

# Clinical Utility of Probiotics Therapy in Managing Mycotoxin Illness

Liam LaTouche, ND

**Liam LaTouche, Hon BSc Kin, ND**, in practice in Barrie, Ontario, and faculty member, Canadian School of Natural Nutrition in Toronto, Ontario, Canada.

Corresponding author: *Liam LaTouche, ND*  
E-mail address: *info@liamlatouche.com*

For those dealing with a complex chronic illness—such as chronic fatigue syndrome, fibromyalgia, frequent infections, neuroendocrine disorders, and autoimmunity—mycotoxins are often overlooked, yet significant, silent killers that deteriorate various systems in the body. The symptoms caused by mycotoxins are often nonspecific and overlap many other health issues, and it's common for standard test results from screening labs to either be normal or borderline abnormal. The subsequent band-aid approach to managing these symptoms may cause delays in proper treatment, potentially resulting in worsened outcomes.

Mycotoxins are secondary metabolites of mold compounds that act as significant triggers of chronic immune activation. Mold toxicity—mycotoxin illness—reflects an immune response to mycotoxin exposure; this is different from mold allergy—immunoglobulin E (IgE)-mediated, immediate hypersensitivity reaction—and mycosis—mold infections.

Common and concerning sources of mycotoxins include *Alternaria*, *Aspergillus*, *Claviceps*, *Fusarium*, and *Penicillium*. Individuals become exposed to mycotoxins through dermal, inhalation, and ingestion routes, although exposure through water-damaged buildings, such as from flooding, can be particularly problematic.

It's postulated that the systemic chronic inflammation associated with mycotoxin exposure relates to abnormalities of pro-inflammatory cytokines, complement activation, and cellular immunity,<sup>1</sup> contributing to various negative health effects (Figure 1).<sup>2</sup>

Management of chronic inflammation, and the subsequent symptoms, is difficult to achieve with persistent mycotoxin exposure; hence, the primary goal is eliminating the source of exposure. In addition, it's imperative to use binders to reduce mycotoxin burden while supporting detoxification and redox capacity to promote immune tolerance.

**Figure 1.** Health Effects of Common Mycotoxins.<sup>2</sup> Laboratory investigation generally involves urine testing for levels of various mycotoxin exposures, such as mass spectrometry or enzyme-linked immunosorbent assay (ELISA).

Mycotoxin	Health effect
Aflatoxins	Hepatotoxic and immunosuppressive
Ochratoxin A	Carcinogenic, genotoxic, immunosuppressive, nephrotoxic and induction of upper urinary tract disease
Fumonisin	Carcinogenic, hepatotoxic, nephrotoxic, immunosuppressive
Deoxynivalenol	Nausea, vomiting, diarrhea, reproductive effects and toxicosis
Tricothecenes	Hepatotoxic, genotoxic, and immunosuppressive
Zearalenone	Carcinogenic, hormonal imbalance, and reproductive effects
Patulin	Neurologic and gastrointestinal

## Signs/Symptoms of Mycotoxin Illness

Clinical presentation of mycotoxin illness can vary greatly from case-to-case, based on such factors as the nature of the exposure and genetic susceptibilities. These multisystem symptoms include but aren't limited to the following types:

General	Fatigue, night sweats, temperature dysregulation, electrical shocks
Musculoskeletal	Myalgia, arthralgia, arthritis
Neurological/psychiatric	Dysautonomia, cognitive impairment, multiple hypersensitivities, numbness/tingling, vertigo, mood swings
Head, ear, eyes, nose, throat:	Headache, chronic rhinosinusitis, conjunctivitis
Cardiorespiratory	Palpitations, shortness of breath, cough, asthma
Gastrointestinal	Abdominal pain, diarrhea, appetite changes, nausea
Genitourinary	Frequent urination

## Mycotoxin Illness Management

### Eliminate or Reduce Mold Exposure

Individuals should consider remediation of the affected space to whatever degree possible using experienced and trained professionals. Additionally, reducing the mycotoxin burden can be achieved with such methods as the use of a dehumidifier, with a relative humidity (RH) of 35-40%; use of a central air filter or portable air purifier; removal of all carpets; use of a high efficiency particulate air (HEPA) canister vacuum; and prevention of seasonal basement flooding.

