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Editorial

Molds and mycotoxins indoors I: Current issues and way forward

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Abstract

Molds are known to cause a variety of allergies in people living or working in mold-infested buildings. Molds produce mycotoxins that can be immunotoxic, neurotoxic, pulmotoxic, nephrotoxic, hepatotoxic, developmental toxic, and carcinogenic in nature. These adverse health effects are more pronounced in immunocompromised and/or genetically predisposed individuals. Even with this understanding, numerical standards for acceptable quantities of molds in indoor environments is not supported by the regulatory agencies like U.S. EPA, FDA, and professional organizations like Institute of Inspection, Cleaning and Remediation (IICRC), American Industrial Hygiene Association (AIHA), and the American Conference of Governmental Industrial Hygienists (ACGIH). Researchers are working to establish acceptable limit of molds in indoor environments based on the genus or species and adverse health effects of their biotoxins, especially mycotoxins; however, lack of standardization of sampling and analysis results in variability across laboratories making exposure assessment and interpretation of the results challenging. Establishment of exposure limits (*e.g.,* reference dose/concentration [RfD/RfC], tolerable daily intake [TDI], slope factor) along with standard and validated methods for sampling, analysis, and reporting of molds and mycotoxins is necessary given the fact that climate change is raising both temperature and humidity resulting in increased mold growth, sporulation, and fungal diseases. This series of three papers outline the issues of molds and biotoxins, current practices, and propose improvements to standardize and validate methods of sampling, analysis, and reporting across testing laboratories.

Keywords: Mold, Mycotoxin, Molds indoor, Mycotoxins toxicity, Adverse health effects of molds, Mold exposure, Mold risk assessment, Mycotoxin exposure, Mycotoxin risk assessment

Molds and Mycotoxins

Molds grow from microscopic spores of many fungi floating in the air and when land on surfaces that are moist; none of the molds grow in the absence of moisture [1,2]. Molds are ubiquitous and found both outdoors and indoors. Outdoors, molds play a vital role in the breakdown of dead organic matters like fallen leaves and dead trees. Molds grow in humid environments outdoors; indoors, they are usually found in damp/steamy and dark areas with poor ventilation (*e.g.,* basements, bathrooms, recently flooded areas, cluttered storages, and kitchens) [1,2]. In outdoor air, levels of molds and their components (*i.e.,* hyphal fragments, spores, and mycotoxins), referred herein as *biotoxins*, remain mostly below the level of health concern, even in high humidity, due to the presence of natural light and continuous movement of air. Similarly, biotoxins are always present in indoor air entering through open windows, with foot traffic and through ventilation. The levels of biotoxins in indoor air is generally equal to or lower than found in the outdoor air [3]; however, can easily reach unsafe levels under high humidity, inadequate ventilation, and poor lighting that may lead to adverse health effects. Molds remain viable long after the source of moisture is removed resulting in biotoxins to remain in indoor air over a prolonged period of time [4].

Mycotoxins are secondary metabolites produced by many molds and capable of producing various adverse health effects on many organ systems, especially in sensitive individuals (*e.g.,* immunocompromised and/or genetically predisposed to slow elimination, like HLA gene alleles); exposure to mycotoxins have even been linked to infants' death [1,5-9]. In general, exposure to biotoxins to individuals living or working in mold-infested buildings can cause a variety of allergies (*e.g.,* hay fever-type symptoms [sneezing, runny nose, red eyes], inflammation, and skin rash) [2,10-20]. Exposure to mycotoxins can cause immunotoxicity, neurotoxicity (*e.g.,* increased anxiety, depression, and cognitive deficits), pulmotoxicity, nephrotoxicity, hepatotoxicity, and even cancer when exposed to elevated levels for prolonged periods of time [1,7-9,21].

Secondary to the exposure to mycotoxins are changes in gut microbiota (dysbiosis) with possible disruption in gut barrier function and translocation of bacterial toxins. Gut dysbiosis increases bacterial lipopolysaccharide (LPS) transport into blood which also increases bacteriotoxin and mycotoxin absorption and transport from intestine to systemic circulation and can modulate their toxicity [21-24]. LPS can also cause inflammation and increases blood brain barrier permeability; increased blood LPS levels have been observed in neurodegenerative diseases, such as Alzheimer's and Parkinsons's diseases [22,24]. Consequently, increased indoor bacterial growth may be of major health concern for higher bacteriotoxin and mycotoxin exposure. Therefore, closer examination of the relationship among gut dysbiosis, LPS, and higher mycotoxins absorption/exposure contributing to mycotoxin-related toxicities is warranted.

