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Sterigmatocystin an emerging mycotoxin–a comprehensive review

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A B S T R A C T

Background: Sterigmatocystin (STC) is an indoor air and food mycotoxin contaminant, which is formed by several fungal genera, most notably *Aspergillus* species. It is considered a potential factor in causing esophageal, gastric, and lung malignancies in humans. It is grouped as a Group 2B carcinogen by the International Agency for Research on Cancer (IARC). STC shares structural and biosynthetic similarities with aflatoxin B1, underscoring its toxicological relevance. Global climate change has expanded the occurrence of STC beyond tropical regions into temperate zones, raising significant food safety concerns. **Amis**: This review consolidates current knowledge on STC occurrence in food, feed, and indoor air, its biosynthesis, methods of isolation and detection, biological activities, and toxicological effects.

Methods: The literature review was conducted using two major scientific databases, PubMed and Google Scholar. The keyword "sterigmatocystin" was applied to retrieve relevant publications, and articles were selected for citation based on the significance of the information they provided.

Results: Analytical advances such as LC-MS/MS, High-Resolution Mass Spectrometry (HRMS), and biosensor technologies have improved detection sensitivity, while biological detoxification strategies using lactic acid bacteria show promise in mitigating contamination. Despite these developments, gaps remain in understanding STC's toxicological mechanisms, Circulation through nutritional pathways, and regulatory thresholds.

Conclusion: This review underscores the importance of continued investigation into the health hazards posed by sterigmatocystin and highlights the need for effective preventive measures. Overall, it distinguishes itself by offering a more comprehensive, current, and integrative perspective, weaving together the chemistry, biology, toxicology, and public health implications of STC into a unified analysis.

Keywords: *secondary metabolites, food poisoning, air pollution, human carcinogen*

1. Introduction

For over a century, mold genera such as *Aspergillus*, *Penicillium*, and *Talaromyces* have served as valuable sources for isolating diverse

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bioactive compounds [1]. According to Bladt et al [1], although an estimated three million fungal species exist worldwide, only about 100,000 have been formally identified. This highlights the vast, untapped potential of fungi to yield novel and promising bioactive molecules. *Aspergillus* is a genus of filamentous fungi first described nearly 300 years ago, now comprising more than 250 recognized species. While many *Aspergillus* species are valued for their secondary metabolites widely applied in medicine and industry, others are pathogenic and produce toxins harmful to humans, birds, and animals [2]. Mycotoxins are highly toxic secondary metabolites predominantly produced by fungi belonging to the genera *Aspergillus*, *Penicillium*, and *Fusarium* [2]. These pathogenic fungi and their toxins are especially problematic in tropical and subtropical regions. However, due to global warming and climate change, their presence and associated risks have increasingly extended into temperate zones [3].

Sterigmatocystin (STC) is recognized as an emerging mycotoxin and a natural carcinogen. The International Agency for Research on Cancer (IARC) has classified it as a Group 2B substance, meaning it is possibly carcinogenic to humans. [4,5]. Belonging to the polyketide class of mycotoxins, STC shares structural similarities with aflatoxin B1. It is mainly produced by *A. versicolor*, though other *Aspergillus* species such as *A. flavus*, *A. parasiticus*, and *A. nidulans* also contribute [6]. In contrast, aflatoxin B1 (AFB1) is primarily associated with *A. flavus* and *A. parasiticus*. Both toxins arise from the same biosynthetic pathways (the alternative oxidase (AOX) and cytochrome C oxidase (COX) pathways); however, literature indicates that AFB1 exerts more severe effects on living organisms compared to STC.

Despite this, available knowledge on the biological effects of sterigmatocystin remains quite restricted [7]. Most of the existing evidence comes from in vitro experiments or animal studies, while human data are minimal. Overall, this review distinguishes itself by presenting a more comprehensive, updated, and integrative account, linking STC's chemistry, biology, toxicology, and public health relevance within a single framework.

Aims:

Owing to global climate change and the appearance of pathogenic moulds and their mycotoxins in the countries located in the temperate zone, there is a massive amount data on sterigmatocystin occurrence, and toxicology, but there is no comprehensive review that collects and summarizes the updated aspects of sterigmatocystin. The aim of this study is to provide an updated and comprehensive review of the occurrence, chemistry, isolation, detection, biology and toxicology of the emerging mycotoxin sterigmatocystin.

