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Serial Urinary Mycotoxin Trends and Clinical Response During Binder Therapy: A Retrospective Case Series

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Abstract

Background

Diagnostic approaches for suspected mold-related illness vary widely. Urinary mycotoxin testing is increasingly used in outpatient practice because it is easy to obtain and provides a quantitative marker that can be followed over time, though its interpretation remains controversial. Elevated urinary mycotoxins are often used with symptoms to support case definition and justify binder-based treatment, even when exposure history is uncertain.

Objective

The aim of this study is to describe serial urinary mycotoxin results, symptom trajectories, binder exposure, and concurrent interventions in a retrospective observational case series of patients receiving binder-centered care for suspected mold-related illness.

Methods

We performed a retrospective case series of six patients treated in an outpatient integrative medicine setting. All had elevated urinary mycotoxins on RealTime Laboratories (Carrollton, Texas, USA) testing and received binder-based protocols centered on activated charcoal, bentonite clay, and chlorella. Clinical outcomes were assessed using patient-reported global improvement estimates and qualitative follow-up documentation. Urinary mycotoxin testing was repeated over time to evaluate biomarker trends.

Results

Urinary mycotoxin trends were variable and did not consistently correspond with clinical outcomes. Some patients showed reductions despite little meaningful improvement, whereas others improved while urinary trends remained mixed or worsened. Clinical improvement observed during binder-based care was often limited or non-sustained, and several patients remained stable or improved after discontinuing binders.

Conclusions

Across these six cases, urinary mycotoxin trends did not consistently parallel patient-reported symptom trajectories. Clinical improvement during binder-based care was heterogeneous and frequently occurred in the context of other concurrent or subsequent interventions. These observations are descriptive and hypothesis-generating and do not establish the validity of urinary mycotoxin testing or the effectiveness of binder therapy.

Categories: Therapeutics, Environmental Health, Integrative/Complementary Medicine
Keywords: binder therapy, integrative medicine, mold-related illness, retrospective case series, urinary mycotoxin testing

Introduction

Exposure to water-damaged buildings has been associated with a range of respiratory, allergic, and nonspecific chronic symptoms [1], yet the clinical and diagnostic approach to suspected mold-related illness remains variable and often poorly standardized. In outpatient environmental and integrative medicine, oral binders are commonly used on the premise that intestinal sequestration may reduce reabsorption of bile-associated toxins, including mycotoxins, which are toxic secondary metabolites produced by certain molds [2]. Urinary mycotoxin testing is also commonly used in some outpatient settings because it provides a quantitative marker that can be followed over time.

Interpretation of urinary mycotoxin testing remains controversial [3]. Some studies report higher urinary mycotoxin detection in environmentally exposed individuals and in selected chronic illness cohorts than in

control populations [4,5], while others suggest that urinary mycotoxin levels may reflect dietary exposure more strongly than environmental burden [6-8]. Major organizations, including the Centers for Disease Control and Prevention and the American College of Medical Toxicology, have cautioned against routine clinical use or overinterpretation of urinary mycotoxin testing because clinical validity and interpretability remain limited [9,10]. At the same time, urinary mycotoxin testing continues to be used in clinical practice because it offers an objective marker in a field where symptoms are often nonspecific and environmental histories may be incomplete.

Urinary mycotoxin testing is often used in clinical practice to help define the case and guide treatment selection [11]. In many settings, elevated urinary mycotoxins plus compatible symptoms are treated as sufficient justification to initiate binder-based care, sometimes before other explanatory models or treatment domains are explored in depth [9,11].

This case series describes the clinical course of six patients in whom urinary mycotoxin findings contributed to case definition, binder-based treatment decisions, and longitudinal monitoring. The aim was to characterize serial urinary mycotoxin results, binder exposure, concurrent care, and patient-reported symptom trajectories and to describe concordance or discordance between urinary trends and clinical course.

Materials And Methods

A retrospective observational case series was conducted using electronic medical records from a telehealth integrative health clinic operating in the United States. All treating clinicians and included patients were located in the United States during the study period. The study interval, March 2021 through July 2023, corresponded to the period during which urinary mycotoxin testing was used to inform binder therapy in routine clinical practice. Electronic medical records were manually screened to identify patients who received binder therapy during this interval. Nine potentially eligible records were assessed against the complete eligibility criteria, of which six met all criteria and were included. No sample size calculation or statistical power analysis was performed because all eligible cases identified within the defined study interval were included.

Eligible patients had baseline urinary mycotoxin testing used in clinical evaluation. Included cases had elevated urinary mycotoxin results at baseline, received binder-based treatment as part of their care, had at least one baseline and one follow-up urinary mycotoxin test, and had clinical documentation of patient-reported symptoms. Patients were excluded if clinical records lacked sufficient documentation of laboratory results or symptom trajectories. Three patients were excluded due to lack of follow-up urinary mycotoxin testing and were therefore not included in the analysis.

All six eligible patients underwent urinary mycotoxin testing through RealTime Laboratories (Carrollton, Texas, USA). Testing was performed using enzyme-linked immunosorbent assay methodology to detect urinary metabolites of ochratoxin, aflatoxin, trichothecenes, zearalenone, and gliotoxin. Results were interpreted using the laboratory's reference ranges and followed descriptively over time.

All patients received binder-based protocols centered on activated charcoal, bentonite clay, and chlorella. Treatment duration and dosing were individualized and are described in the case study summaries below. Follow-up assessments and laboratory testing occurred at non-standardized intervals and reflected real-world practice. Concurrent therapies were not standardized and are described below.

Symptom improvement was quantified using the patient-reported self-estimated percentage of overall improvement relative to baseline or from the prior visit, supplemented by qualitative clinical documentation from follow-up visits.

