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Toxins (Basel). 2013 Apr; 5(4): 605–617.
Published online 2013 Apr 11. doi: 10.3390/toxins5040605

PMCID: PMC3705282
PMID: 23580077

Detection of Mycotoxins in Patients with Chronic Fatigue Syndrome

Joseph H. Brewer,[1,*] Jack D. Thrasher,[2] David C. Straus,[3] Roberta A. Madison,[4] and Dennis Hooper[5]

Toxins (Basel). 2014 Feb; 6(2): 608–623.

Published online 2014 Feb 10. doi: 10.3390/toxins6020608

PMCID: PMC3942754

PMID: 24517907

Deficient Glutathione in the Pathophysiology of Mycotoxin-Related Illness

Frederick T. Guilford[1,*] and Janette Hope[2]

Abstract

Evidence for the role of oxidative stress in the pathophysiology of mycotoxin-related illness is increasing. The glutathione antioxidant and detoxification systems play a major role in the antioxidant function of cells. Exposure to mycotoxins in humans requires the production of glutathione on an “as needed” basis. Research suggests that mycotoxins can decrease the formation of glutathione due to decreased gene expression of the enzymes needed to form glutathione. Mycotoxin-related compromise of glutathione production can result in an excess of oxidative stress that leads to tissue damage and systemic illness. The review discusses the mechanisms by which mycotoxin-related deficiency of glutathione may lead to both acute and chronic illnesses.

ScientificWorldJournal. 2013; 2013: 767482.

Published online 2013 Apr 18. doi: 10.1155/2013/767482

PMCID: PMC3654247
PMID: 23710148

A Review of the Mechanism of Injury and Treatment Approaches for Illness Resulting from Exposure to Water-Damaged Buildings, Mold, and Mycotoxins

Janette Hope*

ntranasal ketoconazole, fluconazole, and itraconazole are frequently used. However, more data is available for intranasal use of amphotericin B.

Reduced glutathione (GSH) can be administered in an intravenous, nebulized, transdermal, oral liposomal, and nasal form.

Nebulized glutathione is the only known treatment for increasing glutathione levels in the epithelial lining fluid, thought to be one of the first lines of defense for oxidative stress [119].

Since olfactory epithelium is the only place where dendritic processes are directly exposed to the environment in the cribiform plate of the ethmoid sinus, intranasal delivery of medications can bypass the blood-brain barrier. It has long been studied as a means to achieve central nervous system effects and has been studied for multiple agents in an effort to treat diseases such as depression, schizophrenia, Alzheimer's and Parkinson's disease [144]. M**edications administered intranasally have reportedly been detected in the cerebrospinal fluid (CSF) as fast as 1 minute after delivery** [144]. Intranasal administration of neuropeptides has been studied and found to have the advantage of bypassing the blood-brain barrier, which has served to limit the effectiveness of systemic therapies on central nervous system (CNS) symptoms. A study of 36 humans administered insulin, vasopressin, and melanocortin (MSH/ACTH) intranasally found that they received direct access to the CSF within 30 minutes, bypassing systemic circulation, as measured by intraspinal and intravenous catheters [145]. Levels of these neuropeptides were found in the CSF within 10 minutes and remained increased for up to 80 minutes. More prolonged sampling of a subgroup of patients receiving MSH/ACTH and vasopressin intranasally found that levels of these neuropeptides in the CSF remained above that of placebos (administered intranasal saline) at 100 to 120 minutes after administration and the authors believed that intranasal administration of neuropeptides could be useful for the treatment of brain diseases such as Alzheimer's disease and obesity.

Given the powerful antioxidant properties of reduced glutathione, clinicians have taken advantage of the transnasal delivery route and have used intranasal glutathione to successfully treat neurocognitive symptoms resulting from exposure to water-damaged buildings

Mold and Asthma, Allergies

Allergy. 2017 Jun; 72(6): 967–974.
Published online 2016 Dec 29. doi: 10.1111/all.13102

PMCID: PMC5434946
PMID: 27925656

Mold and dampness exposure and allergic outcomes from birth to adolescence: data from the BAMSE cohort

J. D. Thacher,[1] O. Gruzieva,[1] G. Pershagen,[1,2] E. Melén,[1,3] J. C. Lorentzen,[2] I. Kull,[1,3,4] and A. Bergström[1]

Conclusion

Exposure to mold or dampness during infancy increased the risk of asthma and rhinitis up to 16 years of age, particularly for nonallergic disease. Early exposure to mold or dampness appeared particularly associated with persistent asthma through adolescence.