2. Methods:

The literature review was conducted using two major scientific databases, PubMed and Google Scholar. The keyword "sterigmatocystin" was applied to retrieve relevant publications, and articles were selected for citation based on the significance of the information they provided.

3. Results:

Occurrence of sterigmatocystin (STC):

Natural sources of Sterigmatocystin:

STC is an emerging mycotoxin produced by several fungi, notably Aspergillus species and genera like *Chaetomium*, *Emericella*, *Penicillium*, and *Bipolaris*. STC production is confirmed in several *Versicolores* strains, including *A. versicolor* and related species, while *A. sydowii* is typically excluded [6,8,9]. Nevertheless, some investigations have indicated that *A. sydowii* may also synthesize STC [5].

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Furthermore, Géry et al [5] reported for the first time that *A. tabacinus* is capable of sterigmatocystin biosynthesis.

Site of production of STC within the mold's colony:

Ámon et al [10] reported that sterigmatocystin is synthesized during the sexual reproduction of *Aspergillus nidulans* as a means of protecting its reproductive structures. Their findings showed that STC production occurs specifically in hyphae located adjacent to hülle cells.

Sterigmatocystin presence in food and feed:

As presented in Table 1, Molds are a major cause of post-harvest diseases in fruits and vegetables, leading to the loss of approximately 20 - 25% of harvested produce in both developed and developing nations. In the United States alone, mycotoxin contamination is estimated to result in annual economic losses of about 1 billion USD [11]. Mycotoxin contamination in cereals like rice, wheat, and maize has created health risks for people and animals in recent years. During the post-harvest stage, fungal infestation can give rise to sterigmatocystin (STC) and aflatoxins in cereal grains [6]. Beyond animal feed, STC has been consistently detected in food items including bread, corn, grains, spices, coffee beans, soybeans, cheese, and pistachios, with STC-producing fungi frequently isolated from these commodities [9]. In a study conducted in China, Zhao et al [12] analyzed 126 tea samples using optimized procedures and found STC in 17 samples, with concentrations ranging from 0.13 to 4.48 µg/kg. While Veršilovskis et al [9] and Zhao et al [12] confirmed the occurrence of STC in food, Dabelić et al [13] reported that STC is relatively rare, appearing only at low concentrations in plants used in traditional Chinese medicine, cereals, spices, beer, and cheese.

Current knowledge on the fate of sterigmatocystin (STC) and its metabolites during food processing is still limited, with most investigations concentrating on its metabolic conversion in animals [6]. In a related study, Pietri et al [4] reported the presence of STC in 101 grated cheese samples obtained from retail outlets in Northern Italy, with concentrations ranging from <LOD to 6.87 µg/kg. Nevertheless, STC occurrence in cheese is generally rare, as aflatoxin-producing fungi are seldom associated with such products. Furthermore, no evidence has yet been documented regarding the transfer of STC or its metabolites into other animal-derived foods, including eggs and meat [11].

Due to the limited characterization of sterigmatocystin (STC), European authorities have advised the systematic collection of reliable data regarding its presence in food and feed. For food products, a limit of quantification (LOQ) below 1.5 µg/kg has been suggested, while the lack of sufficient data currently prevents the establishment of feed limits. In addition, the creation of certified reference materials and the implementation of proficiency testing have been recommended to enhance analytical reliability [11].

Table 1. Summary of STC detection in various foodstuffs

Source	Occurrence of STC	Concentration of STC	Notes / Citations
Cereal grains (rice, rye, barley, wheat, oats, maize)	Contamination during post-harvest	Not specified	STC + aflatoxins detected [6]
Bread, corn, grains, spices, coffee beans, soybeans, cheese,	Regular presence	Not specified	STC-producing fungi isolated [9]