The primary outcome was a descriptive comparison of serial urinary mycotoxin results with patient-reported symptom trajectories during the documented treatment course. Given the small sample size, data were analyzed descriptively. Findings were presented as case summaries and overall patterns, with attention to concordance or discordance between urinary mycotoxin trends and clinical course. Quantitative variables were evaluated descriptively as continuous measures without categorization or transformation. Urinary trends were assessed within individual patients, without between-group comparisons, time adjustment, or inferential statistical analyses.

This study was conducted in accordance with the Declaration of Helsinki and was approved by the Helfgott Institutional Review Board, Portland, Oregon, USA (approval no. 00002896) for one year beginning April 9, 2026. The Helfgott Institutional Review Board waived the requirement for patient informed consent for the collection, analysis, and publication of retrospectively obtained, anonymized data for this non-interventional study.

Results

Overall patterns

Six patients met inclusion criteria and were included in the analysis (three female and three male patients; mean age, 33 years; age range, 30-37 years). Presenting symptoms included fatigue, cognitive dysfunction, gastrointestinal symptoms, respiratory complaints, dermatologic manifestations, pain, and immune-type symptoms. Environmental mold exposure certainty varied across cases, ranging from more plausible or documented exposure histories to unknown, weak, or chronologically inconsistent.

Average binder duration was 11 months (range six to 24 months). Symptom trajectories during binder therapy were heterogeneous. In four cases (Cases 1-3 and 6), improvement during binder therapy was limited or not sustained. Binder dosing was generally conservative and individualized. Documented dosing ranged from one capsule each daily to six to eight capsules each daily. Four patients began with one capsule each daily with plans to increase to two capsules each daily as tolerated; one patient was later increased to two to four capsules each daily, and one patient was titrated to six to eight capsules each daily. Flax fiber (one to three teaspoons with meals) was used in several cases, and modified citrus pectin (one teaspoon) was added in one case. Across these six cases, higher documented binder doses were not consistently accompanied by greater or more sustained clinical improvement.

Urinary mycotoxin trends were heterogeneous and non-linear. Two cases (Cases 2 and 5) showed substantial reduction in urinary mycotoxins, whereas the other four cases showed mixed, fluctuating, or rising patterns. No consistent relationship was observed between urinary mycotoxin trends and symptom trajectories.

In several cases, the clearest clinical improvement appeared temporally associated with interventions outside of binder therapy, including mast cell-targeted treatment, nervous system regulation strategies, antimicrobial therapy, psychiatric medication changes, exercise, stress reduction, and gastrointestinal-focused care. Several patients also remained stable or continued to improve after discontinuing binders.

Case 1: biomarker-symptom discordance despite higher binder dose and falling urinary mycotoxins

A 32-year-old woman with a history of visible mold exposure in a prior home, followed by mold identified in her current home, presented with fatigue, brain fog, chronic pain, depressive symptoms, and gastrointestinal complaints. She was treated with binder therapy for approximately ten months, with gradual titration to six to eight capsules each of activated charcoal, clay, and chlorella daily. She initially reported 40% improvement one month after binder initiation, although lithium and lamotrigine had been started shortly beforehand, making it difficult to attribute this improvement specifically to binders. This early improvement was not sustained, and symptoms later fluctuated. Follow-up urinary mycotoxins were higher than baseline during a period of symptomatic improvement, then later fell below baseline when she reported worsening symptoms, illustrating biomarker-symptom discordance. The clinical course suggested that her later improvement was more temporally aligned with psychiatric medication adjustment, exercise, and low-dose naltrexone than with binder therapy alone, and she subsequently reported feeling the best she had felt in four years. Serial urinary mycotoxin results for Case 1 are shown in Figure *1*.

