for preventing health alterations [22].

In this regard, multiple cases of diseases in humans and sentinel animals have been documented due to contact with contaminated water and fresh food, with incidences of liver cancer, as in some regions of China, and even death from direct contact with microcystins [23]. The most dangerous intoxication was documented in 1996 in Brazil, where 100 out of 131 individuals on dialysis developed acute liver failure due to the presence of cyanotoxins in the water; 52 of these people died [23]. In summary, all research demonstrates that understanding the effects of cyanotoxins is of great interest for maintaining and balancing public health, and if not considered, can lead to significant mass consequences, historically affecting a broad segment of the population according to the situational context where these intoxication events occur.

5. Vulnerability to the Impact of Cyanotoxins

Urbanization efforts, in line with the increase in industrial, agricultural, and livestock activities, have a significant impact on environmental pollution. Water resources directly affect human health [24]. Consequently, exposure to certain environmental contaminants can even lead to neurodegenerative diseases [9].

Various human activities in urban and rural areas generate nutrient accumulation; this organic load promotes eutrophication, increasing bacterial contamination that can disrupt trophic networks [25] and compromise the quality of fresh food from aquatic ecosystems. Eutrophication leads to the uncontrolled growth of algae and photoautotrophic bacteria, namely cyanobacteria [19]. Blooms increase the biomass of cyanobacteria; the loose masses that form can also cause problems in aquatic ecosystems [9].

According to Federici et al. [26] 80% of cyanobacteria blooms in continental waters are toxic, affecting multiple species. For instance, the deaths of cattle, dogs, horses, and even large animals like the 300 elephants that died in Botswana in 2020 were attributed to neurological problems after consuming water contaminated with cyanotoxins [26].

Given these conditions, it is prudent to reference the arguments of Condor and Feliciano [10] who highlight the existence of a great diversity of toxins produced by different genera of cyanobacteria, whose production also depends on environmental factors such as nutrients and temperature, among others. Additionally, the described and currently understood toxicity mechanisms are also diverse, ranging from hepatotoxic effects associated with cylindrospermopsins, nodularins, and microcystins; neurotoxins (saxitoxin, anatoxin-a) and dermatotoxins (lyngbyatoxin-a, aplisiatxin), and LPS [27]. Although science focuses heavily on neurotoxins due to their greater degenerative incidence and the environmental and health

risks they represent, and hepatotoxins due to their greater distribution, incidence, and concomitance with other factors causing liver diseases, they are a priority according to ONU recommendations [28].

This suggests the necessary attention not only to aquatic animals but also to terrestrial life due to the bioaccumulation of cyanotoxins in the food chain [22].

It is a matter of extreme care; altering the functionality of the liver and muscles of various animal species, which are subsequently consumed by humans, can result in concentrations that exceed tolerance levels with a high toxicological load that is difficult to tolerate.

6. Classification of Cyanotoxins, Chemical Structure, and Mechanisms of Action

The essential characteristic of cyanobacteria concerning health hazards depends on their ability to synthesize cyanotoxins. Approximately more than 150 genera and around 2,000 species of cyanobacteria exist [13], with only a few species being toxic. Cyanotoxins are produced in the cytoplasm of these microorganisms, and they release their composition through cell lysis [17], which is a result of physiological processes related to cellular senescence due to pathological causes related to cellular stress, such as the use of algicides like copper sulfate and hydrogen peroxide. Additionally, the production and potency of cyanotoxins vary, necessitating constant monitoring of species with toxic potential to prevent damage or health alterations in humans and at-risk ecosystems [17].

The main cyanotoxins can be segmented according to their mechanisms of action in multicellular organisms and classified into three groups: i) hepatotoxins: microcystins and nodularins; ii) neurotoxins: anatoxin, homoanatoxin-a, guanitoxin, and saxitoxin; and iii) cytotoxins: cylindrospermopsin. Additionally, dermatotoxins include elements like lyngbyatoxin-a and debromoaplysiatoxin, the latter described as agents causing dermatitis.

Microcystins are heptapeptides whose target organ is the liver [9]. Anatoxins, on the other hand, are alkaloids structurally similar to acetylcholine, characterized by activating cholinergic receptors and keeping them activated for an indefinite time [17]. Furthermore, within the group of alkaloids are saxitoxins, which act on sodium channels, blocking the transmission of nerve impulses. Anatoxins and saxitoxins are considered neurotoxins because they affect the nervous system. They alter different nerve pathways, causing paralysis and respiratory failure, which can lead to death [9].

