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Review

Contamination of herbal products by mycotoxins: a review of the public health implications

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Abstract

The use of medicinal plants and their derivatives for the prevention and treatment of various diseases is widespread. However, contamination of these products has become a significant global concern in recent decades due to the potential health risks to consumers. Ensuring the quality and safety of herbal products is therefore essential. Among the most hazardous contaminants are mycotoxins-naturally occurring toxic compounds produced by certain fungi-which pose serious public health risks due to their toxicological effects. This review provides an overview of mycotoxin contamination in herbal products, outlines the permissible contamination limits as established by the European Pharmacopoeia, discusses both clinical and experimental evidence of mycotoxin toxicity, and proposes strategies for preventing contamination.

Keywords:
Medicinal plants, Contamination, Mycotoxins, Toxicity.

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1. Introduction

A medicinal plant is defined as any plant that contains, in one or more of its parts, substances that can be used for therapeutic purposes or serve as precursors for the semi-synthesis of pharmaceutical compounds [1]. Medicinal plants are widely utilized both as home remedies and as raw materials in the pharmaceutical industry [2, 3]. Extracts obtained from these plants have been used in traditional medicine for thousands of years, and these practices remain prevalent today [4, 5]. They are employed to prevent or treat various ailments and health conditions and are often perceived as natural and safe. In many developing countries, herbal medicine remains the primary form of healthcare. Meanwhile, in developed countries, the use of herbal products has grown significantly in recent decades, leading to a substantial increase in the commercialization of medicinal plants.

One of the most critical safety concerns associated with medicinal plants is their contamination by mycotoxins. Mycotoxins are highly toxic secondary metabolites produced by various fungi, including species of *Aspergillus*, *Fusarium*, *Penicillium*, and others [6]. Controlling mycotoxin contamination is particularly challenging due to their low molecular weight, thermal stability, and lack of immunogenicity [7]. Medicinal plants can become contaminated with molds at multiple stages—during harvesting, processing, storage, and transportation. Under favorable environmental conditions, these fungi can persist and proliferate, leading to the accumulation of mycotoxins over time [8]. The primary mycotoxins of public health concern include aflatoxins (AF), ochratoxins (OT), fumonisins (F), zearalenone (ZEA), and trichothecenes (T), particularly deoxynivalenol (DON) and T-2 toxin.

Unfavorable climatic conditions, insect infestations, and poor handling during transport and storage can promote fungal contamination of medicinal plants and lead to the production of mycotoxins [9]. Inadequate packaging further exacerbates the risk of mycotoxin contamination [2]. The presence of mycotoxins not only diminishes the therapeutic efficacy of medicinal plants but can also render the products unsuitable for national and international trade [10]. Therefore, quality control measures and the establishment of regulatory standards for mycotoxin levels in herbal products are essential to protect consumer health [11]. In response to these concerns, the European Pharmacopoeia (EP) has set maximum permissible limits for mycotoxins in medicinal plants and their derived products [12]. In general, herbal medicines are expected to meet the same quality standards as conventional pharmaceutical drugs.

In this review, we provide an overview of the contamination of herbal products by mycotoxins, including reported cases, regulatory limits, the health risks associated with mycotoxin exposure, and strategies for prevention and control aimed at ensuring consumer safety.

2. Generalities

Mycotoxins can contaminate a wide range of agricultural products and foodstuffs [13]. Among them, aflatoxins are the most widespread, particularly in crops grown in hot and humid regions [14]. The proportion of *Aspergillus flavus* strains capable of producing aflatoxins on culture media can reach up to 50% [15, 16]. However, aflatoxins may be absent in some samples despite a high presence of aflatoxin-producing strains, indicating that contamination depends on multiple environmental and physiological factors [17].

Aspergillus flavus exhibits optimal growth at temperatures between 36–38 °C, while aflatoxin production is maximized at temperatures ranging from 25–35 °C [18]. These conditions, commonly found in tropical and subtropical regions, promote both fungal proliferation and mycotoxin production [19]. Each fungal species has a specific minimum water activity (aw) threshold below which it cannot grow or produce toxins [20]. For example, the minimum aw values for the growth of *A. flavus* and *A. parasiticus* are 0.78–0.80 and 0.80, respectively, while their minimum aw values for aflatoxin production are 0.83–0.87 and 0.87, respectively.