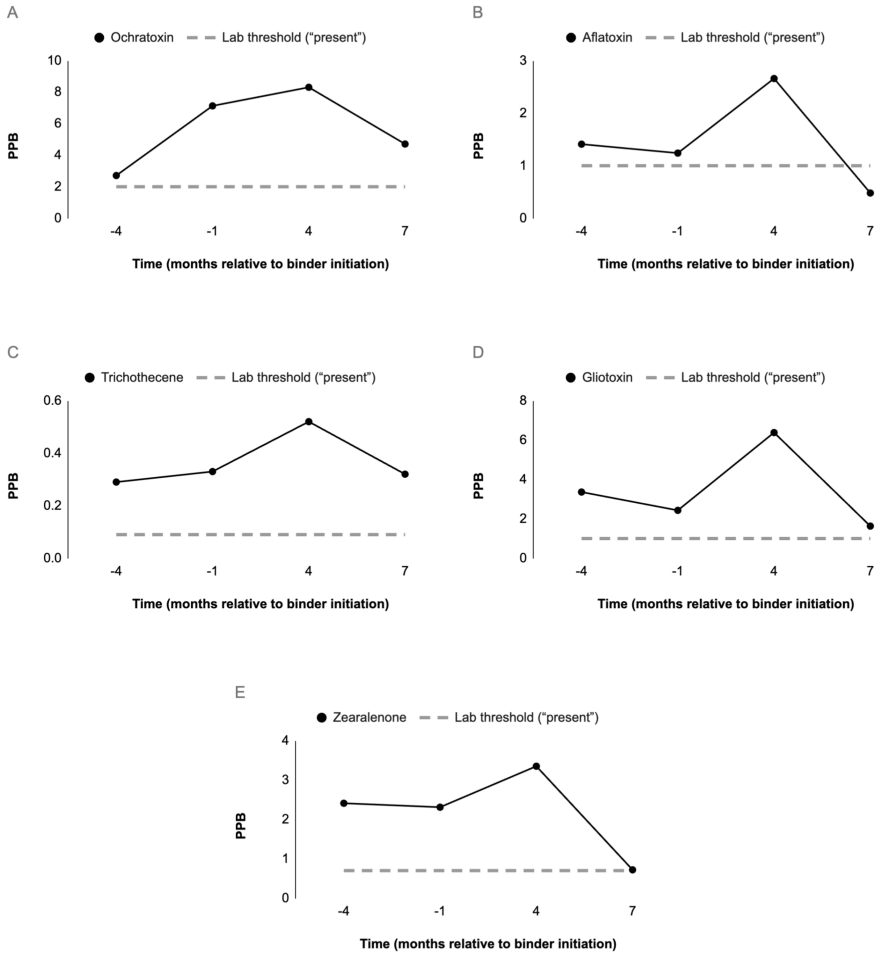


FIGURE 1: Serial urinary mycotoxin results for Case 1

Serial urinary concentrations of (A) ochratoxin, (B) aflatoxin, (C) trichothecene, (D) gliotoxin, and (E) zearalenone before and after binder initiation. Time is expressed in months relative to binder initiation. Black circles connected by solid lines represent measured concentrations; dashed gray lines represent the laboratory threshold for a result classified as present. PPB, parts per billion.

Case 2: minimal clinical gain despite reduced urinary mycotoxins

A 33-year-old woman without a clearly established mold exposure history presented with fatigue, cognitive dysfunction, gastrointestinal symptoms, and respiratory complaints. Limbic retraining and mast cell-targeted therapy were initiated before binders. She was treated with binder therapy for approximately seven months. Early response to binders was minimal. Binder dosing was then increased, and herbal antimicrobials were added, after which she reported a transient improvement. Follow-up urinary mycotoxins decreased substantially, but symptoms later returned to baseline despite these lower urinary levels. No clear period of meaningful improvement attributable specifically to binders was documented, and the brief later improvement was modest, non-sustained, and temporally confounded by the addition of herbal antimicrobials. Serial urinary mycotoxin results for Case 2 are shown in Figure *2*.

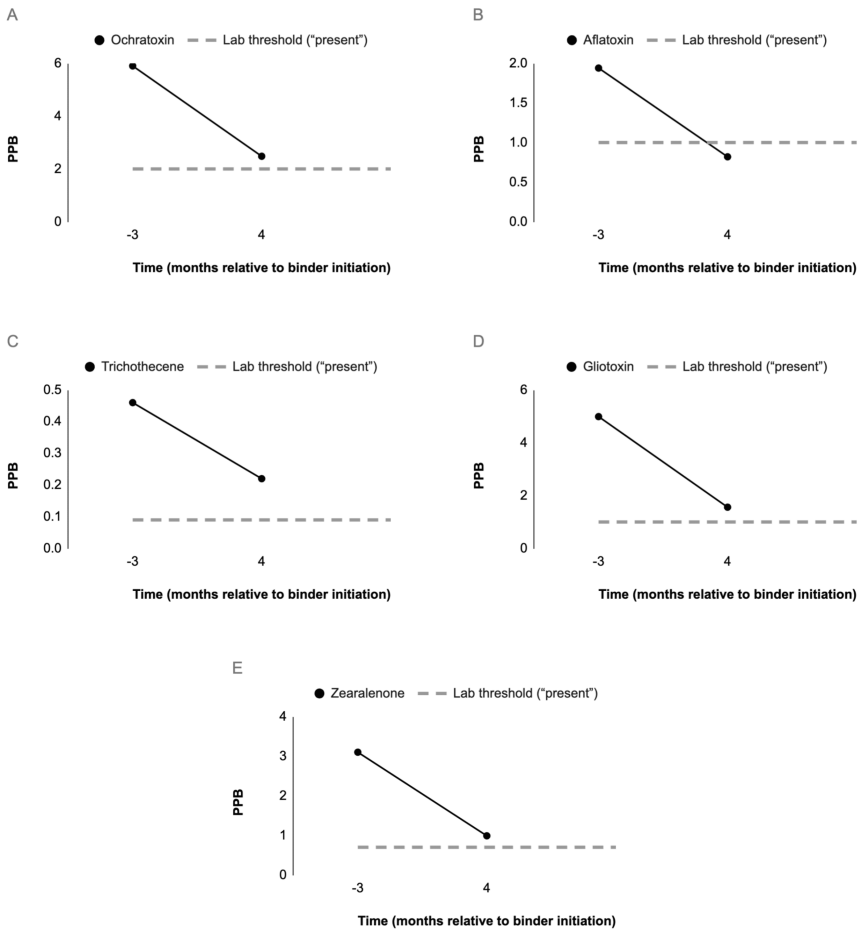


FIGURE 2: Serial urinary mycotoxin results for Case 2

Serial urinary concentrations of (A) ochratoxin, (B) aflatoxin, (C) trichothecene, (D) gliotoxin, and (E) zearalenone before and after binder initiation. Time is expressed in months relative to binder initiation. Black circles connected by solid lines represent measured concentrations; dashed gray lines represent the laboratory threshold for a result classified as present. PPB, parts per billion.

Case 3: symptomatic improvement despite rising urinary mycotoxins

A 32-year-old woman with suspected but unconfirmed mold exposure in a prior residence presented with migraines, asthma, and eczema. She was treated with binders for approximately 11 months. Early response to binders was modest, with 10% self-reported improvement at five months, and dosing was later increased to two to four capsules each of activated charcoal, clay, and chlorella daily. By 11 months, she reported 20% improvement, but also stated that she was unclear whether binders had been helpful and that the most noticeable benefit had occurred during a period of lower stress and increased exercise. Follow-up urinary mycotoxins were mixed initially and later increased across all measured toxins. Her reported improvement occurred during a period of reduced stress and increased exercise, while urinary mycotoxin levels increased and she remained uncertain whether binders had been helpful. Serial urinary mycotoxin results for Case 3 are shown in Figure *3*.