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Source	Occurrence of STC	Concentration of STC	Notes / Citations
pistachios.			
Tea samples (China, Zhao et al)	Detected in 17/126 samples	0.13 – 4.48 µg/kg	[12]
Plants used in traditional Chinese medicine, cereals, spices, beer, and cheese.	Rare occurrence	Low concentrations	[13]
Grated cheese (Italy, Pietri et al)	Detected in 101 samples	LOD – 6.87 µg/kg	[4]
Cheese (general occurrence)	Rare	Not specified	Aflatoxin-producing fungi are uncommon [11]
Animal-derived foods (eggs, meat)	No documented transmission	—	[11]

Sterigmatocystin presence in indoor-air:

Indoor air pollution is considered one of the most significant health hazards, particularly in developed countries where people spend much of their time in transport systems or enclosed buildings. Fungal genera such as *Aspergillus*, *Penicillium*, and *Cladosporium* are frequently identified as major indoor contaminants, contributing to health issues including mycotoxin production, allergic reactions, itching, and the worsening of asthma symptoms. These fungi are also linked to sick-building syndrome, which presents with non-specific complaints such as dizziness, fatigue, irritability, poor concentration, and headaches, along with respiratory and dermatological symptoms like a runny nose, nasal itching, eye irritation, dry throat, breathing difficulties, and skin dryness or rash [5].

According to Dabelić et al [13], sterigmatocystin has been detected in the air of water-damaged buildings and industrial environments. Its presence in indoor air is associated with mycelial fragments and spores of producing fungi, particularly strains within the *Aspergillus* series Versicolores [5]. While several studies suggest that these strains may cause respiratory or skin-related symptoms, the direct relationship between sterigmatocystin exposure and such health effects remains insufficiently explored.

Biosynthesis of sterigmatocystin:

Sterigmatocystin (STC), a potent mycotoxin and natural carcinogen, exhibits structural similarity to aflatoxin B1, as both molecules contain xanthone and furan ring systems (Figure 1 and 2). In *Aspergillus flavus* and *A. parasiticus*, the primary producers of aflatoxin B1, STC follows the same biosynthetic pathway as aflatoxins, functioning as a late-stage intermediate in the aflatoxin B1 pathway [7][14][19]. Furthermore, Molnár et al [15] demonstrated that in *Aspergillus nidulans*, STC biosynthesis proceeds via both the alternative oxidase (AOX) and cytochrome C oxidase (COX) pathways.

Unlike the previously mentioned studies, Pardo and Esteve-Turrillas [25] noted that sterigmatocystin (STC) acts as a precursor to aflatoxins, sharing the same polyketide-derived biosynthetic pathway, which underscores its toxicological relevance. Within this pathway,

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STC serves as an intermediate in the formation of aflatoxin B1 (AFB1) and aflatoxin G1 (AFG1) (Figure 1). In aflatoxin-producing fungi, STC is quickly converted into O-methylsterigmatocystin (OMST), the direct precursor of AFB1 and AFG1. Consequently, STC rarely accumulates; however, species such as Aspergillus nidulans and A. versicolor appear unable to carry out this conversion into OMST.

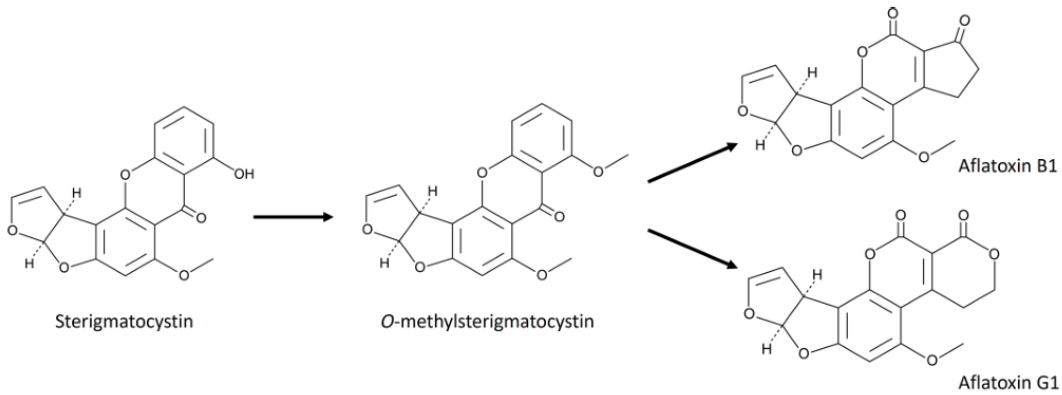

Figure 1: Sterigmatocystin and its derivative O-methylsterigmatocystin undergo conversion within the biosynthetic pathway, resulting in aflatoxin B1 and G1 [25]