Prevalence of Mold-Infestation to Buildings

Mold-affected buildings are still a persistent and common global problem even with improved regulations and building codes [9,16,25-27]. Globally, up to 47% of the homes are reported to have dampness and molds, both in affluent and non-affluent countries [28]. Moisture and molds are found indoors in up to 27% European, 47% American and New Zealand, and 12% Chinese homes [29-40]. Problems of moisture and molds are higher in structures built of timber, drywalls and filled with insulation made of cellulose, foam, mineral wool, plastic and natural fibers, fiberglass, etc. Additionally, constructing weathertight (e.g., energy efficient) buildings to protect from elements like cold, heat, and wind exacerbate moisture indoors when compared with the structures built of concrete. Inside energy efficient buildings, humidity and moisture can build quickly favoring mold growth. This happens commonly, especially in residential buildings, due to one or combination of the reasons like poor maintenance, less than optimal air exchange from malfunctioning HVAC (heating, ventilation, and air conditioning) system or dirty air filters, blocking air exchange vents from cluttering; all leading to high moisture indoors favoring mold growth.

Factors Impacting Indoor Mold Growth and Rise in Infections and Diseases

Growth of molds indoors may result from either a single incident like weather events and flooding, or combined effects of interrelated factors, like overcrowding, poor conditions, socioeconomic situations, globalization, urbanization, and climate change [9,16,41-45]. There is growing evidence of the relationship between climate change and increased fungal growth, sporulation, and allergies [46,47]. This relationship is likely from water and moisture intrusion into buildings from extreme weather events such as cyclones, hailstorms, storm surge, flooding, and temperature extremes, especially in climate-sensitive countries, flood-sensitive locations, and coastal communities [48-50]. A rise in fungal infections in humans [51] is observed with the rise in global temperature, humidity, and population growth **(Figure 1)**.

Consequently, the number of buildings with biotoxin infestations are on the rise along with many of the associated adverse health effects among the inhabitants. The majority of the cases are from socioeconomically deprived neighborhoods like public housing.

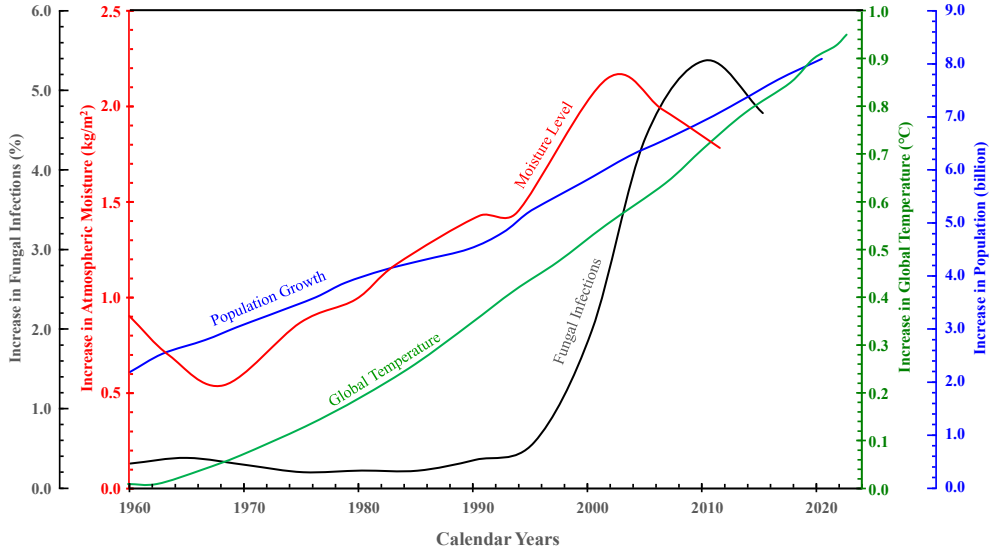

Figure 1. Rise in fungal infections in humans with the rise in temperature, humidity, and population growth. Data were obtained by digitizing graphs using DigitizeIt (version 2.1.2) digitizing software, from NASA 2023 (rise in global temperature) [52], Willett et al. 2023 (rise in global moisture) [53], Zandalinas et al. 2021 (increase in population) [54], and Dantas et al. 2021 (rise in fungal infection in humans) [51]. This is a modification of a Figure from Saghir and Ansari 2024 [55].

People living in such neighborhoods are already associated with health-related quality of life (HRQoL) affecting physical health [56] and vulnerable to any perturbation in their living environment which will likely result in much severe response to exposure to biotoxins. Reasons of high mold growth in socioeconomically deprived neighborhoods include negligence in building maintenance, each unit is usually multi-habited and poorly maintained as owners often conduct reactive maintenance waiting until the components are completely deteriorated before repairing or replacing.