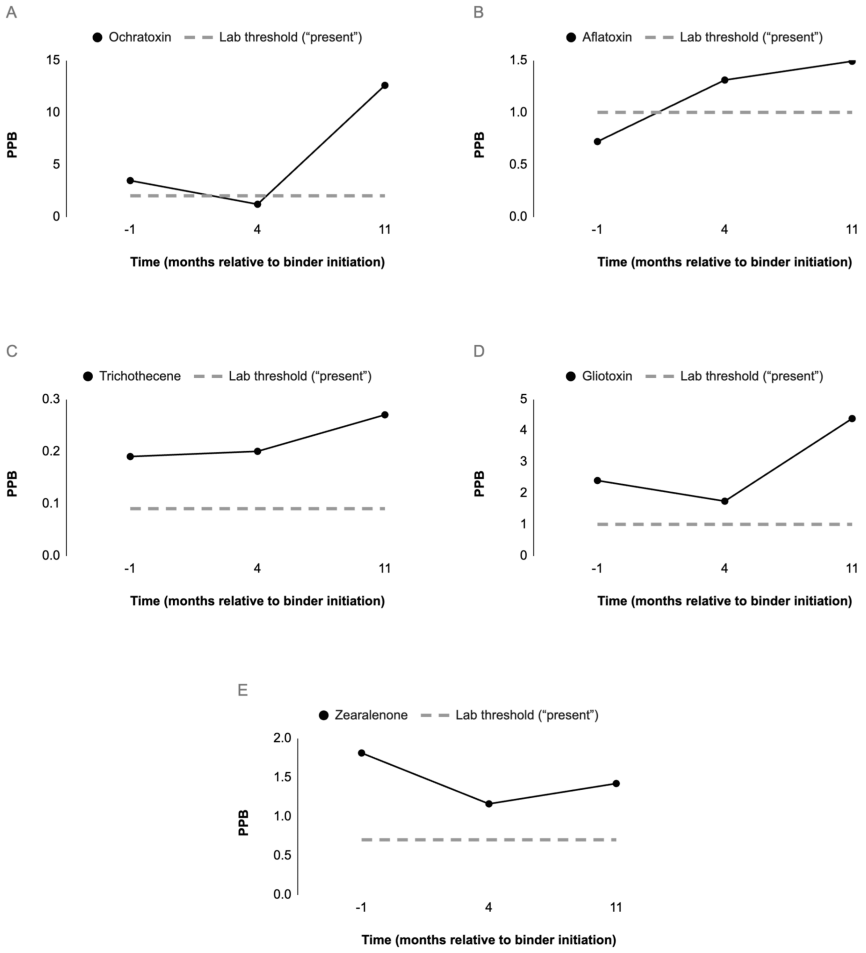


FIGURE 3: Serial urinary mycotoxin results for Case 3

Serial urinary concentrations of (A) ochratoxin, (B) aflatoxin, (C) trichothecene, (D) gliotoxin, and (E) zearalenone before and after binder initiation. Time is expressed in months relative to binder initiation. Black circles connected by solid lines represent measured concentrations; dashed gray lines represent the laboratory threshold for a result classified as present. PPB, parts per billion.

Case 4: modest improvement during binder-based care with mixed urinary mycotoxin trends and patient-attributed benefit from lifestyle interventions

A 30-year-old man with exposure to a musty, previously water-damaged basement, though without confirmed visible mold, presented with brain fog, fatigue, joint pain, and loose stool. He was treated with binders for approximately eight months. Early improvement was most apparent in loose stool and joint pain, which largely resolved. Later, he also reported improvement in brain fog and fatigue. However, he specifically stated that his greatest gains in resiliency, cognition, and fatigue occurred during periods of cold showers, sauna, resistance training, and high-intensity exercise. Follow-up urinary mycotoxins were initially reduced and later mixed. Among the six cases, the early improvement in joint pain and loose stool showed the closest temporal association with binder initiation, whereas the broader gains were attributed by the patient primarily to lifestyle interventions. Serial urinary mycotoxin results for Case 4 are shown in Figure *4*.