### Detox and Redox Support

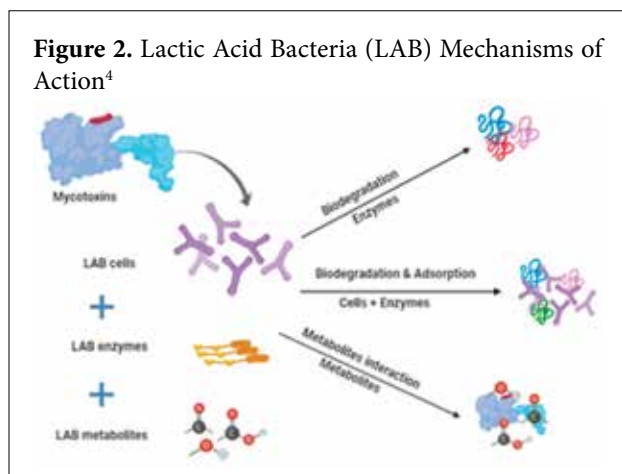
Supporting phase 2 detoxification pathways, namely glutathione conjugation and glucuronidation, is important in promoting the elimination of mycotoxins, managing

oxidative damage, and enhancing the threshold to toxic load. This is achieved with nutrients such as *N*-acetyl cysteine (NAC), vitamins C and E, selenium, and calcium D-glucarate. In addition, silybins from milk thistle are an excellent hepato-protectant that facilitates glutathione conjugation of xenotoxins, via the upregulation of glutathione *S*-transferase (GST).<sup>3</sup>

### Sequestering Agents (Binders)

Sequestering agents are used to bind mycotoxins in the digestive tract to aid in elimination by preventing enterohepatic recirculation.

Figure 2 shows the three primary mechanisms of action that appear to be involved in the binding and degradation of mycotoxins by lactic acid bacteria (LAB):<sup>4</sup> (1) degradation by LAB enzymes, (2) adsorption by LAB cells, and (3) the interaction of mycotoxins with LAB metabolites.



**Figure 2.** Lactic Acid Bacteria (LAB) Mechanisms of Action<sup>4</sup>

**Figure 3.** Application of Lactic Acid Bacteria (LAB) for Degradation of Mycotoxins<sup>9</sup>

Micronutrient	Target Mycotoxin	Degradation%
<i>Lactobacillus plantarum</i> CG, <i>L. plantarum</i> LC-709	Aflatoxin B1	80%
<i>L. amylophilus</i> , <i>L. plantarum</i>	Aflatoxin B1	50%
<i>L. casei</i> LUCK 0920, <i>L. brevis</i> LUCK 0944, <i>L. plantarum</i> LUCK 0945	Aflatoxins (B1, B2, G1, G2)	~50%
<i>L. plantarum</i> , <i>Lactobacillus lactis</i>	Aflatoxin B1	81%
<i>Streptococcus thermophilus</i> , <i>L. bulgaricus</i> , <i>L. plantarum</i>	Aflatoxin M1	11–74%
<i>L. casei</i>	Aflatoxin B1	49.2%
<i>L. paracasei</i> LUCK 0920, <i>L. brevis</i> LUCK 0944, <i>L. plantarum</i> LUCK 0945	Aflatoxin B1	36–55%
<i>L. plantarum</i> C99	Aflatoxin B1	90%
Lactic acid bacteria strains	Aflatoxins B1 and B2	ND
<i>L. casei</i> LUCK 0920, <i>L. brevis</i> LUCK 0944, <i>L. plantarum</i> LUCK 0945	Ochratoxin A	~50%
<i>L. aridophilus</i> VM21, <i>Rhodospirillum rubrum</i> VM12	Ochratoxin A	40%
<i>L. aridophilus</i> VM21, <i>Rhodospirillum rubrum</i> VM12	Fusarin	80%
<i>Pediococcus parvulus</i>	Ochratoxin A	90%
<i>L. plantarum</i> C99	Ochratoxin A	97%
<i>L. brevis</i> 2023	Fusarin	ND
<i>L. plantarum</i> GT 88	Deoxynivalenol	56–66%
Lactic acid bacteria strains	Deoxynivalenol, Zearalenone B1, Zearalenone B2	55%, 42%, and 100%
Lactic acid bacteria	Fusarin B1	36–47%
Lactic acid bacteria	Zearalenone	46–79%
<i>L. plantarum</i> CG, LUCK 0920, <i>L. plantarum</i> AL	Zearalenone	ND
<i>L. paracasei</i> , <i>L. bulgaricus</i>	Zearalenone	55%