Similarly, the minimum aw values for the growth of *Aspergillus ochraceus*, *Penicillium cyclopium*, and *Penicillium viridicatum* are 0.77–0.83, 0.81–0.85, and 0.83, respectively. Their respective minimum aw thresholds for ochratoxin A production are 0.83–0.87, 0.87–0.90, and 0.83–0.86 [21].

3. Admissible limits of contamination

The European Pharmacopoeia has established maximum permissible limits for mycotoxin contamination in herbal medicinal products: 2 µg/kg for aflatoxin B1 (AFB1) and 4 µg/kg for total aflatoxins [12].

For other mycotoxins, there is generally no harmonized limit in the European Pharmacopoeia; these toxins are not yet regulated for medicinal plants.

4. Contamination reports

A total of 39 commercially available ginger products from six manufacturers were analyzed using reversed-phase liquid chromatography (RPLC) with fluorescence detection. Among these, 26 samples were found to be contaminated with aflatoxins (AF) at concentrations ranging from 1 to 31 ng/g, while 29 samples contained ochratoxin A (OTA) at levels between 1 and 10 ng/g. Ten samples were free of both AF and OTA. Additionally, 10 finished ginseng products were analyzed using liquid chromatography–tandem mass spectrometry (LC-MS/MS); three of them contained AF at 0.1 ng/g, and four were contaminated with OTA at concentrations ranging from 0.4 to 1.8 ng/g [22]. In Spain, 84 samples of aromatic and medicinal herbs were examined using ELISA to detect various mycotoxins. The contamination rates were notably high: 99% for T-2 toxin, 98% for zearalenone (ZEA), 96% for aflatoxins, 63% for OTA, 62% for deoxynivalenol (DON), 61% for citrinin, and 13% for fumonisins (FB). All samples showed multi-mycotoxin contamination, with nearly 87% containing four or more mycotoxins simultaneously—most commonly AF, T-2, and ZEA [23]. In Argentina, 152 dried medicinal and aromatic herbs were tested using thin-layer chromatography (TLC) and high-performance liquid chromatography (HPLC). The analysis confirmed the presence of aflatoxins, ochratoxins, and fumonisins B1 and B2 [16].

In India, 15 commercially marketed spices were screened for multiple mycotoxins, including aflatoxins, rubratoxins, ochratoxin A, citrinin, zearalenone, and sterigmatocystin. Coriander and fennel had the highest number of positive samples and the highest levels of contamination, while cinnamon, cloves, yellow moss, and Indian mustard showed no detectable mycotoxins. Aflatoxins were the most frequently detected, with significantly lower incidence rates for the other toxins [24].

In Morocco, 10 olive samples obtained from retail outlets and supermarkets were analyzed by HPLC with fluorescence detection. All samples were contaminated with OTA and AFB, while 80% also contained citrinin [25]. In Turkey, visibly moldy dried figs deemed unfit for consumption were analyzed using liquid chromatography coupled with mass spectrometry. The results revealed the presence of several fungal metabolites, including fumonisins B2 and B4, patulin, HT-2 toxin, and zearalenone [26]. In a separate study conducted in Turkey, both OTA and AFB1 were detected in local and imported dried figs [27].

5. Mycotoxicities

5.1. Aflatoxins

Aflatoxins are among the most potent naturally occurring carcinogens, affecting both humans and various animal species [28, 29]. The International Agency for Research on Cancer (IARC) has classified aflatoxins in Group 1, indicating they are carcinogenic to humans [30]. In humans, acute aflatoxicosis—resulting from a single or multiple high-level exposures—can be fatal, while chronic exposure is associated with hepatocellular carcinoma, immunosuppression, and growth retardation [31, 32]. Epidemiological studies have demonstrated a strong correlation between dietary aflatoxin exposure and the incidence of liver cancer [33].

Aflatoxin B1 (AFB1) is considered the most toxic and carcinogenic aflatoxin, and one of the most potent known hepatocarcinogens in humans [29, 34]. Animal studies have shown that AFB1 can also produce teratogenic and embryotoxic effects [35]. Moreover, synergistic interactions between AFB1 exposure and chronic hepatitis B virus (HBV) infection significantly increase the risk of developing hepatocellular carcinoma [36, 37]. Similar synergistic effects have also been reported with hepatitis C virus (HCV) infection [38].