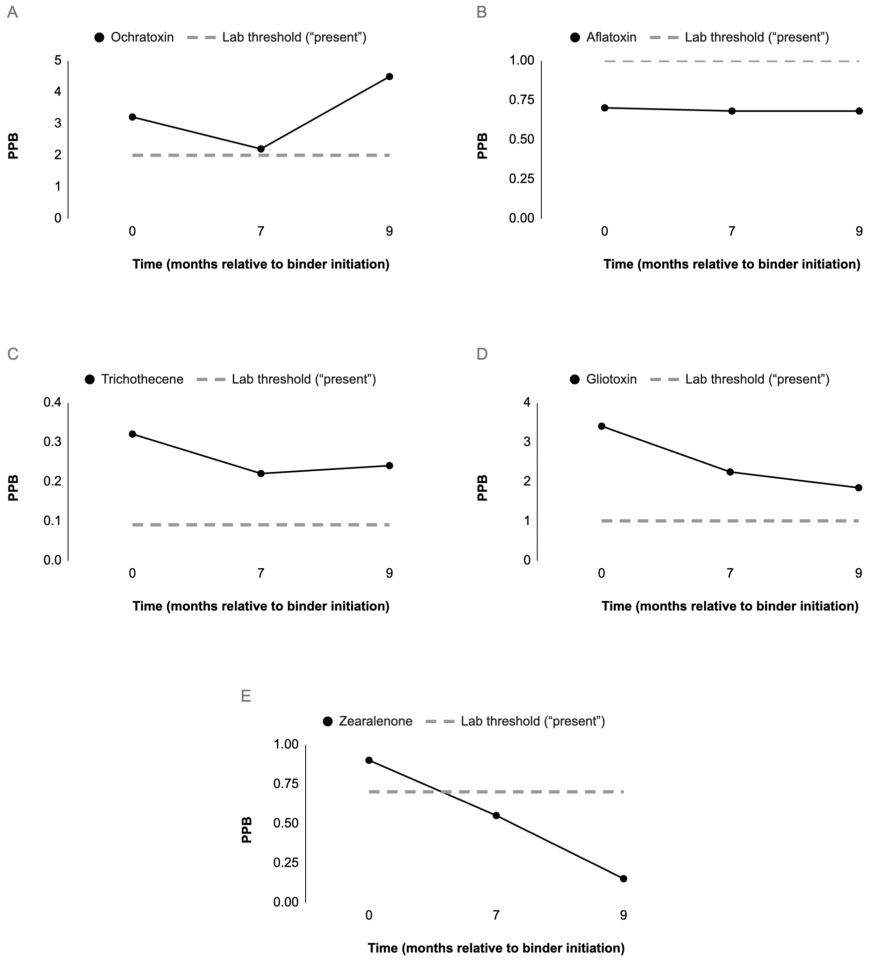


FIGURE 4: Serial urinary mycotoxin results for Case 4

Serial urinary concentrations of (A) ochratoxin, (B) aflatoxin, (C) trichothecene, (D) gliotoxin, and (E) zearalenone before and after binder initiation. Time is expressed in months relative to binder initiation. Black circles connected by solid lines represent measured concentrations; dashed gray lines represent the laboratory threshold for a result classified as present. PPB, parts per billion.

Case 5: major recovery with falling urinary mycotoxins despite no home remediation

A 37-year-old man with mold identified in his home, although with symptom onset predating residence there, presented with chronic idiopathic urticaria, angioedema, post-prandial fatigue, tingling/numbness, and food-reactive symptoms. Prior to binder initiation, he received mast cell-targeted therapy and reported a 25% overall improvement, which he described as the most relief he had experienced in 10 years. He then underwent binder therapy for approximately six months. After binder initiation, he reported some additional short-term improvement, but later felt better after reducing and then discontinuing binders. At seven months off binders, he reported 90%-95% overall recovery and attributed the greatest benefit to mast cell-targeted care, exercise, breathwork, and cold showers. Follow-up urinary mycotoxins fell substantially during treatment, despite the home never being remediated. Overall, the largest improvement began before binder initiation, and continued recovery was documented after binders were reduced and discontinued. Serial urinary mycotoxin results for Case 5 are shown in Figure *5*.

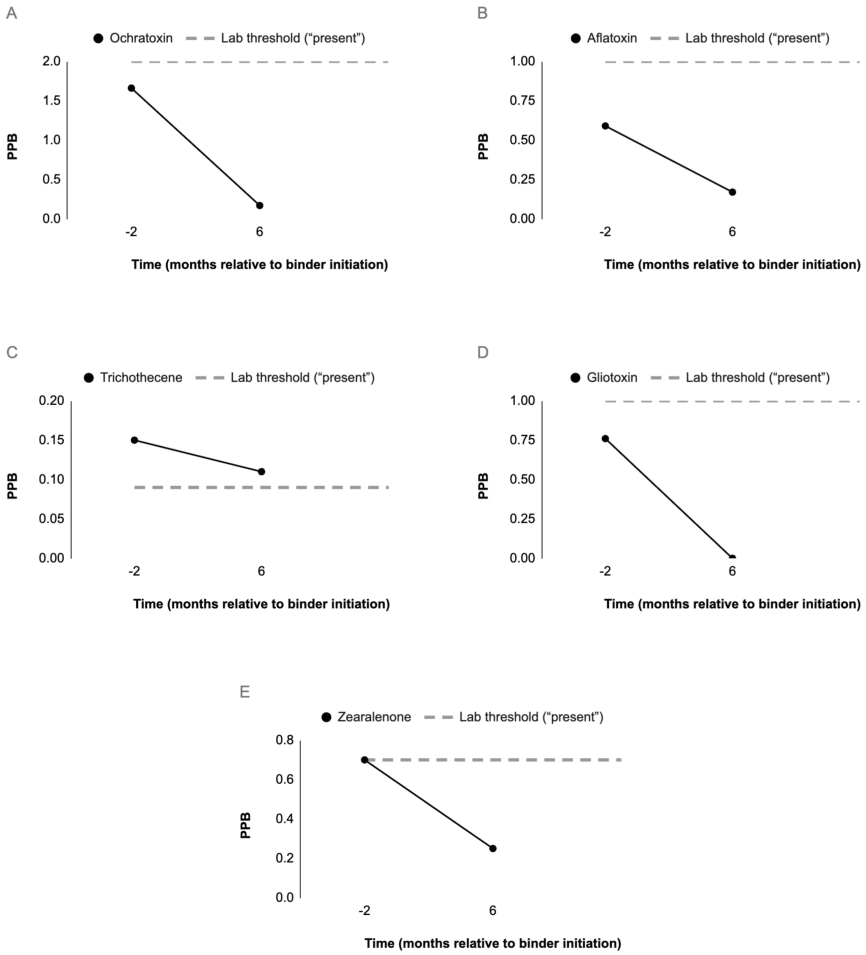


FIGURE 5: Serial urinary mycotoxin results for Case 5

Serial urinary concentrations of (A) ochratoxin, (B) aflatoxin, (C) trichothecene, (D) gliotoxin, and (E) zearalenone before and after binder initiation. Time is expressed in months relative to binder initiation. Black circles connected by solid lines represent measured concentrations; dashed gray lines represent the laboratory threshold for a result classified as present. PPB, parts per billion.

Case 6: major pre-binder improvement, with mixed later urinary mycotoxins

A 34-year-old man with a prior history of living in a moldy apartment, but without symptom onset clearly following that exposure, presented with ulcerative colitis, fatigue, brain fog, intermittent bloody/mucusy stools during flares, and acne. Prior to binder initiation, he underwent gastrointestinal-focused treatment with autoimmune paleo diet, elemental diet, herbal antimicrobials, probiotics, and ImmunoLin, and reported 90% overall improvement. He then underwent binder therapy for approximately two years. Two months into his binder course, he experienced regression in fatigue and bloody/mucusy stools, and over the subsequent binder phase he reported little to no major additional benefit, aside from possible minor gains in food tolerance and exercise resiliency. Follow-up urinary mycotoxins were mixed, with later increases in several markers. The largest improvement was documented before binder initiation, during gastrointestinal-directed treatment. No major additional improvement was documented during the subsequent binder-treatment period. Serial urinary mycotoxin results for Case 6 are shown in Figure *6*.

