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# A Review of Inhalation Exposure to Mold and Adverse Health Outcomes

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# A REVIEW OF INHALATION EXPOSURE TO MOLD AND ADVERSE HEALTH OUTCOMES

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## I. INTRODUCTION

The discovery that some molds are capable of producing toxic metabolites, known as "mycotoxins", has unearthed the notion of adverse health outcomes via inhalation exposure to mold. Molds have been implicated as the causative agents for a wide array of health problems, ranging from headaches and memory loss to pulmonary hemorrhage and hepatic failure.[1] Before the lucrative nature of mold litigation was so widespread, the primary concern for mold exposure was ingestion of mold-contaminated foodstuffs. The

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1. S. G. Revankar, *Clinical Implications of Mycotoxins and Stachybotrys*, 325 AM. J. MED. SCI. 262, 262-74 (2003).

perception of mold as an indoor hazard has since expanded to incorporate the potential of developing illnesses from residing or working in buildings with measurable levels of mold.[2]

The recent propagation of mold litigation tends to stem from the limited understanding of the human health hazards from residing or working in mold "contaminated" buildings (i.e. a building with measurable levels of mold), the mass media coverage of "toxic mold", and insurer "bad faith".[3] A slow but steady rise in the number of water damage claims has been observed from 1987 to 1998. However, in 1999 the number of claims skyrocketed to over 2500 claims, a 53-fold increase from the previous year. To date, this surge in claims has not yet subsided. Florida now ranks among the top three States in the U.S. with the most first party insurance mold claims. Based on the latest published tally, over 3,900 claims have been filed in Florida alone, a number that is 150 times that filed in Wisconsin, a state of similar size, and 3.8 times that filed in New York, a state with similar population size (Figure 1).[4] Discrepancies in mold claims across the U.S. may be due to the high variability of mold levels based on seasonal and geographical differences[5] as well as sampling techniques.[6]

An example of the limited understanding for the levels of mold and mycotoxins that may cause adverse health problems is exhibited in one of the most notable legal suits in Florida: *Martin County v. Centex-Rooney Construction Co., Inc.* In this case, building occupants complained of headaches, fatigue, and chest tightness a few weeks after moving into the new Martin County courthouse and office building. Though double the number of indoor air samples was taken, the average concentration of fungi was half of the concentration obtained in outdoor air samples.[7] Despite this normal ratio between indoor/outdoor levels of mold, the detection of mold growth in parts of the building was pegged as the causative agent. After the trial, Centex-Rooney and the other companies were

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2. Y. Assouline-Dayan et al., *Studies of Sick Building Syndrome. IV. Mycotoxicosis*, 39 J. ASTHMA 191, 191-201 (2002).

3. D. J. Graziano & M. M. Collins, *Proliferating Mold Litigation: Why Mold Is Not the Next "Asbestos"*, LXXVII FLA. BAR J. 72, 72 (2003).

4. *Breakdown of Mold-Related Insurance Claims*, 2 COLUMNS-MOLD 57, 57 (2003), *available at* http://www.harrismartin.com/tour/pdfs/beyondtheheadlines.pdf.

5. National Allergy Bureau, *NAB: Pollen & Mold Counts*, (2002), http://www.aaaai.org/nab/pollen.stm; B. G. Shelton et al., *Profiles of Airborne Fungi in Buildings and Outdoor Environments in the United States*, 68 APPL. ENVIRON. MICROBIOL. 1743, 1743-53 (2002).

6. R. E. Gots et al., *Indoor Health: Background Levels of Fungi*, 64 AIHA J. (FAIRFAX, VA) 427, 427-38 (2003).

7. M. J. Hodgson et al., *Building-Associated Pulmonary Disease from Exposure to Stachybotrys Chartarum and Aspergillus Versicolor*, 40 J. OCCUP. ENVIRON. MED. 241, 241-49 (1998).

asked to pay over $14.2 million, which included $8.8 million in damages.[8] A win made possible by the "general acceptance", under the Frye Standard, that mold and mycotoxins are harmful. The legal and medical literature is replete with cases and studies that only contribute additional uncertainty to the existence of an association between inhalation mold exposure and adverse health outcomes. This paper provides a review of the major scientific studies on this issue to date.