Methods of isolation and detection of Sterigmatocystin:

As summarized in Table (2) and Figure (2), several analytical methods were used to detect the presence of STC in foodstuffs. Michael and Rodricks [17] described a qualitative and quantitative method for detecting sterigmatocystin (STC) in grain samples. Initially, the samples were extracted using acetonitrile: water (9:1) with potassium chloride (KCl). The extract was then partitioned first with hexane and subsequently with chloroform ($CHCl_3$), followed, when necessary, by silica gel column chromatography. This process yielded an extract containing STC. Its presence was assessed by comparing fluorescence intensity on TLC plates with that of a pure STC standard, and visualization was enhanced using an $AlCl_3$ spray reagent. STC was further isolated on preparative TLC plates, and its identity was confirmed through the formation of acetate derivatives and acid-treated derivatives.

Kubosaki et al [8] demonstrated that single nucleotide polymorphism (SNP)-based PCR amplification, employing a high-discrimination DNA polymerase (HiDi DNA polymerase), is a reliable and robust screening approach for identifying STC-producing fungal strains.

Pardo and Esteve-Turrillas [25] reviewed various analytical methods for detecting sterigmatocystin (STC) in food. Among these, liquid chromatography (LC) is the most commonly applied technique due to its strong resolution capacity, often paired with detectors such as diode array (DAD), fluorescence (FD), or mass spectrometry (MS). LC-MS/MS provides superior sensitivity and selectivity, enabling trace-level detection of STC and simultaneous analysis of multiple mycotoxins across diverse food matrices. High-resolution mass spectrometry (HRMS), including LC-HRMS/MS with Orbitrap or QTOF systems, further enhances accuracy by allowing unambiguous identification.

Immunoassays like ELISA offer a rapid and cost-effective screening option, though they are generally less specific and sensitive compared to LC-MS/MS. More recently, innovative biosensors, such as graphene oxide-aptamer FRET-based aptasensors, have been developed, enabling one-step, highly selective detection of STC in foods like chili and pepper with minimal interference.

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Table 2. Advantages and limitations of STC's analysis methods

Technique	Principle	Advantages	Limitations	Citation
TLC-based extraction (Michael & Rodricks)	TLC with fluorescence, STC standard. visualization with $AlCl_3$ spray reagent	Simple, cost-effective, qualitative & quantitative	Lower sensitivity, labor-intensive, requires derivatization	[17]
SNP-PCR (Kubosaki et al)	SNP-based PCR amplification using HiDi DNA polymerase to identify STC-producing fungal strains	Reliable, robust, highly specific for strain screening	Limited to fungal strain detection, not direct toxin quantification	[8]
Liquid Chromatography (LC)	Reverse-phase C18 columns with DAD, FD, or MS detectors	High resolution, widely used	Requires instrumentation, moderate sensitivity	[25]
LC-MS/MS	LC coupled with tandem MS for trace-level detection and multiresidue analysis	Highest sensitivity & selectivity, detects multiple mycotoxins simultaneously	Expensive, requires skilled operation	[25]
High-Resolution MS (HRMS)	LC-HRMS/MS with Orbitrap or QTOF detectors for unambiguous identification & non-targeted screening	Extreme selectivity, detects emerging/unknown compounds	High cost, complex data analysis	[25]
ELISA (Immunoassay)	Antibody-based detection of STC and other	Rapid, cost-effective, high-throughput	Less specific & sensitive, matrix interferences	[25]

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Technique	Principle	Advantages	Limitations	Citation
	mycotoxins	screening		
Graphene oxide-aptamer FRET biosensor	Aptamer-based fluorescence resonance energy transfer for one-step STC detection	Highly selective, minimal interference, innovative	Still emerging, with limited availability	[25]

Biological activities of sterigmatocystin:

Inhibition of *Plasmodium falciparum*:

Fungi have long been acknowledged as reliable sources of antimalarial agents, with numerous metabolites demonstrating significant anti-plasmodial properties [18]. In their study, Niu et al [18] successfully isolated sterigmatocystin (STC) from *Penicillium janthinellum*. Their findings revealed that STC inhibited the growth of *Plasmodium falciparum* in blood with an IC_{50} of 34 µM and reduced the infectivity of sexual-stage parasites in mosquitoes with an IC_{50} of 48 µM. Notably, STC exhibited no acute toxicity toward human kidney cells at concentrations up to 64 µM. These results suggest that STC could represent a promising lead compound for malaria control and serve as a valuable marker for exploring the molecular mechanisms of malaria transmission.