Hepatotoxin

The study of these toxins is crucial for understanding their effects and developing regulatory strategies among ecosystems, especially those vulnerable to blooms of toxic



cyanobacteria that produce hepatotoxins. Hepatotoxins (microcystins and nodularins) are produced by about eleven genera. (Table 1):

Table 1. Genera of Cyanobacteria Producing Hepatotoxins

Anabaenopsis	Nodularia	Planktothrix
Dolichospermum	Nostoc	Pseudanabaena
Hapalosiphon	Microcystis	Synechocystis
Lyngbya	Oscillatoria	

Source: Taken from Silva [17].

These cyanobacteria are the most common in freshwater bodies [17], which raises concerns about these toxins. They are cyclic peptides formed by seven amino acids, five of which are D-amino acids and two L-amino acids. This composition determines a series of variants based on the L-isomeric amino acids present in the cyclic chain. To date, more than 100 variants of microcystins (MCs) have been described. These variants arise from different combinations of amino acids and various other alterations (such as methylation or demethylation of several functional groups). The most common variants are microcystin-LR (leucine-arginine), microcystin-RR (arginine-arginine), and microcystin-YR (tyrosine-arginine) [17].

Neurotoxins

Neurotoxins affect the nervous system and are known for their rapid action, which in the worst cases, leads to death by respiratory failure within minutes of entering the body [21]. Their neurotoxic alkaloids act on cholinergic synapses or voltage-dependent ion channels, directly blocking nerve impulses in skeletal muscles, causing muscle paralysis and death by asphyxiation. The genera of cyanobacteria that produce neurotoxins are listed in Table 2:

Table 2. Genera of cyanobacteria producing neurotoxins.

Anabaenopsis*	Hapalosiphon*	Pseudanabaena*
Dolichospermum*	Oscillatoria*	Sphaerospermopsis
Chrysochlorum	Planktothrix*	Trichodesmium

Legend: (*) genera with species producing hepatotoxins. Source: Taken from Silva [17]

Neurotoxins can vary in their chemical structure. Anatoxin (or anatoxin-a) is a bicyclic secondary amine alkaloid structurally related to homoanatoxin-a, differing only in the presence of a propionyl group instead of an acetyl group [17]. On the other hand, guanitoxin, formerly known as anatoxin-a (S), is a methylphosphoryl ester of N-hydroxyguanidine and is the only known natural organophosphate. According to this classification, intoxication with this bioactive metabolite leads to progressive clinical signs of muscle fasciculations, reduced movement, abdominal breathing, cyanosis, seizures, and

death, which can occur within minutes to a few hours, depending on the affected animal species and the amount of toxin ingested [17].

Neurotoxins act as postsynaptic blockers of nicotinic and cholinergic receptors by irreversibly binding to acetylcholine receptors, overstimulating muscle contractions and causing muscle exhaustion [17]. Despite various studies on the toxicology of this neurotoxic alkaloid, some authors believe that the available database is not sufficient to determine a tolerable daily intake level due to the high level of uncertainty regarding long-term exposure.

6.2. Cytotoxins

Among the cytotoxins produced by cyanobacteria, cylindrospermopsin is the most well-known [29]. This toxic alkaloid was first described in 1979, when 148 people were hospitalized with symptoms of hepatotoxicity on Palm Island, associated with a bloom of the cyanobacterium *Cylindrospermopsis raciborskii* in a drinking water reservoir. Additionally, other species of cyanobacteria producing cylindrospermopsins have been identified, including: *Aphanizomenon ovalisporum* (reclassified as *Chrysochlorum*), *Aphanizomenon flos-aquae*, *Umezakia natans*, *Raphidiopsis curvata*, *Anabaena bergii*, *Anabaena lapponica*, and *Lyngbya wollei* [29].

The widespread distribution of cylindrospermopsin-producing species, along with the invasive nature of the primary toxin producer (*C. raciborskii*), poses a significant global water management issue [30]. It consists of a tricyclic alkaloid formed by a guanidine group combined with a hydroxymethyluracil. Due to its zwitterionic nature (electrically neutral chemical compound), this cyanotoxin is highly soluble in water. Additionally, natural structural variants have been identified, such as 7-epi-CYN (7-epicylindrospermopsin) and 7-deoxy-CYN (7-deoxycylindrospermopsin) [30].