LAB degrade mycotoxins by production of proteolytic enzymes and prevent their absorption through the digestive tract,<sup>5</sup> with several LAB strains being identified as having that capacity (Figure 3).<sup>6–9</sup>

*Lactobacillus rhamnosus* uses a variety of adsorption mechanisms, such as hydrogen bonding and ionic or hydrophobic interaction, to bind mycotoxins.<sup>10–12</sup>

Various LAB produce metabolites, such as acids; carbon dioxide (CO<sub>2</sub>); hydrogen peroxide (H<sub>2</sub>O<sub>2</sub>); phenyllactic acid; fatty acids; reuterin; and bioactive, low-molecular-weight peptides, that mitigate growth of mold and subsequent mycotoxin production.<sup>13,14</sup>

An RCT examining the effects of *Lactobacillus rhamnosus* LC705 and *Propionibacterium freudenreichii* subsp. *shermanii* strains on liver-cancer risk, mediated by aflatoxin exposure, found that a five-week intervention led to a statistically significant reduction in aflatoxin exposure in the intervention group and a decreased the risk of liver cancer.<sup>15</sup>

In addition to LAB, *Saccharomyces boulardii* (*S. boulardii*) has demonstrated mycotoxin binding effects,<sup>16</sup> as well as the ability to reverse mycotoxin-induced injury by attenuating the “p38 mitogen-activated protein kinase (MAPK)” signaling pathway to modulate expression of inflammatory cytokines and apoptotic genes.<sup>17</sup>

Activated charcoal (AC) has also been found to bind several mycotoxins, including aflatoxin B<sub>1</sub>, deoxynivalenol, ochratoxin, and zearalenone.<sup>18–20</sup> Due to possible concurrent adsorption of essential nutrients,<sup>21</sup> AC should be taken at least two hours after ingestion of food, medication, or supplements. Concomitant use of an oral multimineral formula or IV nutrient therapy can be considered based on the duration of the treatment.

### Conclusions

With the growing awareness of the multisystem health risks associated with mycotoxin exposure, probiotic therapy offers practitioners a valuable therapeutic tool in the management of mycotoxin illness, to be used in concert with detox and redox support and general exposure-avoidance strategies.

### References

- Chronic immune activation (Shoemaker, RC, et al. Research Committee Report on Diagnosis and Treatment of Chronic Inflammatory Response Syndrome Caused by Exposure to the Interior Environment of Water-Damaged Buildings. Research Committee Report on Diagnosis and Treatment of Chronic Inflammatory Response Syndrome Caused by Exposure to the Interior Environment of Water-Damaged Buildings, [www.survivingmold.com/docs/POA\\_MOLD\\_7\\_27\\_10\\_final.pdf](http://www.survivingmold.com/docs/POA_MOLD_7_27_10_final.pdf).
- Omotayo OP, Omotayo AO, Mwanza M, Babalola OO. Prevalence of Mycotoxins and Their Consequences on Human Health. *Toxicol Res*. 2019;35(1):1–7. doi:10.5487/TR.2019.35.1.001
- Zhao J, Agarwal R. Tissue distribution of silibinin, the major active constituent of silymarin, in mice and its association with enhancement of phase II enzymes: implications in cancer chemoprevention. *Carcinogenesis*. 1999; 20:2101–2108.
- Muhialdin BJ, Saari N, Meor Hussin AS. Review on the Biological Detoxification of Mycotoxins Using Lactic Acid Bacteria to Enhance the Sustainability of Foods Supply. *Molecules*. 2020;25(11):2655. doi:10.3390/molecules25112655