Biological detoxification and binding of sterigmatocystin on Lactobacilli

Lactic acid bacteria (LAB) are recognized as effective agents for biological detoxification, acting as a natural barrier against the entry of mycotoxins into the food chain. Their protective mechanisms involve suppressing the growth of mycotoxin-producing fungi, binding toxins to their cell surfaces, and, in rare cases, degrading the toxins. LAB occur naturally in both humans and animals. Several investigations have shown that certain LAB strains can bind mycotoxins such as aflatoxin B1 (AFB1) to their surfaces [3]. In the study by Kosztik et al [3], it was demonstrated that mycotoxin exposure did not hinder bacterial growth, and notably, the binding capacity of Lactobacillus strains for sterigmatocystin (STC) was approximately twice that observed for aflatoxin B1.

Binding of sterigmatocystin to human serum albumin:

Sterigmatocystin is a precursor of aflatoxin B1 biosynthesis. As they share the same biosynthetic pathway, the toxic effects of these two mycotoxins are similar.

The affinity of xenobiotics to human albumin plays an important role in determining the magnitude of biological effects of these bioactive molecules on human tissues.

The binding affinity of (camptothecin, warfarin, and naproxen) to albumin was not significantly affected by STC. On human serum albumin (HSA), STC binds to the Heme site (FA1). The acute toxicity of STC is relatively low. Although, acute exposure to STC can cause hepatocellular necrosis in certain species. It is nephrotoxic in rats and monkeys. Also, in vitro studies suggested that STC has genotoxic effects [16].

Effects of STC on common Crap liver:

Cereals are frequently incorporated into fishmeal for aquaculture feeds. However, the presence of filamentous fungi in cereals [9] has

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contributed to increased mycotoxin contamination in such diets. The liver is the primary target organ of aflatoxin B1 (AFB1) toxicity, whereas sterigmatocystin (STC) can affect both the liver and kidneys [20]. In the study conducted by Kövesi et al [21], the short-term effects of dietary exposure to AFB1 (100 µg/kg feed), STC (1000 µg/kg feed), and their combination (100 µg AFB1 + 1000 µg STC/kg feed) were evaluated in one-year-old common carp (*Cyprinus carpio*) by examining lipid peroxidation and the glutathione redox system at both biochemical and gene expression levels. The results indicated that STC markedly reduced glutathione (GSH) levels, while glutathione peroxidase activity remained unchanged. In contrast, AFB1 or the combined treatment (AFB1+STC) produced weaker effects compared to STC alone. The authors concluded that the individual actions of AFB1 and STC on different parameters may interact synergistically or antagonistically under multi-toxin exposure.

Sterigmatocystin toxicology:

In London, in 1962, almost 100,000 turkeys died in an unfamiliar veterinary disaster. The death of turkeys was linked to the contamination of peanut meals with aflatoxin, “turkey X disease”. Since that time, research in the field of mycotoxins has become an important scientific issue. Nowadays, mycotoxicosis and mycotoxin contaminations are considered as main health problems in the third world. For a long time, the countries located in the tropical region have been fighting the risk of mycotoxins [11].

In humans, the toxic impact of sterigmatocystin (STC) appears to be less pronounced than that of aflatoxins; however, its significance cannot be disregarded [11]. Contrary to the observations of Ráduly et al [11] and Géry et al [5], in vivo studies have shown that STC can induce toxic effects in several animal species, including mice, rats, pigs, fish, chickens, monkeys, and ruminants, depending on the species, exposure frequency, and route of administration. In one trial, sheep fed the highest dose of STC (16 mg/kg feed, approximately 0.3 mg/kg body weight per day) exhibited no observable signs of toxicity. Therefore, the precise toxicity profile of STC in livestock remains uncertain [11].