The median lethal dose (LD_{50}) of AFB1 varies across species, ranging from 1 to 50 mg/kg body weight in most experimental animals [39]. Among these, rats are particularly susceptible to AFB1-induced carcinogenesis, while mice exhibit greater resistance [40]. Additionally, exposure to aflatoxins has been shown to induce lung carcinogenesis in several animal models [41, 42, 43].

5.2. Ochratoxin A

Ochratoxin A (OTA) exhibits a wide range of toxic effects, including nephrotoxicity, teratogenicity, genotoxicity, neurotoxicity, hepatotoxicity, and immunosuppression [44]. The International Agency for Research on Cancer (IARC) has classified OTA as a possible human carcinogen (Group 2B) [30]. Notably, chronic exposure to low doses of OTA may be more toxic than acute high-dose exposure [45].

OTA has been implicated in the development of Balkan endemic nephropathy, a fatal kidney disease affecting certain populations in Eastern Europe [46, 47]. Additionally, early-life exposure to OTA has been associated with an increased risk of testicular cancer [48].

The median lethal dose (LD_{50}) of OTA in experimental animals ranges from 0.2 to 58.3 mg/kg, depending on the species [44]. Teratogenic effects—such as craniofacial malformations and reduced birth weight—have been reported in several animal models following prenatal OTA exposure [45, 49].

5.3. Fumonisins

The International Agency for Research on Cancer (IARC) has classified fumonisin B1 (FB1) as possibly carcinogenic to humans (Group 2B) [30, 50]. Human exposure to fumonisins has been linked to increased risks of esophageal and liver cancers [50, 51, 52]. Fumonisins frequently contaminate corn, and epidemiological studies have reported a higher incidence of esophageal cancer in populations from regions where corn and corn-based products are dietary staples [53, 54]. Furthermore, FB1 has demonstrated hepatotoxic, nephrotoxic, and immunosuppressive effects in various animal models [55].

5.4. Zearalenone

The International Agency for Research on Cancer (IARC) classifies zearalenone (ZEA) as possibly carcinogenic to humans [30]. Produced by *Fusarium* species and isolated from medicinal plants, ZEA has demonstrated cytotoxic effects in mammalian cells [56] and exhibits competitive binding to estrogen receptors in vitro [57].

ZEA exposure has been linked to a range of health effects, including early onset of puberty in children [58, 59, 60], esophageal cancer [61], cervical cancer, endometrial hyperplasia [62], and estrogenic effects in postmenopausal women [63]. Animal studies further confirm ZEA's adverse impact on reproductive physiology [64, 65, 66], as well as its potential to induce chronic progressive hematotoxicity, testicular atrophy, cataracts, retinopathy and nephropathy [67].

5.5. Trichothecenes (Deoxynivalenol and T-2)

Deoxynivalenol (DON) has been shown to alter brain neurochemistry [68]. Chronic exposure to low doses of DON can impair growth in children [69], while high doses may induce vomiting and, in severe cases, can be fatal [70, 71]. Additionally, DON exerts immunosuppressive effects [72].

T-2 toxin is associated with a range of toxic effects, including carcinogenesis, immunosuppression, neurotransmitter imbalances, weight loss, growth retardation, oral lesions, diarrhea

and vomiting [73, 74, 75]. Epidemiological studies have also linked T-2 contamination to Kashin-Beck disease, a chronic osteoarthropathy [76].

Experimental exposure to T-2 has caused degenerative joint changes in animal models [77, 78, 79]. Moreover, a single dose of T-2 administered to rats resulted in metabolic disruptions, alterations in metabolic pathways, and changes in the intestinal microbiota [75]. Table 1 summarizes the characteristics of contamination in medicinal plant products.