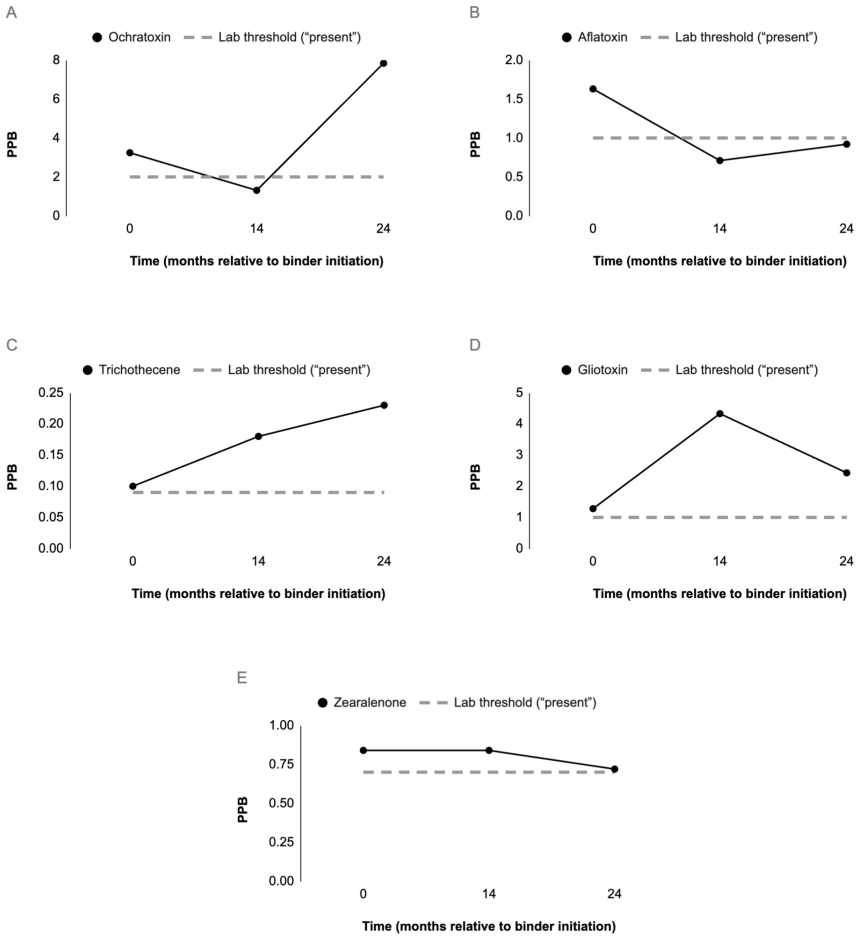


FIGURE 6: Serial urinary mycotoxin results for Case 6

Serial urinary concentrations of (A) ochratoxin, (B) aflatoxin, (C) trichothecene, (D) gliotoxin, and (E) zearalenone before and after binder initiation. Time is expressed in months relative to binder initiation. Black circles connected by solid lines represent measured concentrations; dashed gray lines represent the laboratory threshold for a result classified as present. PPB, parts per billion.

Summary of the cases

Exposure histories, urinary mycotoxin trends, clinical responses during binder therapy, and interventions associated with improvement are summarized in Table *1*.

Case	Exposure history	Urinary mycotoxin trend	Clinical response with binders	Interventions associated with improvement
1	Prior visible mold exposure; later mold identified in current home	Mycotoxins higher during symptom improvement, and later lower when symptoms worsened	Early symptom improvement not sustained	Medication changes + exercise + low-dose naltrexone
2	No clearly established mold exposure history	Mycotoxins decreased substantially	Minimal overall symptom improvement	Binder escalation + later antimicrobials
3	Suspected, but unconfirmed prior mold exposure	Four of five mycotoxins increased	Mild symptomatic improvement despite rising mycotoxin levels	Stress reduction + exercise
4	Exposure to musty, water-damaged basement; mold unconfirmed	Mycotoxins initially lower, later mixed	Modest improvement in joint pain & loose stools	Binders for joint pain & loose stools Exercise + sauna + cold exposure for resiliency, brain fog & fatigue
5	Mold confirmed in home upon mold inspection; symptoms predated residence	Mycotoxins fell substantially despite no remediation	Symptom improvements began pre-binder, felt better & maintained progress when off binders	Mast cell activation syndrome treatment + exercise + breathwork
6	Prior moldy residence; symptoms did not follow exposure	Mycotoxins mixed initially; later several markers increased	Major improvement occurred pre-binder; no meaningful improvement after binders	Gastrointestinal- directed therapy

TABLE 1: Case-specific summary of exposure context, urinary mycotoxin trends, clinical response during binder-centered care, and the interventions most closely associated with improvement.

Several cases had uncertain or chronologically inconsistent exposure histories. Urinary mycotoxin trends were variable and did not reliably track with symptom trajectories. In multiple cases, the clearest improvement occurred during periods involving treatments outside of binders or was attributed by the patient to those interventions.

Discussion

Summary of observations

Across these six cases, urinary mycotoxin results contributed to case definition, binder-based treatment decisions, and longitudinal monitoring. Serial urinary mycotoxin trends did not consistently parallel patient-reported symptom trajectories, and clinical courses during binder-based care were heterogeneous. This series describes what occurred following this clinical decision pathway in routine practice; it does not validate the assay or estimate the effectiveness of binder therapy.