Even though maintenance of housing is strongly correlated with the income of the households, a significant number of biotoxins infested buildings are also from affluent neighborhoods, mostly rental properties. Mold infestation in rental buildings from affluent neighborhoods is mostly due to negligence of landlords and/or management as they are responsible for keeping structures fit and habitable by timely and proper repairs. When leaking pipes, windows, and roofs are not fixed in timely fashion or properly, moisture may buildup in areas not directly visible, such as, inside drywalls or insulations resulting in mold growth.

Collection of Samples from Inside the Buildings

To determine whether the level of molds inside a building is higher than the background (*i.e.,* outside) due to the infestation of mold from water ingress, condensation, or dampness, one needs to identify source(s) and extent of moisture and determine presence, location, and extent of mold growth. This usually begins with a suspicion of mold due to complaints from the inhabitant(s) of an odor, moisture damage, excess humidity, damaged waterpipes/water leakage, flood, presence of molds, and/or health effects described above. At this stage, the complaint is investigated by gathering information of the incident followed by visual inspection of the premises to assess the severity of the problem. After this, additional investigations are planned, if required, to determine the presence or absence of elevated mold levels indoors by additional mold sampling, and if necessary, investigating suspected hidden areas where molds may be growing. This additional investigation is likely performed by qualified environmental specialist/consultant. In addition to determining presence of biotoxins, sometimes, samples are also taken from the area(s) of concern to determine presence and the number of colony-forming units (CFUs) of bacteria and levels of endotoxins produced.

Currently, indoor mold sampling methods are neither guided by any regulation nor well validated or standardized. There is no standardized collection protocol, and a wide variety of collection methods are used to collect molds from air, surfaces, and settled dust. In most cases, methods used by the commercial entities are proprietary and often guarded as *"trade secret"*; not outlined in the test results/reports, published on laboratories' websites, peer-reviewed scientific publications, or commercially available. However, some of the common characteristics of sampling strategies depend on the size and nature of the building, available resources, availability of methods, and information needed. All mold sampling strategies are systematic in nature and generally follow recommended decision tree outlined in **(Figure 2)**. It is important to note that such sampling only captures a small window of time, and a large temporospatial variability exists in the levels and number of species of molds inside buildings.

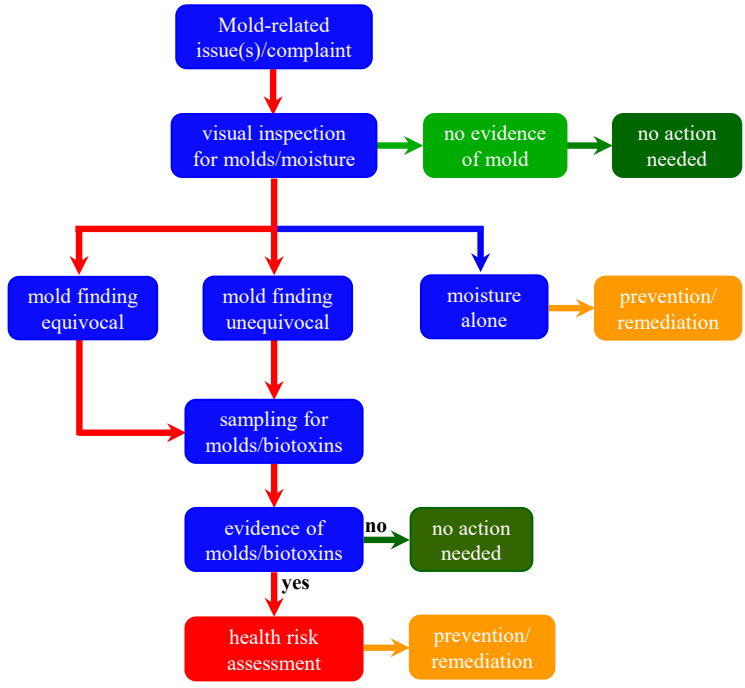

Figure 2. Decision flow chart for inspection of buildings with suspected mold infestation and action(s) when buildings are infested with molds.

Collection of Biological Samples from Inhabitant(s)

Urine, and sometimes blood, of inhabitant(s) are collected and analyzed to determine mycotoxins. Fecal samples are also often collected from inhabitant(s) and analyzed for commensal and dysbiotic bacteria, normal and dysbiotic molds, parasites, and several nutritional biomarkers. Collected samples are usually sent to mold testing laboratories for analysis, they utilize a variety of methods due to the lack of guidelines, regulations, and/or validated/standardized analytical methods.