Table 1. Mycotoxins contaminating medicinal plant products

Mycotoxin	Main Source	Major Toxic Effects	IARC Classification	LD_{50} Range
Aflatoxin B_1	*Aspergillus flavus*, *A. parasiticus*	Severe hepatotoxicity, mutagenic, immunosuppressive, highly potent liver carcinogen	Group 1 (carcinogenic to humans)	~0.3–1 mg/kg (rat, oral)
Aflatoxins (B_2, G_1, G_2)	*A. flavus*, *A. parasiticus*	Similar effects to AFB_1 but less potent: hepatotoxicity, genotoxicity	G1: Group 1; B2, G2: Group 2B	1–10 mg/kg (rat, oral)
Ochratoxin A (OTA)	*Aspergillus ochraceus*, *A. carbonarius*, *Penicillium verrucosum*	Nephrotoxic, immunotoxic, teratogenic, possible renal carcinogen	Group 2B	20–50 mg/kg (rat, oral)
Zearalenone (ZEN)	*Fusarium graminearum*, *F. culmorum*	Estrogenic activity (endocrine disruptor), fertility disorders	Group 3	1–3 g/kg (rat, oral)
Fumonisins (especially FB_1)	*Fusarium verticillioides*, *F. proliferatum*	Nephrotoxicity, hepatotoxicity, disruption of sphingolipid metabolism	Group 2B	10–20 mg/kg (rat, oral)
T-2 Toxin (Trichothecene)	*Fusarium sporotrichioides*, *F. poae*	Strong inhibition of protein synthesis, dermal toxicity, severe immunosuppression	Group 3	1–5 mg/kg (mouse, oral)
Deoxynivalenol (DON / Vomitoxin)	*Fusarium graminearum*, *F. culmorum*	Immunosuppression, vomiting, anorexia; inhibition of protein synthesis	Group 3	46–78 mg/kg (mouse, oral)

6. Prevention

6.1. Physical methods

Improved harvesting, drying and post-harvest processes have significantly reduced ochratoxin A levels in medicinal plants [80]. Gamma radiation treatment has proven to be an effective method for preventing fungal deterioration in medicinal plants during long-term storage. In one study, gamma irradiation reduced the colony-forming units (CFU) per gram in all treated samples—including *Peumus boldus*, *Camellia sinensis*, *Maytenus ilicifolia*, and *Cassia angustifolia*—after 30 days of storage, with no detectable aflatoxin contamination [81]. However, heat treatment is generally ineffective since aflatoxins are heat-stable compounds [82].

6.2. Biological methods

Several authors recommend the use of essential oils as natural alternatives to synthetic fungicides for preventing fungal contamination and mycotoxin production in medicinal plants. In vitro studies demonstrated that 16 essential oils—such as orange, bergamot, lemon, coriander, eucalyptus, anise, lavender, chamomile (blue), tea tree, basil, geranium, rose, savory, thyme, viola, and oregano—exhibited antifungal activity against 21 fungal strains isolated from herbal drugs [83]. Another study showed that

essential oils derived from 12 medicinal plants, including anise, caraway, fennel, thyme, spearmint, basil, chamomile, marigold, hazanbul, qyssum, ghafath, and cinnamon, effectively inhibited both the growth and mycotoxin production of *Aspergillus flavus*, *Aspergillus parasiticus*, *Aspergillus ochraceus*, and *Fusarium moniliforme*, with the degree of inhibition depending on the concentration of the oils used [84].

In addition to essential oils, phenolic compounds found in plants offer promising antifungal properties. Compounds such as chlorophorin, iroko, maakianin, caffeic acid, ferulic acid, and vanillic acid have demonstrated varying degrees of inhibition against the growth of *Fusarium verticillioides* and its production of fumonisin B1 (FB1) [85]. Furthermore, Ajowan seed extract (*Trachyspermum ammi* (L.) Sprague ex Turrill) has shown potential as a biologically safe method to protect herbal medicines from aflatoxins by degrading AFG1, AFG2, AFB1, and AFB2 [86].

7. Conclusions

The data presented in this review clearly indicate that fungal contamination of herbal products is unavoidable, with specific factors favoring the growth of toxigenic strains and the production of mycotoxins. To protect consumer safety, the European Pharmacopoeia has established strict limits for the most hazardous mycotoxins in medicinal plants and their derivatives. Numerous studies report varying contamination levels, influenced by factors such as the country of origin, the plant species, and the detection methods employed. Mycotoxicity is linked to a range of human health issues, with clinical symptoms depending on the type and dose of mycotoxins ingested. Therefore, to ensure the safety of herbal medicines and their products, it is crucial to implement strategies that utilize natural substances for decontamination and prevention of mycotoxin formation.

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