These cases do not establish that urinary mycotoxin testing is uniformly invalid or never clinically useful. Rather, they show that elevated baseline urinary mycotoxin findings were followed by variable clinical courses during binder-based care. This series does not address all uses of urinary mycotoxin testing, and the findings should be interpreted as descriptive and hypothesis-generating rather than as an assessment of the test's ability to predict treatment response.

Comparison with prior urinary mycotoxin studies

Previous studies have demonstrated that urinary mycotoxins can be detected in selected populations with reported mold exposure or chronic multisystem illness. Hooper et al. detected mycotoxins in urine and other biological specimens from patients with histories of environmental mold exposure, while specimens from patients without known exposure were below the study's detection thresholds [5]. Brewer et al. subsequently reported that 104 of 112 patients (93%) with chronic fatigue syndrome had at least one urinary mycotoxin detected, with more than 90% reporting current or prior exposure to a water-damaged building [4]. Similarly, Wu et al. reported at least one urinary mycotoxin in 92.4% of 236 patients with myalgic encephalomyelitis/chronic fatigue syndrome and a reported history of chronic mold exposure [12].

These studies support the observation that urinary mycotoxins may be detectable in selected symptomatic and mold-exposed populations. However, they primarily evaluated the presence or prevalence of urinary

mycotoxins rather than whether urinary concentrations correspond with illness severity or change in parallel with clinical recovery [4,5,12]. The present series, therefore, addresses a different clinical question. Although all six patients had elevated baseline urinary mycotoxins, subsequent changes in those values frequently did not correspond with symptom trajectories.

Interpretation is further complicated by evidence that urinary mycotoxins can reflect exposures other than water-damaged buildings. Human biomonitoring studies have detected urinary mycotoxin biomarkers in populations exposed through diet and occupational settings and have identified food consumption as an important contributor to urinary mycotoxin exposure [6-8]. Consequently, detection in urine establishes that a compound or metabolite has been excreted but does not necessarily identify its source or establish that it is responsible for a patient's symptoms. This distinction is consistent with cautions from the Centers for Disease Control and Prevention and the American College of Medical Toxicology regarding the clinical interpretation of urinary mycotoxin testing for illness attributed to indoor mold exposure [9,10].

Exposure histories were uncertain or chronologically inconsistent in several cases

Case definition often relied heavily on elevated urinary mycotoxins plus symptoms, even when environmental exposure was unknown, weakly supported, or chronologically inconsistent. Some patients had plausible or documented exposure histories, but others did not. For example, in Case 5, symptoms began before the home in which mold was later identified, whereas in Case 6, the reported mold exposure occurred in a prior residence and did not clearly align with symptom onset.

This differs from several published reports in which stronger evidence of environmental exposure formed part of the clinical case definition. Brewer et al. reported current or previous water-damaged-building exposure in more than 90% of their cohort [4], while the patients studied by Shoemaker and House were selected on the basis of exposure potential to water-damaged buildings in addition to multisystem symptoms [13]. The case reported by Quinn and Sandberg-Lewis similarly involved a documented long-term household mold exposure that ultimately required remediation [14]. These differences may be clinically relevant. The discordance observed in the present series raises the hypothesis that urinary findings may be more informative when interpreted alongside a coherent exposure history and symptom chronology rather than as a stand-alone case-defining marker. This series cannot determine how these factors should be weighted in clinical decision-making.

Improvement was not clearly attributable to binder-based care

Binder-based care often did not produce meaningful or sustained improvement in this cohort. Most patients experienced limited net change, and early improvement was frequently not sustained. Although binder dosing was often titrated upward as tolerated, no consistent relationship was observed between greater binder exposure and more meaningful or sustained clinical improvement. In multiple cases, the strongest or most durable gains appeared more closely linked to other treatment domains.

These findings differ from those reported by Shoemaker and House, who evaluated cholestyramine in patients with multisystem illness associated with water-damaged buildings [13]. In their study, 26 patients completed a prospective time-series protocol and 13 participated in a double-blind, placebo-controlled component. Two weeks of cholestyramine was associated with improvement in symptom counts and visual contrast sensitivity, whereas patients initially receiving placebo did not demonstrate comparable improvement during the placebo period. Symptoms subsequently worsened following re-exposure to the water-damaged buildings and improved again following another course of cholestyramine [13].

Several important differences limit direct comparison with the present series. Shoemaker and House used cholestyramine rather than the activated charcoal, bentonite clay, and chlorella protocols used in our patients; selected patients according to a specific exposure and multisystem illness framework; used a relatively short protocol with standardized outcome measures; and did not evaluate serial urinary mycotoxin concentrations as a marker of treatment response [13]. The contrasting clinical outcomes could, therefore, reflect differences in patient selection, exposure certainty, binder selection, treatment design, or other unmeasured factors rather than a true discrepancy in binder effectiveness.

A published case report by Quinn and Sandberg-Lewis provides another relevant comparison. Their patient improved within a broader environmental medicine approach that included confirmed environmental exposure and remediation as well as mast cell stabilization, gastrointestinal binders, sinus-directed therapy, and later antifungal treatment [14]. Because several treatments were employed, that report does not isolate the contribution of binders, but it illustrates a clinical scenario in which treatment was embedded within a more comprehensive exposure-directed strategy. In the present series, by comparison, exposure histories were frequently less certain and clinical improvement was often more closely associated with interventions outside of binder therapy.

Serial urinary trends and symptom trajectories were frequently

discordant

Urinary mycotoxin trends did not reliably correspond with symptom trajectories in this series. Some patients showed substantial reductions in urinary mycotoxins without meaningful improvement, whereas others improved while urinary trends were mixed or increased. In one case, the patient reported feeling better when mycotoxin levels were higher; in another, improvement occurred alongside increases across nearly all measured toxins.