Determination of Molds and Their Components in Collected Samples

Like sampling, there are neither regulatory guidelines nor well validated/standardized protocols/methods to determine and analyze molds, biotoxins, bacteria, parasites, and nutritional biomarkers in the samples collected from mold-infested buildings and their inhabitant(s). Therefore, a wide variety of methods are used by mold testing laboratories. Methods used by the commercial entities are usually proprietary in nature and often guarded as *"trade secret"* and publicly not available.

Samples collected from inside the buildings (air, surface and settled dust, and/or swab) are sent to mold testing laboratories for the analysis of biotoxins. These samples are analyzed for molds by either culture-based assay, non-culture-based analyses (*i.e.,* microscopy and spore counting), or by molecular analysis (e.g., quantitative polymerase chain reaction [qPCR]); combination of methods are also used when samples are collected using specific procedures [57]. The presence and abundance of spores and hyphae in collected samples is mostly determined by counting them microscopically. The presence of bacteria in samples collected from damp, wet, and/or areas with water, is determined by a culture-based assay to determine the number of CFUs. Before deciding which analytical method to use, specific strengths and weaknesses of each method are evaluated.

Biological samples (*i.e.,* urine, blood and/or feces) are also sent to mold testing laboratories for the analysis of mycotoxins, bacteria, parasites, and nutritional biomarkers. Blood when collected, and urine are analyzed for a list of mycotoxins using **liquid chromatography/mass spectrometry** (LC-MS/MS), enzyme linked immunosorbent assays (ELISA), or some other undisclosed analytical methods. There are, however, detailed analytical procedures available in the literature that can be standardized for use by the laboratories [58-62]. Fecal samples are analyzed for the presence of normal, commensal, and dysbiotic bacteria and yeast by culture-based assays; abundance of yeast is determined by microscopic counting. The presence and abundance of parasites and nutritional biomarkers in feces are also determined by commonly accepted methods.

Exposure Assessment

Exposure of inhabitant(s) to molds from mold-infested buildings and their potential toxic secondary metabolite(s), *i.e.,* mycotoxins is ascertained by determining molds and mycotoxins in biological samples. Currently there are no U.S. EPA or other federal regulations or standards for airborne molds and biotoxins; therefore, results of the sampling cannot be used to determine compliance of buildings with any regulatory standards. Similarly, there is no health-based standard for exposure to molds and mycotoxins (*e.g.,* reference dose (RfD) or tolerable daily intake (TDI)) indoors; therefore, levels detected in biological samples cannot be used for risk assessment in a straightforward manner without further evaluation utilizing publicly available (mostly peer-reviewed scientific papers) animal and human data. Even though health impacts of exposure to mycotoxins inside residential buildings are not well studied, adverse health effects in occupational settings and in animal studies have been reported [57]. Health impacts from mycotoxin exposure vary depending on the type of mycotoxin, nature of exposure (*i.e.,* acute versus chronic, duration, frequency), its kinetics and dose-response relationship. An interim approach for the risk assessment of mycotoxins from indoor environments can use the risk values set for ingestion (*e.g.,* maximum levels for aflatoxins in various nuts, grains, dried figs, and milk is 0.5-15 µg/kg and for patulin in apple juice is 50 µg/kg [9]) following considerations for the differences between oral and inhalation routes of exposure, absorption, distribution, metabolism, excretion (ADME), and kinetics.

Impact of the Lack of Harmonization of Sampling and Analytical Methods

One of the issues that scientists working on indoor molds and exposure to inhabitant(s) continuously encounter is the lack of standardization of methods of sampling (*e.g.*, air, dust, water, breathing zone air, urine, blood), storage, transport, extraction, and analysis. Each laboratory has its own *"trade secret"* methods which cannot be found either in peer-reviewed scientific papers or on their websites. This is very frustrating as the lack of standardization/harmonization makes scientists difficult to follow data from different laboratories when different and especially *"trade secret"* methods of analysis are used, raising doubts of the findings.

Conclusions

As mentioned earlier, variety of allergies (*e.g.,* skin rashes, flue and hay fever-type symptoms [sneezing, runny nose, red eyes], inflammation, and respiratory and eye irritation) are known to be caused by biotoxins (*i.e.,* hyphal fragments, spores, and mycotoxins) in people living or working in mold-infested buildings [2,10-20]. Additionally, exposure to mycotoxins can cause fatigue, nausea, immunotoxicity, neurotoxicity (*e.g.,* dizziness, increased anxiety, depression, and cognitive deficits), pulmotoxicity, nephrotoxicity, hepatotoxicity, birth defects, and cancer usually following high levels of exposure for prolong periods of time [1,7-9,21]. Exposure to mycotoxins can also make individuals vulnerable to microbial diseases [21]. These adverse health effects may even be more pronounced in sensitive (*e.g.,* immunocompromised, and/or genetically predisposed to slow elimination, like HLA gene alleles) individuals [1,5,7-9,55].