## II. HISTORICAL OVERVIEW OF MOLD EXPOSURE AND ADVERSE HEALTH OUTCOMES

Mold-related illnesses due to ingestion of contaminated foodstuffs date as far back as the Middle Ages where the development of acute peripheral ischemia or "St. Anthony's Fire" was believed to be caused by consumption of moldy rye contaminated with ergot alkaloids.[9] Between 1942 and 1948, a strange illness claimed the lives of at least 100,000 people in the Soviet Union. The illness, alimentary toxic aleukia, was subsequently attributed to the consumption of bread made from wheat contaminated with trichothecene mycotoxins produced by *Fusarium sporotrichioides* and *Fusarium poae*. A more recent tragedy was the outbreak of gastrointestinal disorder in India in the summer of 1987. Thousands of residents of Srinagar and the surrounding areas in the Kashmir Valley became ill after consuming rain-damaged wheat. Both *Aspergillus* and *Fusarium* species were isolated from the wheat samples.[10]

Mold-related illness from ingestion of contaminated foodstuffs is not limited to humans; livestock have routinely suffered the ill effects of mold-contaminated feed, with one of the largest poisonings occurring in a western part of the former Soviet Union (Ukraine) during the 1930s. During this time, tens of thousands of horses suffered from severe inflammation of the skin and respiratory tract, hemorrhaging, tissue necrosis, hematological disorders, and neurological disorders. Scientists later identified that the horses were consuming fodder contaminated with *Stachybotrys chartarum*.[11] Moreover, in the early 1960's, a group of mycotoxins

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8. Centex-Rooney Constr. Co., Inc. v. Martin County, Fla., 706 So. 2d 20, (Fla. 4th DCA, 1997).

9. S. Christopoulos et al., *Saint-Anthony's Fire*, 358 LANCET 1694, 1694 (2001); R. A. Etzel, *Mycotoxins*, 287 JAMA 425, 425-27 (2002).

10. R. V. Bhat et al., *Outbreak of Trichothecene Mycotoxicosis Associated with Consumption of Mould-Damaged Wheat Production in Kashmir Valley, India*, 1 LANCET 35, 35-37 (1989).

11. M. A. Akkmeteli, *Epidemiological Features of the Mycotoxicoses*, 31 ANN. NUTR. ALIMENT. 957, 957-75 (1977).

classified as aflatoxins were identified as the causative agents of the infamous "Turkey X" disease.[12] This disease was responsible for the death of over 100,000 fowl in England that had consumed feed contaminated with *Aspergillus* species.

Finally, the discovery of the class of mycotoxins known as fumonisins has its roots in the 1970 outbreak of equine leukoencephalomalacia in South Africa. The mycotoxin-producing *Fusarium verticillioides* was isolated from moldy corn fed to the horses.[13] In light of these historical catastrophes, the levels of some mycotoxins in foods and feeds are routinely monitored by the U.S. Food and Drug Administration.[14]

## III. INHALATION STUDIES ON MOLD EXPOSURE

The above examples serve to strengthen the association between the consumption of foodstuffs contaminated with mycotoxins and adverse health effects, but provide no evidence for illness related to inhalation of mold spores, and possibly, mycotoxins. The notion of mold inhalation resulting in subsequent adverse health outcomes has been extensively studied. One of the earliest studies investigating the association between inhalation to mold and adverse health outcomes was that by Kozak which documented the type of molds present in seven homes where children with asthma/allergy resided.[15] As reviewed by Fung,[16] there have been seven case reports, three case-control studies, and 17 cross-sectional studies that have investigated this exposure-outcome relationship. Each study design provides a different angle in evaluating the possibility of disease resulting from inhaled mold but each has inherent limitations. Thus, it is necessary to review the compilation of studies rather than focusing on a single study or article. Briefly, the case reports instigate the hypothesis of an association between inhalation of mold and adverse health effects; however, they do not incorporate the use of appropriate controls to test this hypothesis. Cross-sectional studies provide a snapshot of the situation without underlying baselines or controls for comparison. Without a proper comparison group or follow-up over

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12. S. G. Revankar, *Clinical Implications of Mycotoxins and Stachybotrys*, 325 AM. J. MED. SCI. 262, 262-74 (2003).

13. W. F. O. Marasas, *Discovery and Occurrence of the Fumonisins: A Historical Perspective*, 109 ENVIRON. HEALTH PERSPECT. 239, 239-43 (2001).