Sci Rep. 2014; 4: 3833.
Published online 2014 Feb 10. doi: 10.1038/srep03833

PMCID: PMC3918926
PMID: 24509902

A common fungal volatile organic compound induces a nitric oxide mediated inflammatory response in *Drosophila melanogaster*

Arati A. Inamdar[a,1] and Joan W. Bennett[1]

Using a *Drosophila* model, we previously demonstrated truncated life span and neurotoxicity with exposure to 1-octen-3-ol, the volatile organic compound (VOC) responsible for much of the musty odor found in mold-contaminated indoor spaces. In this report, using biochemical and immunological assays, we show that exposure to 0.5 ppm 1-octen-3-ol induces a nitric oxide (NO) mediated inflammatory response in hemocytes, *Drosophila* innate immune cells. Moreover, exposed *Drosophila* brains show increased peroxynitrite expression. An increase in nitrite levels is observed with toluene and 1-octen-3-ol but not with 1-butanol. Pharmacological inhibitors of nitric

oxide synthase (NOS) namely, L-NAME, D-NAME and minocycline, and NOS mutants show improvements of life span among 1-octen-3-ol exposed flies. Exposure to 1-octen-3-ol also induces NOS expression in larval tracheal tissues and remodels tracheal epithelial lining. These findings suggest a possible mechanistic basis for some of the reported adverse health effects attributed to mold exposure and demonstrates the utility of this *in vivo Drosophila* model to complement existing model systems for understanding the role of inflammation in VOC-mediated toxicity.

Iran J Basic Med Sci. 2016 Apr;19(4):430-8.
Evaluation of antioxidant and cytoprotective activities of Artemisia ciniformis extracts on PC12 cells.
„…resulted in significant decrease in generation of the reactive oxygen species (ROS) and ***increase in the activity of SOD****“*
Mojarrab M

Am J Respir Crit Care Med. 1999 Dec;160(6):1943-6.

Nitric oxide and proinflammatory cytokines in nasal lavage fluid associated with symptoms and exposure to moldy building microbes.

Hirvonen MR[1], Ruotsalainen M, Roponen M, Hyvärinen A, Husman T, Kosma VM, Komulainen H, Savolainen K, Nevalainen A.
Author information
Abstract

Epidemiological data indicate that living or working in a moldy building is associated with increased risk of respiratory symptoms and disease related to inflammatory reactions, but biochemical evidence linking cause and effect is still scarce. The staff working in a mold-contaminated school, and a reference group without such exposure, were studied. Nasal lavage was performed and health data were collected with a questionnaire at the end of the spring term, after a 2.5-mo summer vacation, and at the end of the fall term. Here we show that concentrations of tumor necrosis factor alpha (TNF-alpha), interleukin-6 (IL-6), and nitric oxide (NO) in nasal lavage fluid were significantly higher in the exposed than in the control subjects at the end of the first exposure period. These inflammatory mediators decreased to reference group concentrations during the period when there was no exposure and the production of NO and IL-6 increased again during the reexposure in the fall term. Reports of cough, phlegm, rhinitis, eye irritation, and fatigue paralleled the changes in the measured inflammatory markers. These results point to an association between inflammatory markers in the nasal lavage fluid, the high prevalence of respiratory symptoms among the occupants, and chronic exposure to molds in the indoor environment.

Ann Allergy Asthma Immunol. 2006 Oct;97(4):539-45.

Mold damage in homes and wheezing in infants.

Cho SH[1], Reponen T, LeMasters G, Levin L, Huang J, Meklin T, Ryan P, Villareal M, Bernstein D.

Author information

Lung India. 2013 Oct-Dec; 30(4): 273–276.
doi: 10.4103/0970-2113.120594

PMCID: PMC3841680
PMID: 24339481

Does climate mould the influence of mold on asthma?

Ashutosh Nath Aggarwal and Arunaloke Chakrabarti[1]

Overall, relationship between climate factors and mold species, extent and geography suggests a complex multifactorial mechanism. It appears likely that climate changes can trigger more fungal spore production and alter the distribution timing and pattern. Increased environmental spore load in turn can worsen asthma control in susceptible patients. However, aero-allergen profiles differ greatly in different geographic regions. Moreover, limited availability of extensive aerobiological and epidemiological datasets over long periods presents difficulties in confirming such associations.