Even with the understanding of possible adverse health effects from exposure to molds and biotoxins, with the current understanding, numerical standards for acceptable quantities of molds in indoor environments cannot be scientifically justified. Consequently, most professional organizations like IICRC, AIHA, and ACGIH do not support numerical limits for molds in indoor environments.

There have been attempts by researchers to set up acceptable limits of molds in indoor environments based on the genus or species and adverse health effects of their biotoxins, especially mycotoxins. However, variability in mold levels from the use of different methods of measurement, made this approach challenging as results may not be comparable among mold testing laboratories. As an alternative, comparing indoor and outdoor airborne mold spores has been

suggested as a more meaningful [63,64]; as in principle, amounts of molds indoors are less and of similar types than those found outdoors [3].

Assessing exposure to biotoxins, and bacteria/endotoxins is even more challenging as approaches currently used (*e.g.,* airborne spores in breathing zone or internalized molds in feces; mycotoxins in urine and/or blood) provide only a snapshot of mold exposure without considering the long-term exposure or exposure from different environments. While results of these tests can confirm exposure, they do not provide information about when or where the exposure has occurred. To establish the source of exposure, indoor mold exposure pathways are necessary showing exposure to molds that produce biotoxins found in the exposed individual(s). Source(s) of exposure is further established by determining the amount of time inhabitant(s) spend at the building in question and at other building(s) (*e.g.,* at work) and whether other building(s) is/are mold infested. To establish timeline and the extent of exposure to mycotoxins, kinetic information is used along with any preexisting condition(s) that may influence ADME of mycotoxins in question. Additionally, other factors are also incorporated when calculating timeline of exposure since certain genetic condition(s) like genetic predisposition at the HLA/DR gene is known to slow elimination of mycotoxins. For example, elimination of ochratoxin A and mycophenolic acid has been reported to be ~10- and ~213-fold slower, respectively, in individuals with the HLA/DR gene than those without it [55].

One of the biggest challenges in mold sampling and exposure assessment is interpretation of the results due to a lack of standardization and therefore, results can be highly variable, ambiguous, and difficult to interpret that may lead to inconsistent conclusions. These issues can easily be addressed by standardizing and validating methods, instrumentations, sampling techniques, collecting replicates (*e.g.,* $n \geq 3$), analytical procedures, and reporting of the results. These requirements should be mandated to all mold testing laboratories through regulations or by binding recommendations from a taskforce as many points of controversy arise from a lack of consistency in collection and analysis of molds, biotoxins, bacteria, and endotoxins.

We now have evidence that climate change is raising both earth temperature and humidity resulting in increased mold growth, sporulation, and fungal diseases **(Figure 1)**. Consequently, rise in mold-infested buildings is expected, which in turn will increase the number of patients with severe mold- and biotoxin-related diseases needing better health management. Henceforth, mandating mold testing laboratories to standardize and validate methods for sampling, analysis, and reporting is required. This will help in consistency of results across the testing laboratories avoiding current controversies around results from different laboratories. Additionally, more animal studies are needed to develop inhalation exposure limits (*e.g.,* RfC, TDI, slope factor) for molds and biotoxins, especially mycotoxins, responsible for, at least those causing severe adverse health effects. As data are being generated, an interim approach of relative abundance of molds indoors versus outdoors, NOAEL of certain mold spores and mycotoxins in animals [65], allowable levels of certain mycotoxins in food and/or drinks for ingestion [9], and other available information can be used after correction for the differences among the routes of exposure, ADME/kinetics.