14. G. E. Wood, *Mycotoxins in Foods and Feeds in the United States*, 70 J. ANIM. SCI. 3941, 3941-49 (1992).

15. P. P. Kozak, Jr. et al., *Currently Available Methods for Home Mold Surveys. II. Examples of Problem Homes Surveyed*, 45 ANN. ALLERGY 167, 167-76 (1980).

16. F. Fung & W. G. Hughson, *Health Effects of Indoor Fungal Bioaerosol Exposure*, 18 APPL. OCCUP. ENVIRON. HYG. 535, 535–44 (2003).

time, it is difficult to establish a relationship between exposure and outcome. An additional limitation with most of the cross-sectional studies published on mold is the reliance on self-administered or self-reported data with little or no confirmatory tests. Tables 1 and 2 outline the reported cross-sectional studies to date. These studies range from having as little as 14 self-reported questionnaires to indirect assessment of 13,495 children and 14,799 adults.

Of the three study designs, case-control studies, if properly conducted, give the best insight for establishing if an association exists from being exposed to some factor (e.g. mold spores) with the development of symptoms or illness, since they incorporate controls or a comparison group as part of their study population. Several shortcomings have been identified in the mold-related case-control studies, namely: lack of assessment of water damage, recall bias, and the absence of physical or laboratory data to support the reported symptoms. Of noteworthy mention, is the attempt of a double-blind placebo control trial to study the effect of exposure to mold, however, the study found no significant association.[17] Unfortunately, it also lacked proper controls and used questionable methodology. It should be noted that even if a study ascertains a significant association, the establishment of causation is much more stringent and requires adherence to Hill's criteria for causation.[18]

Nevertheless, a surge of mold exposure investigations followed the initial case report on the pediatric asthma/allergy cases by Kozak.[19] Mycotoxins produced from the mold species *Stachybotrys chartarum*, coined "toxic mold", were belied in the investigation of an outbreak of unexplained illness in a water-damaged home in 1986.[20] Residents complained of having headaches, sore throats, diarrhea, fatigue, dermatitis, and depression, and claimed that the symptoms subsided after remediation of the home. However, in such a case, the possibility of the Hawthorne effect cannot be ruled out. Since this report, numerous outbreaks of building-related illnesses have claimed certain genera of mold as the causative agent.[21] The first of the three major case-control studies thus far on

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17. H. W. Meyer et al., *Double Blind Placebo Controlled Exposure to Moulds*, 5 PROCEEDINGS OF THE 9TH INTERNATIONAL CONFERENCE ON INDOOR AIR QUALITY AND CLIMATE 19, 19-22 (2002).

18. A. S. Persad et al., *Linking Exposure to Disease: Causation in the Scientific Arena*, 3 COLUMNS-MOLD 6, 6-7, 56-57 (2003).

19. P. P. Kozak, Jr. et al., *Currently Available Methods for Home Mold Surveys. II. Examples of Problem Homes Surveyed*, 45 ANN. ALLERGY 167, 167-76 (1980).

20. W. A. Croft et al., *Airborne Outbreak of Trichothecene Toxicosis*, 20 ATMOS. ENVIRON. 549, 549-52 (1986).

21. M. J. Hodgson et al., *Building-Associated Pulmonary Disease from Exposure to Stachybotrys Chartarum and Aspergillus Versicolor*, 40 J. OCCUP. ENVIRON. MED. 241, 241-49 (1998); E. Johanning et al., *Health and Immunology Study Following Exposure to Toxigenic*

inhalation mold exposure was conducted in the Netherlands in 1990 involving school-aged children.[22] Their analysis showed no significant relationship between observed mold exposure and respiratory disease, but showed a significant likelihood of such an association in the presence of dust mites and/or mold, although the effect of mold alone could not be parsed.

One of the most coveted reports published on mold exposure and adverse health outcomes followed a few years later. In 1994, the Centers for Disease Control and Prevention (CDC) investigated one of the most serious outbreaks of human disease attributed to mold.[23] Ten infants from Cleveland, Ohio, were diagnosed with acute idiopathic pulmonary hemorrhage. The CDC conducted a case-control study and initially found an association between pulmonary hemorrhage (PH) and exposure to molds. However, the CDC later rescinded its earlier report and determined that, based on the collected data, it was not possible to conclude a causal association between PH and exposure to molds.[24] Interestingly, a similar cluster of PH cases was found during the same time in Chicago, Illinois.[25] Though these patients presented with very similar symptoms, molds were not identified or implicated as the causal agents. In this latter cluster of cases, *Serratia* species, *Staphylococcus aureus*, and respiratory syncytial virus were identified in three of the eight cases.