Front Cell Infect Microbiol. 2018; 8: 60.
Published online 2018 Feb 26. doi: 10.3389/fcimb.2018.00060

PMCID: PMC5834427
PMID: 29535978

Mycotoxin: Its Impact on Gut Health and Microbiota

Winnie-Pui-Pui Liew and Sabran Mohd-Redzwan*

Fecal: Clostridiales and Bacteroidales were increased in a dose-dependent manner of AFB1. In contrast, Lactobacillales from Firmicutes, Streptococcus sp. and Lactococcus sp. were decreased in a dose-dependent manner of AFB1 exposure.

Toxicol Sci. 2016 Mar; 150(1): 54–63.
Published online 2015 Nov 25. doi: 10.1093/toxsci/kfv259

PMCID: PMC5009611
PMID: 26612839

Aflatoxin B_1 Induced Compositional Changes in Gut Microbial Communities of Male F344 Rats

Jincheng Wang,*† Lili Tang,† Travis C. Glenn,*† and Jia-Sheng Wang*†,1

he 2 *Lactobacillales* from *Firmicutes*, *Streptococcus sp.*and *Lactococcus sp.* had the largest decrease, appearing in a dose-dependent manner.

Toxins (Basel). 2017 Jul; 9(7): 228.
Published online 2017 Jul 18. doi: 10.3390/toxins9070228

PMCID: PMC5535175
PMID: 28718805

Emerging *Fusarium* and *Alternaria* Mycotoxins: Occurrence, Toxicity and Toxicokinetics

Sophie Fraeyman,[1] S
, et al

demonstrated the growth inhibition of *Streptococcus thermophilus* and different strains of the genus *Bifidobacterium* and *Lactobacillus* by ENN A1 and ENN B1, while ENN A inhibited *Saccharomyces cerevisia*

Zentralbl Veterinarmed A. 1998 Oct;45(8):453-8.

Measurement of antibacterial activities of T-2 toxin, deoxynivalenol, ochratoxin A, aflatoxin B1 and fumonisin B1 using microtitration tray-based turbidimetric techniques.

Ali-Vehmas T[1], Rizzo A, Westermarck T, Atroshi F.

Among the tested strains, Streptococcus agalactiae was found to be sensitive to all the toxins, with the exception of OTA. T-2 toxin and FB1 were the most effective in slowing down the growth of Staphylococcus aureus. A

Zentralbl Veterinarmed A. 1998 Oct;45(8):453-8.

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Poult Sci. 1997 Sep;76(9):1205-11.

Influence of a superactivated charcoal on the toxic effects of aflatoxin or T-2 toxin in growing broilers.

Edrington TS[1], Kubena LF, Harvey RB, Rottinghaus GE.

Of the blood parameters altered by AF (decreased cholesterol, inorganic phosphorus, total protein, and urea nitrogen, and increased mean corpuscular volume and hematocrit in Experiment 1; decreased albumin and total protein, and increased creatine kinase in Experiment 2) only urea nitrogen, hematocrit, and inorganic phosphorus (Experiment 1) and hematocrit (Experiment 2) were comparable to controls when SAC was included in the diet. Feeding T-2 toxin decreased serum cholesterol, total protein, urea nitrogen, and mean corpuscular volume; however, only cholesterol and mean corpuscular volume were improved with the addition of SAC (Experiment 1)

Vet Immunol Immunopathol. 2005 Oct 18;108(1-2):199-209.

The effects of mycotoxins, fungal food contaminants, on the intestinal epithelial cell-derived innate immune response.

Bouhet S[1], Oswald IP.
Author information

Abstract

Mycotoxins are structurally diverse fungal metabolites that can contaminate a variety of dietary components consumed by animals and humans. It is considered that 25% of the world crop production is contaminated by mycotoxins. The clinical toxicological syndromes caused by ingestion of moderate to high amounts of mycotoxins and their effect on the immune system have been well characterized. However, no particular attention has been focused on the effects of mycotoxins on the local intestinal immune response. Because of their location, intestinal epithelial cells (IECs) could be exposed to high doses of mycotoxins. As a component of the innate local immune response, intestinal epithelial cells have developed a variety of mechanisms which act to reduce the risk of infection by microorganisms or intoxication by toxic compounds. This review summarises the innate immune response developed by intestinal epithelial cells and reports the literature concerning the effects of mycotoxins on them. Particularly, the effects of mycotoxins on the maintenance of a physical barrier by epithelial cells will be discussed together with their effect on extrinsic protective components of the innate intestinal immunity: mucus secretion, antimicrobial peptide generation, IgA and pro-inflammatory cytokine release.