References

1. Empting LD. Neurologic and neuropsychiatric syndrome features of mold and mycotoxin exposure. Toxicol Ind Health. 2009 Oct-Nov;25(9-10):577-81.
2. U.S. EPA (Environmental Protection Agency). A Brief Guide to Mold, Moisture and Your Home. 2023. https://www.epa.gov/mold/brief-guide-mold-moisture-and-your-home (accessed November 18, 2024).
3. Palaty C. Mould Assessment in Indoor Environments-Review of Guidelines and Evidence. Prepared by the Metaphase Health Research Consulting Inc, Canada, for National Collaborating Centre for Environmental Health. Canada 2010 May 14. https://ncceh.ca/sites/default/files/Mould_Assessment_May_2010.pdf (accessed November 18, 2024).
4. Straus DC. Molds, mycotoxins, and sick building syndrome. Toxicol Ind Health. 2009 Oct-Nov;25(9-10):617-35.
5. Croft WA, Jarvis BB, Yatawara CS. Airborne outbreak of trichothecene toxicosis. Atmos Environ (1967). 1986 Jan 1;20(3):549-52.
6. Dearborn DG, Yike I, Sorenson WG, Miller MJ, Etzel RA. Overview of investigations into pulmonary hemorrhage among infants in Cleveland, Ohio. Environ Health Perspect. 1999 Jun;107 Suppl 3(Suppl 3):495-9.
7. Köhler JR, Casadevall A, Perfect J. The spectrum of fungi that infects humans. Cold Spring Harb Perspect Med. 2014 Nov 3;5(1):a019273.
8. Harding CF, Pytte CL, Page KG, Ryberg KJ, Normand E, Remigio GJ, et al. Mold inhalation causes innate immune activation, neural, cognitive and emotional dysfunction. Brain Behav Immun. 2020 Jul;87:218-28.
9. WHO (World Health Organization). Mycotoxins. WHO Publications. Geneva: WHO; 2023. https://www.who.int/news-room/fact-sheets/detail/mycotoxins (accessed November 18, 2024).
10. Creasia DA, Thurman JD, Wannemacher RW Jr, Bunner DL. Acute inhalation toxicity of T-2 mycotoxin in the rat and guinea pig. Fundam Appl Toxicol. 1990 Jan;14(1):54-9.
11. Johanning E, Landsbergis P, Gareis M, Yang CS, Olmsted E. Clinical experience and results of a Sentinel Health Investigation related to indoor fungal exposure. Environ Health Perspect. 1999 Jun;107 Suppl 3(Suppl 3):489-94.
12. Robbins CA, Swenson LJ, Nealley ML, Gots RE, Kelman BJ. Health effects of mycotoxins in indoor air: a critical review. Appl Occup Environ Hyg. 2000 Oct;15(10):773-84.
13. Górny RL. Filamentous microorganisms and their fragments in indoor air--a review. Ann Agric Environ Med. 2004;11(2):185-97.
14. Górny RL, Reponen T, Willeke K, Schmechel D, Robine E, Boissier M, et al. Fungal fragments as indoor air biocontaminants. Appl Environ Microbiol. 2002 Jul;68(7):3522-31.
15. Hooper DG, Bolton VE, Guilford FT, Straus DC. Mycotoxin detection in human samples from patients exposed to environmental molds. Int J Mol Sci. 2009 Apr 1;10(4):1465-75.
16. WHO (World Health Organization). WHO Guidelines for Indoor Air Quality: Dampness and Mould. Geneva: WHO; 2009.
17. Brewer JH, Thrasher JD, Straus DC, Madison RA, Hooper D. Detection of mycotoxins in patients with chronic fatigue syndrome. Toxins (Basel). 2013 Apr 11;5(4):605-17.

18. Baxi SN, Portnoy JM, Larenas-Linnemann D, Phipatanakul W; Environmental Allergens Workgroup. Exposure and Health Effects of Fungi on Humans. J Allergy Clin Immunol Pract. 2016 May-Jun;4(3):396-404.

19. Thrasher JD, Prokop C, Roberts C, Hooper D. A family with ME/CFS following exposure to molds, mycotoxins and bacteria in a water-damaged home: a case report. Int J Clin Toxicol. 2016;4:15.

20. Jakšić D, Sertić M, Kifer D, Kocsubè S, Mornar Turk A, Nigović B, et al. Fungi and their secondary metabolites in water-damaged indoors after a major flood event in eastern Croatia. Indoor Air. 2021 May;31(3):730-44.

21. Bennett JW, Klich M. Mycotoxins. Clin Microbiol Rev. 2003 Jul;16(3):497-516.

22. Martins IJ. Overnutrition Determines LPS Regulation of Mycotoxin Induced Neurotoxicity in Neurodegenerative Diseases. Int J Mol Sci. 2015 Dec 10;16(12):29554-73.

23. Guerre P. Mycotoxin and Gut Microbiota Interactions. Toxins (Basel). 2020 Dec 4;12(12):769.

24. Sharma A, Martins IJ. The role of Microbiota in the Pathogenesis of Alzheimer's Disease. Acta Scientific Nutritional Health. 2023 Jul;7(7):108-18.

25. Dewsbury M, Law T, Potgieter J, FitzGerald D, McComish B, Chandler T, et al. Scoping study of condensation in residential buildings. Australian Building Codes Board. Available from: https://www.abcb.gov.au/sites/default/files/resources/2020//Scoping_Study_of_Condensation_in_Residential_Buildings_Appendices.pdf (accessed November 18, 2024).

26. Major JL, Boese GW. Cross Section of Legislative Approaches to Reducing Indoor Dampness and Mold. J Public Health Manag Pract. 2017 Jul/Aug;23(4):388-95.