Studies by Flappan and Elidemir also reported cases of children with pulmonary hemorrhage and have claimed *Stachybotrys* as the causative agent.[26] However, neither the

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*Fungi (Stachybotrys Chartarum) in a Water-Damaged Office Environment*, 68 INT. ARCH. OCCUP. ENVIRON. HEALTH 207, 207-18 (1996); J. Q. Jarvis & P. R. Morey, *Allergic Respiratory Disease and Fungal Remediation in a Building in a Subtropical Climate*, 16 APPL. OCCUP. ENVIRON. HYG. 380, 380-88 (2001); C.-S. Li et al., *Indoor Pollution and Sick Building Syndrome Symptoms among Workers in Day-Care Centers*, 52 ARCH. ENVIRON. HEALTH 200, 200-07 (1997); M. Seuri et al., *An Outbreak of Respiratory Diseases among Workers at a Water-Damaged Building - A Case Report*, 10 INDOOR AIR 138, 138-45 (2000).; G.-H. Wan & C.-S. Li, *Dampness and Airway Inflammation and Systemic Symptoms in Office Building Workers*, 54 ARCH. ENVIRON. HEALTH. 58, 58-63 (1999).

22. A. P. Verhoeff et al., *Damp Housing and Childhood Respiratory Symptoms: The Role of Sensitization to Dust Mites and Molds*, 141 AM. J. EPIDEMIOL. 103, 103-10 (1995).

23. CDC, *Update: Pulmonary Hemorrhage / Hemosiderosis among Infants--Cleveland, Ohio, 1993-1996*, 46 MORB. MORTAL. WKLY. REP. 33, 33-35 (1997).

24. CDC, *From the Centers for Disease Control and Prevention. Update: Pulmonary Hemorrhage / Hemosiderosis among Infants-Cleveland, Ohio, 1993-1996*, 283 JAMA 1951, 1951-53 (2000).; E. H. Page & D. B. Trout, *The Role of Stachybotrys Mycotoxins in Building-Related Illness*, 62 AIHAJ 644, 644-48 (2001).

25. CDC, *Current Trends. Acute Pulmonary Hemorrhage among Infants--Chicago, April 1992-November 1994*, 44 MORB. MORTAL. WKLY. REP. 67, 67, 73-74 (1995).

26. S. M. Flappan et al., *Infant Pulmonary Hemorrhage in a Suburban Home with Water Damage and Mold (Stachybotrys Atra)*, 107 ENVIRON. HEALTH PERSPECT. 927, 927-30 (1999); O. Elidemir et al., *Isolation of Stachybotrys from the Lung of a Child with Pulmonary*

prevalence of PH in the general population nor the investigation of other risk factors or causative agents were addressed in either study. Another study on mold exposure and PH did not implicate *Stachybotrys*, but other mycotoxin-producing genera namely, *Penicillium* and *Trichoderma*.[27] Mold exposure among adults has been suggested as the causative agent for the infamous "sick building syndrome" (SBS), with workers complaining of compromised health due to mold growth within a structure. One case report documented a worker with a hypersensitive respiratory illness from working in an environment where different genera of molds were isolated by air and surface sampling.[28] However, the study lacked sufficient evidence to propel the hypothesis that mold exposure may be related to the worker's illness, considering eight other employees, six of which were considered exposed, did not report any illnesses similar to the original case. Additionally, elevated antibody levels were found in only two of the employees, one of which was categorized as being unexposed.

Williamson investigated the association of mold exposure and asthma among physicians by a matched case-control study.[29] The study did not directly focus on mold but household dampness and claimed that a strong correlation existed between mold exposure and dampness. No significant association was found between the presence of mold and asthma, although significant correlates were found for heavy mold presence. The study failed to address that the presence of mold does not necessarily translate to internalization via inhalation or ingestion. A similar situation is found in a study linking SBS to *Stachybotrys chartarum*. This mold was assumed to be the causative agent based on its presence in bulk samples that were taken from a Tennessee school; however, concentration levels or confirmation of actual exposure was not ascertained.[30]

As described by Persad, exposure route and levels as well as dose are important considerations in assessing if molds or the mycotoxins they may produce are related to adverse health effects.[31] The presence of mold does not imply the presence of mycotoxins.