Prior literature provides reasons to interpret such serial changes cautiously. Hope described a clinical case in which urinary mycotoxin concentrations increased substantially during glutathione administration without concurrent sequestrant therapy compared with periods when both were used, suggesting that urinary concentrations may be influenced by changes in mobilization and excretion rather than simply reflecting a static body burden [2]. Human biomonitoring studies also demonstrate that urinary mycotoxin concentrations can reflect ongoing dietary or occupational exposure [6-8]. Thus, changes in urinary concentration could theoretically reflect changes in exposure, absorption, metabolism, mobilization, or excretion.

Importantly, the published studies demonstrating urinary mycotoxin detection in mold-exposed or chronically ill populations have primarily evaluated cross-sectional detection or prevalence rather than validating serial urinary concentrations against longitudinal clinical outcomes [4,5,12]. The present cases, therefore, raise a clinically relevant hypothesis: a falling urinary mycotoxin concentration should not necessarily be assumed to indicate clinical recovery, nor should a stable or rising concentration necessarily indicate clinical deterioration. Prospective studies using standardized sampling, exposure assessment, treatment protocols, and validated symptom measures would be needed to determine whether serial urinary testing provides clinically meaningful monitoring information.

Observed findings and hypotheses

Taken together, the present findings are compatible with prior literature demonstrating that urinary mycotoxins can be detected in selected symptomatic or environmentally exposed patients [4,5,12], but they do not support assuming that the magnitude or direction of urinary values will reliably correspond with clinical status. They also differ from limited published evidence reporting clinical improvement following cholestyramine in more tightly selected water-damaged-building cohorts [13].

Several hypotheses could explain these differences, including variability in exposure certainty, dietary contributions to urinary levels, differences among binder agents, differences in patient selection, weak exposure-symptom linkage in some patients, or greater clinical importance of other treatment domains. These possibilities were not directly tested and should remain hypotheses rather than conclusions.

Clinically, these observations suggest that urinary mycotoxin findings may be best interpreted as one component of a broader assessment that includes environmental exposure history, symptom chronology, alternative explanations for chronic symptoms, and response to treatment. Similarly, serial urinary values should not substitute for assessment of the patient's clinical course. Larger prospective studies are needed to determine whether particular patient characteristics, exposure patterns, or treatment approaches identify subgroups in whom urinary mycotoxin testing or binder therapy has greater clinical utility.

Limitations

This retrospective case series included six patients with heterogeneous clinical presentations and mold exposure histories, individualized binder regimens, non-standardized follow-up intervals, and concurrent therapies. Symptom outcomes relied on patient-reported improvement estimates from routine clinical visits rather than validated instruments. These factors limit interpretation of clinical trends and attribution of improvement specifically to binders. Urinary mycotoxins were measured using a single enzyme-linked immunosorbent assay platform, without independent assessment of assay-specific variability or potential cross-reactivity.

Three of nine screened patients were excluded because they lacked repeat urinary mycotoxin testing, potentially introducing selection bias if retesting was related to symptoms or treatment response. Dietary mycotoxin exposure was not systematically assessed or controlled, and environmental mold exposure was not consistently confirmed, limiting interpretation of serial urinary concentrations. Because the series included only patients with elevated baseline urinary mycotoxins who received binders and underwent repeat testing, the findings may not generalize to other patients.

In the absence of a control group and standardized outcome assessments, causal effects of binder therapy cannot be determined. The findings should, therefore, be interpreted as descriptive and hypothesis-generating rather than evidence of urinary mycotoxin test validity or binder effectiveness.

Conclusions

In this retrospective case series of six patients receiving binder-centered care for suspected mold-related illness, serial urinary mycotoxin concentrations did not consistently correspond with patient-reported symptom trajectories. Clinical responses during binder therapy were heterogeneous, and greater binder exposure was not consistently associated with greater or more sustained improvement. In several cases, the clearest clinical gains occurred during interventions outside of binder therapy or persisted after binders were discontinued.

These findings do not establish the validity or invalidity of urinary mycotoxin testing or the effectiveness or ineffectiveness of binder therapy. Rather, they suggest that elevated urinary mycotoxin results and subsequent changes in those values should be interpreted within the broader clinical context, including environmental exposure history, symptom chronology, alternative explanations for illness, and the patient's clinical response. Prospective studies using standardized exposure assessment, treatment protocols, urinary testing procedures, and validated clinical outcomes are needed to determine whether urinary mycotoxin testing can reliably identify treatment-responsive subgroups or serve as a meaningful marker of clinical recovery.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Acquisition, analysis, or interpretation of data: Danielle Kemeny, Benjamin Zimmerman, Michael Ruscio, Scott Spiridigliozzi, Jacob Vonfeldt

Drafting of the manuscript: Danielle Kemeny, Scott Spiridigliozzi, Jacob Vonfeldt

Critical review of the manuscript for important intellectual content: Benjamin Zimmerman, Michael Ruscio, Scott Spiridigliozzi, Jacob Vonfeldt

Concept and design: Michael Ruscio, Scott Spiridigliozzi, Jacob Vonfeldt

Supervision: Michael Ruscio

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. Helfgott Institutional Review Board issued approval (00002896). **Animal subjects:** All authors have confirmed that this study did not involve animal subjects or tissue. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** The Ruscio Institute, LLC paid NUNM for research consulting services that included Benjamin Zimmerman's contributions to this manuscript. **Financial relationships:** Michael Ruscio declare(s) employment and stock/stock options from The Ruscio Institute for Functional Health. Compensation for clinical services and ownership/equity interest in The Ruscio Institute, LLC, which operates the Ruscio Institute for Functional Health. Jacob Vonfeldt and Scott Spiridigliozzi declare(s) employment from The Ruscio Institute for Functional Health. Employment from The Ruscio Institute, LLC. Provides clinical services. Danielle Kemeny declare(s) personal fees from The Ruscio Institute for Functional Health. The Ruscio Institute, LLC. Independent contractor providing research services. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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