These elements interfere with various metabolic pathways, triggering hepatotoxic, general cytotoxic, and neurotoxic effects, in addition to having carcinogenic potential [17]. Toxicity is mediated by the inhibition of glutathione, protein synthesis, and cytochrome P450, with the uracil and hydroxyl moiety at C7 crucial for toxicity. Intoxication can cause damage to the liver, kidneys, thymus, lungs, stomach, and heart [17]. Traditionally, cyanotoxins have been classified according to their chemical composition into peptides, alkaloids, and the presence of lipopolysaccharide (LPS), or eventually according to their toxic derivatives: hepatotoxins, neurotoxins, or dermatotoxins [30] (Table 3).



Table 3. Organs affected by recognized toxic metabolic compounds according to the existing classification of cyanobacteria.

Cyanotoxins	Primary organ or process affected	Genera of cyanobacteria associated
Microcystins	Liver	<i>Anabaena</i> , <i>Nostoc</i> , <i>Oscillatoria</i> , <i>Planktothrix</i> , <i>Anabaenopsis</i> , <i>Microcystis</i>
Nodularins	Liver	<i>Nodularia</i>
LPS	Cell, cytotoxicity	<i>Any cyanobacteria</i> , as it is a component of the Gram-negative bacterial membrane
Anatoxin-a	Cholinergic connections	<i>Oscillatoria</i> , <i>Anabaena</i> , <i>Aphanizomenon</i> , <i>Planktothrix</i>
Aplysiatoxins	Skin	<i>Lyngbya</i> , <i>Schizothrix</i> , <i>Planktothrix</i> (<i>Oscillatoria</i>)
Cylindrospermopsins	Liver	<i>Cylindrospermopsis</i> , <i>Aphanizomenon</i> , (<i>Umezakia</i>)
Lyngbyatoxin-a	Skin, gastrointestinal tract	<i>Lyngbya</i>
Saxitoxins	Neuronal connections, nerve impulse transmission repression	<i>Lyngbya</i> , <i>Aphanizomenon</i> , <i>Cylindrospermopsis</i> , <i>Anabaena</i>

Source: Taken from Andrinolo and Sedan [30].

If one reviews Table 3, a widespread occurrence of deferred toxins with analogous combinations of structural order can be perceived. Andrinolo and Sedan [30] in this sense, explain that these analogous segments are known collectively as toxins according to an STXs classification that groups all their limitations, and the same applies to the MCs, which are noted for maintaining possible variants only in some of them. The toxicological relevance for the environment lies in the typical variations in toxicity from raw cell states, with a higher incidence in biological assays involving rodent species and aquatic animals such as fish or crustaceans; this differentiation can be attributed to toxins that are distinguishable by a combination of chemical, biochemical, and immunological effects [22].

Therefore, evaluations of one or multiple toxins are not sufficient to segment the risk generated by the combination of cyanotoxins in a bloom, since these issues point to a conflict among bioassays because they fail to completely rule out elements that can be used complementarily in the analysis; leading to a false negative diagnosis when evaluations are applied to bodies of water or food, potentially interacting with these elements [30].

6.3. Consequences of the presence of cyanotoxins

Throughout this presentation, multiple types of health conditions have been highlighted that can intensify due to factors that transcend the growth of cyanotoxins,

attributable to transcribed levels of genes (peptide synthetase and polyketide synthase) [30]. This condition leads to an increase in these toxins without inhibition assays being able to alter them [31], hence the need to use costly assays for their detection in many cases. Additionally, the effects of components such as iron, while not directly influencing cyanotoxin production, do increase synthesis and impact toxicity levels [31].

On the other hand, while toxins have target organs or processes, they are capable of affecting or altering other organs and/or processes. In this regard, microcystins interact with the liver and are directly considered hepatotoxins, but they can also affect other organs such as the kidneys, lungs, and intestines [27]. Exposure to these types of components through direct contact with the eyes, mucous membranes, and even the ear, as well as ingestion of contaminated water and inhalation, constitutes a risk that can leave lasting health consequences for the individual [24].

In general, two types of intoxications are described: acute intoxication with microcystins causing significant hepatic damage, affecting the cytoskeleton by directly generating necrosis that alters hepatocytes [24] and chronic intoxication with microcystins, which has a wider range of symptoms and cannot go unnoticed; furthermore, this type of pathology is associated with high rates of liver cancer promotion [24]. At certain times, cyanobacterial blooms can produce highly harmful toxins as documented throughout this report, highlighting the impact of microcystins on health by affecting liver function [31].