5. Wu Q, Jezkova A, Yuan Z, Pavlikova L, Dohnal V, Kuca K. Biological degradation of aflatoxins. *Drug Metab Rev.* 2009;41(1):1-7. doi:10.1080/03602530802563850
6. Abbès S, Salah-Abbès JB, Sharafi H, Jebali R, Noghbi KA, Oueslati R. Ability of *Lactobacillus rhamnosus* GAF01 to remove AFM1 in vitro and to counteract AFM1 immunotoxicity in vivo. *J Immunotoxicol.* 2013;10(3):279-286. doi:10.3109/1547691X.2012.718810
7. Nurul Adilah Z, Liew WP, Mohd Redzwan S, Amin I. Effect of High Protein Diet and Probiotic *Lactobacillus casei* Shirota Supplementation in Aflatoxin B<sub>1</sub>-Induced Rats. *BioMed Res Int.* 2018;2018:9568351. doi:10.1155/2018/9568351
8. Skrinjar M, Rasić JL, Stojčić V. Lowering of ochratoxin A level in milk by yoghurt bacteria and bifidobacteria. *Folia Microbiol (Praha).* 1996;41(1):26-28. doi:10.1007/BF02816335
9. Muhialdin BJ, Saari N, Meor Hussin AS. Review on the Biological Detoxification of Mycotoxins Using Lactic Acid Bacteria to Enhance the Sustainability of Foods Supply. *Molecules.* 2020;25(11):2655. doi:10.3390/molecules25112655
10. Turbic A, Ahokas JT, Haskard CA. Selective in vitro binding of dietary mutagens, individually or in combination, by lactic acid bacteria. *Food Addit Contam.* 2002;19(2):144-152. doi:10.1080/02652030110070067
11. Lahtinen SJ, Haskard CA, Ouwehand AC, Salminen SJ, Ahokas JT. Binding of aflatoxin B<sub>1</sub> to cell wall components of *Lactobacillus rhamnosus* strain GG. *Food Addit Contam.* 2004;21(2):158-164. doi:10.1080/02652030310001639521
12. Chapot-Chartier MP, Kulakauskas S. Cell wall structure and function in lactic acid bacteria. *Microb Cell Fact.* 2014;13(Suppl 1)(suppl 1):S9. doi:10.1186/1475-2859-13-S1-S9
13. Gerez CL, Torino MI, Rollán G, de Valdez GF. Prevention of bread mould spoilage by using lactic acid bacteria with antifungal properties. *Food Control.* 2009;20(2):144-148. doi:10.1016/j.foodcont.2008.03.005
14. Niderkorn V, Boudra H, Morgavi DP. Binding of Fusarium mycotoxins by fermentative bacteria in vitro. *J Appl Microbiol.* 2006;101(4):849-856. doi:10.1111/j.1365-2672.2006.02958.x
15. El-Nezami HS, Polychronaki NN, Ma J, et al. Probiotic supplementation reduces a biomarker for increased risk of liver cancer in young men from Southern China. *Am J Clin Nutr.* 2006;83(5):1199-1203. doi:10.1093/ajcn/83.5.1199
16. Agawane SB, Lonkar PS. Effect of probiotic containing *Saccharomyces boulardii* on experimental ochratoxicosis in broilers: hematobiochemical studies. *J Vet Sci.* 2004;5(4):359-367. doi:10.4142/jvs.2004.5.4.359
17. Chang C, Wang K, Zhou SN, Wang XD, Wu JE. Protective Effect of *Saccharomyces boulardii* on Deoxynivalenol-Induced Injury of Porcine Macrophage via Attenuating p38 MAPK Signal Pathway. *Appl Biochem Biotechnol.* 2017;182(1):411-427. doi:10.1007/s12010-016-2335-x
18. Ahn JY, Kim J, Cheong DH, Hong H, Jeong JY, Kim BG. An In Vitro Study on the Efficacy of Mycotoxin Sequestering Agents for Aflatoxin B<sub>1</sub>, Deoxynivalenol, and Zearalenone. *Animals (Basel).* 2022;12(3):333. doi:10.3390/ani12030333
19. Rotter RG, Frohlich AA, Marquardt RR. Influence of dietary charcoal on ochratoxin A toxicity in Leghorn chicks. *Can J Vet Res.* 1989;53(4):449-453.
20. Bhatti SA, Khan MZ, Saleemi MK, Hassan ZU, Khan A. Ameliorative role of dietary activated carbon against ochratoxin-A induced oxidative damage, suppressed performance and toxicological effects. *Toxin Rev.* 2022;41(1):108-118. doi:10.1080/15569543.2020.1848870
21. Kihal A, Rodríguez-Prado M, Calsamiglia S. The efficacy of mycotoxin binders to control mycotoxins in feeds and the potential risk of interactions with nutrient: a review. *J Anim Sci.* 2022;100(11):skac328. doi:10.1093/jas/skac328