Since the toxicity of STC on humans and livestock is not clear, an illustration of the mechanism’s toxicity, through which it exerts its toxic effects on humans and animals, was needed.

Sterigmatocystin mechanism of toxicity:

As previously noted in this review, sterigmatocystin (STC) has been designated by the IARC as a Group 2B possible human carcinogen [4], [5], due to its demonstrated tumor-inducing capacity in diverse cell culture systems and animal models. Nevertheless, internationally harmonized regulations specifying maximum allowable concentrations of STC in food remain inadequately established [11]. Díaz Neito et al [22] reported that STC exhibits relatively low acute oral toxicity, with LD_{50} values ranging from 120 to 166 mg/kg body weight. Documented findings include malignant and premalignant changes—such as angiosarcomas and hepatocellular carcinomas—occurring in brown fat following oral administration of STC (Figure 2). Although STC is both genotoxic and carcinogenic, its potency is approximately three orders of magnitude lower than that of aflatoxin B1. In hepatic and pulmonary tissues, several CYP enzymes metabolize STC into multiple hydroxy derivatives, which are subsequently excreted via urine and bile [11].

As revised by Ráduly et al, [11], the reactive epoxi-adducts of STC can covalently bind to DNA and generate the STC-N_7-guanine adducts. These derivatives are proposed to be responsible for the mutagenicity of STC. In addition, Pfeiffer et al, [23] proposed that the hydroxylation of the aromatic ring in the STC parent structure creates a catechol. The created derivative could produce mutagenicity; when it reacts with DNA. In comparison to the epoxide derivative, catechol was formed as a major metabolite in the liver microsomes of humans

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and rats.

Recently, the mechanism of toxicity by which STC can induce human oesophageal and gastric cancers has been illustrated. STC can induce DNA double-strand disruptions in humans immortalized bronchial epithelial cell line. This effect may lead to adenocarcinomas. In addition, an *in vivo* experiment in a rat model also confirmed these findings [11].

The presence of STC in the dust of indoor settings may result in inhalation of these mycotoxins by the inhabitants [5]. *In vivo* experiment on rats carried out by Jakšić et al, [24], the rats were exposed to air contaminated with STC in concentrations (0.3 mg STC/kg of lung weight). They clarified that, STC in naturally occurring concentrations may induce DNA-single strand breaks in rat lungs with insignificant cytotoxicity.