27. Telfar-Barnard L, Bennett J, Howden-Chapman P, Jacobs DE, Ormandy D, Cutler-Welsh M, et al. Measuring the Effect of Housing Quality Interventions: The Case of the New Zealand "Rental Warrant of Fitness". Int J Environ Res Public Health. 2017 Nov 7;14(11):1352.

28. Weinmayr G, Gehring U, Genuneit J, Büchele G, Kleiner A, Siebers R, et al. Dampness and moulds in relation to respiratory and allergic symptoms in children: results from Phase Two of the International Study of Asthma and Allergies in Childhood (ISAAC Phase Two). Clin Exp Allergy. 2013 Jul;43(7):762-74.

29. Haverinen-Shaughnessy U. Prevalence of dampness and mold in European housing stock. J Expo Sci Environ Epidemiol. 2012 Sep;22(5):461-7.

30. Norbäck D, Zock JP, Plana E, Heinrich J, Tischer C, Jacobsen Bertelsen R, et al. Building dampness and mold in European homes in relation to climate, building characteristics and socio-economic status: The European Community Respiratory Health Survey ECRHS II. Indoor Air. 2017 Sep;27(5):921-32.

31. Gunnbjörnsdóttir MI, Franklin KA, Norbäck D, Björnsson E, Gislason D, Lindberg E, et al. Prevalence and incidence of respiratory symptoms in relation to indoor dampness: the RHINE study. Thorax. 2006 Mar;61(3):221-5.

32. Bornehag CG, Sundell J, Sigsgaard T. Dampness in buildings and health (DBH): Report from an ongoing epidemiological investigation on the association between indoor environmental factors and health effects among children in Sweden. Indoor Air. 2004;14 Suppl 7:59-66.

33. Sun Y, Sundell J. On associations between housing characteristics, dampness and asthma and allergies among children in Northeast Texas. Indoor Built Environ. 2013 Aug;22(4):678-84.

34. LBNL (Lawrence Berkeley National Laboratory). Prevalence of Building Dampness. 2024. https://iaqscience.lbl.gov/prevalence-building-dampness (accessed November 18, 2024).

35. Ingham T, Keall M, Jones B, Aldridge DRT, Dowell AC, Davies C, et al. Damp mouldy housing and early childhood hospital admissions for acute respiratory infection: a case control study. Thorax. 2019 Sep;74(9):849-57.

36. Howden-Chapman P, Matheson A, Crane J, Viggers H, Cunningham M, Blakely T, et al. Effect of insulating existing houses on health inequality: cluster randomised study in the community. BMJ. 2007 Mar 3;334(7591):460.

37. New Zealand Government. One in Five Homes Damp. 2019. https://www.stats.govt.nz/news/one-in-five-homes-damp (accessed November 18, 2024).

38. Cai J, Liu W, Hu Y, Zou Z, Shen L, Huang C. Associations between home dampness-related exposures and childhood eczema among 13,335 preschool children in Shanghai, China: A cross-sectional study. Environ Res. 2016 Apr;146:18-26.

39. Liu W, Huang C, Hu Y, Zou Z, Shen L, Sundell J. Associations of building characteristics and lifestyle behaviors with home dampness-related exposures in Shanghai dwellings. Build Environ. 2015 Jun 1;88:106-15.

40. Cai J, Li B, Yu W, Wang H, Du C, Zhang Y, et al. Household dampness-related exposures in relation to childhood asthma and rhinitis in China: A multicentre observational study. Environ Int. 2019 May;126:735-46.

41. Baker E, Lester L, Beer A, Bentley R. An Australian geography of unhealthy housing. Geogr Res. 2019 Feb;57(1):40-51.

42. Sharpe RA, Taylor T, Fleming LE, Morrissey K, Morris G, Wigglesworth R. Making the Case for "Whole System" Approaches: Integrating Public Health and Housing. Int J Environ Res Public Health. 2018 Oct 24;15(11):2345.

43. Keall M, Baker MG, Howden-Chapman P, Cunningham M, Ormandy D. Assessing housing quality and its impact on health, safety and sustainability. J Epidemiol Community Health. 2010 Sep;64(9):765-71.

44. Fisk WJ. Review of some effects of climate change on indoor environmental quality and health and associated no-regrets mitigation measures. Build Environ. 2015 Apr 1;86:70-80.

45. Antova T, Pattenden S, Brunekreef B, Heinrich J, Rudnai P, Forastiere F, et al. Exposure to indoor mould and children's respiratory health in the PATY study. J Epidemiol Community Health. 2008 Aug;62(8):708-14.

46. Beggs PJ, Bambrick HJ. Is the global rise of asthma an early impact of anthropogenic climate change? Environ Health Perspect. 2005 Aug;113(8):915-9.