Therefore, emphasis is placed on the importance of prior information before managing essential life resources, such as water quality, as an essential precursor to favoring the proliferation of cyanobacteria, which varies depending on whether a toxin-producing organism is involved in bodies of water used for consumption [32]. Typically, these organisms interact with water and calcium carbonate, resulting in pH values above 8.5 to 9.1, associated with high MC concentrations ($\geq 5 \mu\text{g/L}$). In this context, it is important to consider that light intensity and pH are triggering factors for the proliferation of cyanotoxins *in vitro* [32]. However, the influence of conductivity, temperature, TN:TP ratio, and trophic index [33]; also stand out; these are essential aspects to consider during molecular analysis of samples collected in different ecosystems [23].

7. Discussion

It can be said that regulations for studies regarding the proper management of water and fresh foods require the supervision of environmental components. Research such as that by Miglione et al. [20] proposes implementing electrochemical biosensors that have an applicable proportion for detecting natural toxins such as cyanotoxins. This has become an effective resource that has increased in



the last decade, providing valuable tools to understand the dynamics of these toxins in ecosystems, their impact on the food chain, and their health consequences.

The presence of cyanotoxins in water and fresh foods, and their passage into the gastrointestinal tract, implies bioaccessibility [16]. This is a crucial aspect when evaluating the risks of exposure, which can be direct through the consumption of fresh or minimally processed foods, with attention needed on the effects of cooking processes [20]. The ingestion of foods contaminated with cyanotoxins is the most common route of chronic exposure to them, following the ingestion of contaminated drinking water. However, the presence of cyanotoxins in food does not guarantee absorption, as this depends on several factors such as whether the toxins are in their free form or not [16], in addition to consumer susceptibility and toxic load; whether consumption is direct or through vectors; and whether there has been synergy with other exposure routes or toxins, which could increase the toxic load.

From minimally processed to highly processed foods, they undergo processes such as brining or other substances and/or cooking before ingestion. Heat can cause significant changes in the food matrix. In general, the effect of food processing depends on different aspects such as the type of processing, the type of compound considered, the composition and structure of the matrix, and the potential presence of other components that could affect the absorption of the mentioned compound [34]. Therefore, recent studies emphasize the importance of correlating *in vivo* and *in vitro* data on food digestion.

Although static *in vitro* models are simplified and do not reproduce all dynamic aspects of the gastrointestinal tract, only the main conditions such as pH, enzymes, and salt concentrations, these models are increasingly useful for predicting *in vivo* digestion in some cases and offer numerous advantages in evaluating the breakdown dynamics of some toxins. Some studies on marine toxins, for example, have been conducted using static *in vitro* models. The ideal *in vitro* digestion technique should provide accurate results quickly and serve as a tool for rapid analysis of food models with different structures and compositions. However, the most important thing is not just to understand these aspects, but to use science for risk management and prevention, taking actions based on these results to preserve the health of people who constantly interact with elements with high toxicological burden. In the long term, the implications of not doing so could be irreversible and even difficult to control.

8. Conclusions

The exploration of new development models must uphold a commitment to producing safe food and drinking water free from toxins that can harm the human body; this must be promoted through ecosystem conservation. Environmental

sustainability is crucial for all productive schemes, achieved through management practices that prioritize the care and conservation of water resources, ensuring quality of life.

Studies on water quality do not always encompass comprehensive analyses, often focusing on physicochemical properties while neglecting cellular and molecular bioindicators, as well as microbiological analyses crucial in current environmental conditions. Hence, there is a need for greater research into the presence of cyanotoxins in Latin American water bodies, especially in countries lacking regulations or controls ensuring safe water supply and availability of cyanotoxin-free food.

Governments worldwide should conduct continuous long-term monitoring and, if necessary, strengthen intervention efforts, considering not only the presence of cyanotoxins in aquatic ecosystems but also their impact on environmental services and the food chain. This affects not only the health of aquatic organisms that serve as fishery resources but also terrestrial organisms that form the dietary base for many communities.

Effectively managing risks associated with cyanotoxin presence hinges on implementing systematic monitoring schemes. Therefore, promoting accurate and appropriate assessment involving scientific input is crucial to ensure precise and reliable analyses that reflect the true state of ecosystems in each context, enabling measures to preserve human, animal, and environmental health.

Raising awareness among decision-makers, along with training and capacity building in research and management, is crucial in any management initiative to enhance awareness of this emerging risk, which often compromises not only health but also lives.

The food industry must implement control and monitoring strategies to prevent contamination of drinking water and fresh or minimally processed foods. Developing risk management models is critical to ensuring the supply of safe food and safeguarding consumer health.

9. Referencias

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