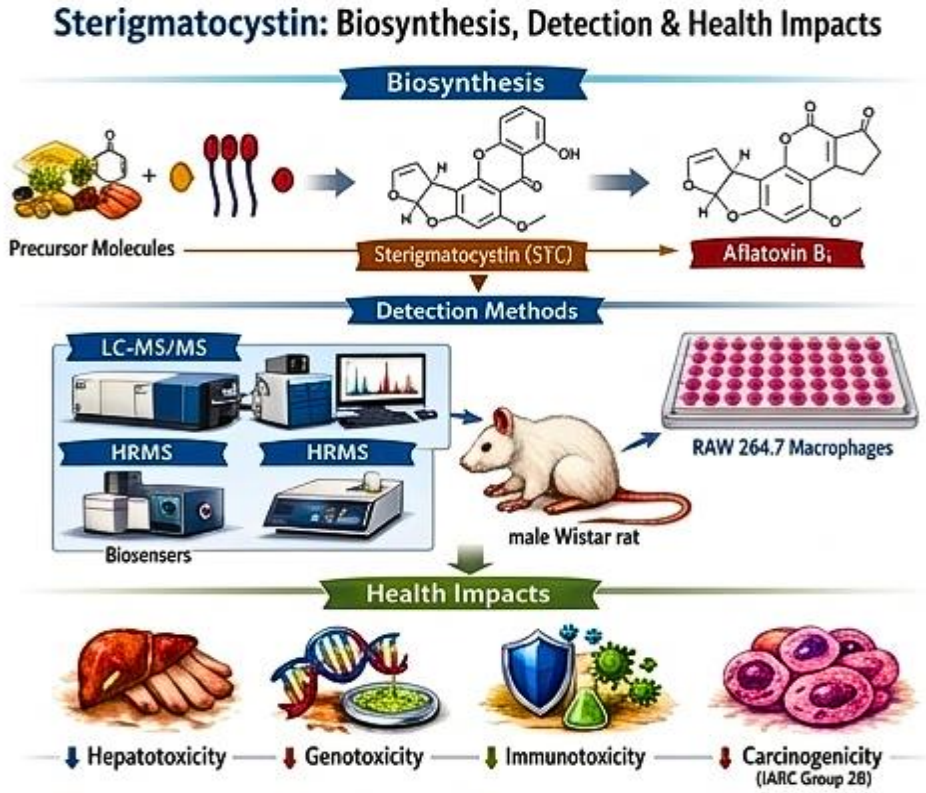

Figure 2: Sterigmatocystin: Biosynthesis, Detection and Health Impacts

Preventive strategy of sterigmatocystin poisoning:

To our knowledge, there is no specific treatment of sterigmatocystin poisoning, although a well-documented case of human poisoning with aflatoxin B1 (structurally similar to STC) was reported by Ráduly et al, [11]. They stated that "A young woman attempted suicide using purified aflatoxin B1 (AFB1), ingesting 5.5 mg over two days, followed six months later by a cumulative dose of 35 mg within two weeks. Despite these exposures, successive diagnostic evaluations, including liver imaging (X-ray and ultrasound) as well as urine and blood analyses, revealed no pathological findings over the years. The absence of clinical symptoms or physiological abnormalities was attributed

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to her robust physical health and adequate nutritional status".

Until now, there is limited data on the treatment of mycotoxin poisoning and it is still not specific. Therefore, the best strategy for preventing of mycotoxin poisonings is to block the access of mycotoxins to the food and feed chain. E.g. monitoring of the pre-harvest and post-harvest practices that ensure the mycotoxin-free agricultural products [11].

4. Conclusion

STC is an emerging food safety hazard due to its carcinogenicity and broad occurrence in crops, foods, and indoor settings. Although structurally related to aflatoxin B1, STC remains less studied, with limited data on its toxicological mechanisms, metabolism, and long-term effects in humans and livestock. Current evidence highlights its genotoxicity, hepatotoxicity, and nephrotoxicity, as well as its potential role in malaria control and biological interactions with lactic acid bacteria. Advances in analytical methodologies have enhanced detection capabilities, yet regulatory frameworks remain incomplete, particularly for feed. Preventive strategies should prioritize monitoring pre- and post-harvest practices, improve analytical standards, and expand toxicological studies. Ultimately, STC must be considered a critical mycotoxin requiring further investigation to safeguard food security and human health.

ABBREVIATIONS AND ACRONYMS

STC: sterigmatocystin, **IARC:** International Agency for Research on Cancer, **LC-MS/MS:** Liquid Chromatography/Mass Spectroscopy, **HRMS:** High-Resolution Mass Spectrometry, **AFB1:** aflatoxin B1, **AFG1:** aflatoxin G1, **HAS:** human serum albumin, **AOX:** alternative oxidase, **COX:** cytochrome C oxidase, **LOD:** Limit of Detection, **LOQ:** Limit of Quantification, **OMST:** O-methylsterigmatocystin, **HiDi DNA polymerase**: high-discrimination DNA polymerase, **SNP:** single nucleotide polymorphism, **DAD:** diode array, **FD:** Fluorescence Detector, **MS:** Mass Spectrometry, **QTOF:** Quadrupole Time-of-Flight Mass Spectrometry, **ELISA:** Enzyme-Linked Immunosorbent Assay, **FRET:** Förster Resonance Energy Transfer, **IC_{50}:** Half Maximal Inhibitory Concentration, **LAB:** Lactic acid bacteria, **HAS**: human serum albumin, **GSH:** glutathione, **CYP:** Cytochrome P450 superfamily.

ETHICAL APPROVAL

Not Applicable

CONFLICT OF INTEREST

The authors affirm that neither financial interests nor personal relationships compromised the integrity of this work.

AUTHORS' CONTRIBUTIONS

JS: conceptualization of the study, **MSF:** conceptualization, review. **MEOA**: literature review, writing original draft, review and editing.

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