47. Shea KM, Truckner RT, Weber RW, Peden DB. Climate change and allergic disease. J Allergy Clin Immunol. 2008 Sep;122(3):443-53; quiz 454-5.

48. Hurlimann AC, Warren-Myers G, Browne GR. Is the Australian construction industry prepared for climate change?. Build Environ. 2019 Apr 15;153:128-37.

49. Lane K, Charles-Guzman K, Wheeler K, Abid Z, Graber N, Matte T. Health effects of coastal storms and flooding in urban areas: a review and vulnerability assessment. J Environ Public Health. 2013;2013:913064.

50. Vardoulakis S, Dimitroulopoulou C, Thornes J, Lai KM, Taylor J, Myers I, et al. Impact of climate change on the domestic indoor environment and associated health risks in the UK. Environ Int. 2015 Dec;85:299-313.

51. Dantas KC, Mauad T, de André CDS, Bierrenbach AL, Saldiva PHN. A single-centre, retrospective study of the incidence of invasive fungal infections during 85 years of autopsy service in Brazil. Sci Rep. 2021 Feb 17;11(1):3943.

52. NASA (National Aeronautics and Space Administration). Global Climate Change. Vital Signs of the Planet–Global Temperature, Latest Annual Average Anomaly: 2023. 2023. https://climate.nasa.gov/vital-signs/global-temperature/ (accessed November 18, 2024).

53. Willett KM, Dunn RJ, Thorne PW, Bell S, de Podesta M, Parker DE, et al. HadISDH. land: Gridded global monthly land surface humidity data version 4. 5. 1.2022 f. NERC EDS Centre for Environmental Data Analysis. 13 June 2023. https://catalogue.ceda.ac.uk/uuid/8956cf9e31334914ab4991796f0f645a (accessed November 18, 2024).

54. Zandalinas SI, Fritschi FB, Mittler R. Global Warming, Climate Change, and Environmental Pollution: Recipe for a Multifactorial Stress Combination Disaster. Trends Plant Sci. 2021 Jun;26(6):588-99.

55. Saghir SA, Ansari RA. HLA gene variations and mycotoxin toxicity: Four case reports. Mycotoxin Res. 2024 Feb;40(1):159-173.

56. Rocha V, Ribeiro AI, Severo M, Barros H, Fraga S. Neighbourhood socioeconomic deprivation and health-related quality of life: A multilevel analysis. PLoS One. 2017 Dec 13;12(12):e0188736.

57. Prezant B, Weekes DM, Miller JD. Recognition, evaluation, and control of indoor mold. Fairfax, Virginia: American Industrial Hygiene Association; 2008.

58. Reiter E, Zentek J, Razzazi E. Review on sample preparation strategies and methods used for the analysis of aflatoxins in food and feed. Mol Nutr Food Res. 2009 Apr;53(4):508-24.

59. Turner NW, Bramhmbhatt H, Szabo-Vezse M, Poma A, Coker R, Piletsky SA. Analytical methods for determination of mycotoxins: An update (2009-2014). Anal Chim Acta. 2015 Dec 11;901:12-33.

60. Xie L, Chen M, Ying Y. Development of Methods for Determination of Aflatoxins. Crit Rev Food Sci Nutr. 2016 Dec 9;56(16):2642-64.

61. Cao X, Li X, Li J, Niu Y, Shi L, Fang Z, et al. Quantitative determination of carcinogenic mycotoxins in human and animal biological matrices and animal-derived foods using multi-mycotoxin and analyte-specific high performance liquid chromatography-tandem mass spectrometric methods. J Chromatogr B Analyt Technol Biomed Life Sci. 2018 Jan 15;1073:191-200.

62. Lindemann V, Schleiner T, Maier U, Fels H, Cramer B, Humpf HU. Analysis of mold and mycotoxins in naturally infested indoor building materials. Mycotoxin Res. 2022 Aug;38(3):205-20.

63. Meklin T, Reponen T, McKinstry C, Cho SH, Grinshpun SA, Nevalainen A, et al. Comparison of mold concentrations quantified by MSQPCR in indoor and outdoor air sampled simultaneously. Sci Total Environ. 2007 Aug 15;382(1):130-4.

64. Codina R, Fox RW, Lockey RF, DeMarco P, Bagg A. Typical levels of airborne fungal spores in houses without obvious moisture problems during a rainy season in Florida, USA. J Investig Allergol Clin Immunol. 2008;18(3):156-62.

65. Flemming J, Hudson B, Rand TG. Comparison of inflammatory and cytotoxic lung responses in mice after intratracheal exposure to spores of two different Stachybotrys chartarum strains. Toxicol Sci. 2004;78:267-75.