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*Hemosiderosis*, 104 PEDIATRICS 964, 964-66 (1999).

27. W. E. Novotny & A. Dixit, *Pulmonary Hemorrhage in an Infant Following 2 Weeks of Fungal Exposure*, 154 ARCH. PEDIATR. ADOLESC. MED. 271, 271-75 (2000).

28. D. Trout et al., *Bioaerosol Lung Damage in a Worker with Repeated Exposure to Fungi in a Water-Damaged Building*, 109 ENVIRON. HEALTH PERSPECT. 641, 641-44 (2001).

29. I. J. Williamson et al., *Damp Housing and Asthma: A Case-Control Study*, 52 THORAX 229, 229-34 (1997).

30. C. M. Scheel et al., *Possible Sources of Sick Building Syndrome in a Tennessee Middle School*, 56 ARCH. ENVIRON. HEALTH 413, 413-17 (2001).

31. A. S. Persad et al., *Mold in the Literature: Evaluating Human Health Risks from Mold-Contaminated Buildings*, 2 COLUMNS-MOLD 6, 6-7, 54-55 (2003).

Limitations due to mold spore size, the amount of toxin that can be produced and naturally released in the indoor air environment all hinder the association that sufficient toxin can be inhaled to produce adverse health outcomes.

With the presence of mold not equilibrating to actual mold exposure, the problem lies in confirming if a person has actually been exposed. Croft reported on four instances of trichothecene exposure with clinical confirmation by urine testing.[32] However, the methodology used and the claims set forth have been questioned.[33], Some of the flaws identified include the lack of proper controls, use of ill-supported tests, lack of valid confirmation of mycotoxin, no assessment of length of time indoors, possible mold exposure at another location, occupational exposure, presence of other chemicals, family history, or smoking history.

## IV. CLOSING REMARKS

At present, no studies have shown that inhalation of mold spores in humans at levels expected in mold-contaminated indoor environments are responsible for causing measurable health effects.[34] In the legal setting, the theory that mold may cause adverse health outcomes is slowly being challenged as exemplified in the 2001 Texan case of *Allison v. Fire Insurance Exchange*, whereby a suit was served against the insurance company because of the presence of mold and a claim that health problems resulted. Though the jury awarded the plaintiff over $32 million, no remuneration was allotted for personal injury damages because of the incorporation of scientific evidence. Additionally, after appeal, the actual damages award was reduced to slightly over $4 million (in addition to prejudgment and postjudgment interest) and compensation for mental anguish was removed.[35] However, in an effort to "protect" the public, Rep. John Conyers, Jr. introduced the U.S. Toxic Mold Safety and Protection Act (H.R. 5040),[36] also known as "The Melina Bill". The Act mandates the generation of guidelines to prevent mold growth and standards for mold remediation as well

32. W. A. Croft et al., *Clinical Confirmation of Trichothecene Mycotoxicosis in Patient Urine*, 23 J. ENVIRON. BIOL. 301, 301-20 (2002).

33. R. E. Gots, *Mycotoxins and Disease: A Critique of a Key but Scientifically Flawed Study*, 2 COLUMNS-MOLD 4, 4-5, 57 (2003); A. S. Persad et al., *Mold in the Literature: Evaluating Human Health Risks from Mold-Contaminated Buildings*, 2 COLUMNS-MOLD 6, 6-7, 54-55 (2003).

34. C. A. Robbins et al., *Health Effects of Mycotoxins in Indoor Air: A Critical Review*, 15 APPL. OCCUP. ENVIRON. HYG. 773, 773-84 (2000); A. I. Terr, *Stachybotrys: Relevance to Human Disease*, 87 ANN. ALLERGY ASTHMA IMMUNOL. 57, 57-63 (2001).

35. Allison v. Fire Ins. Exch., 98 S.W.3d 227, (Tex. 3rd Dist. Ct. App. 2002).

36. *U.S. Toxic Mold Safety and Protection Act*, http://www.house.gov/conyers/Mold_Bill.pdf.

as a national insurance program and tax credits for mold inspections. Though no federal regulations currently exist for mold remediation, the Institute of Inspection, Cleaning and Restoration Certification (Vancouver, Canada), an independent, non-profit certification body has recently released a mold remediation standard, to serve as a guideline.[37]

37. *S520 - Standard and Reference Guide for Mold Remediation*, http://www.iicrc.org/.

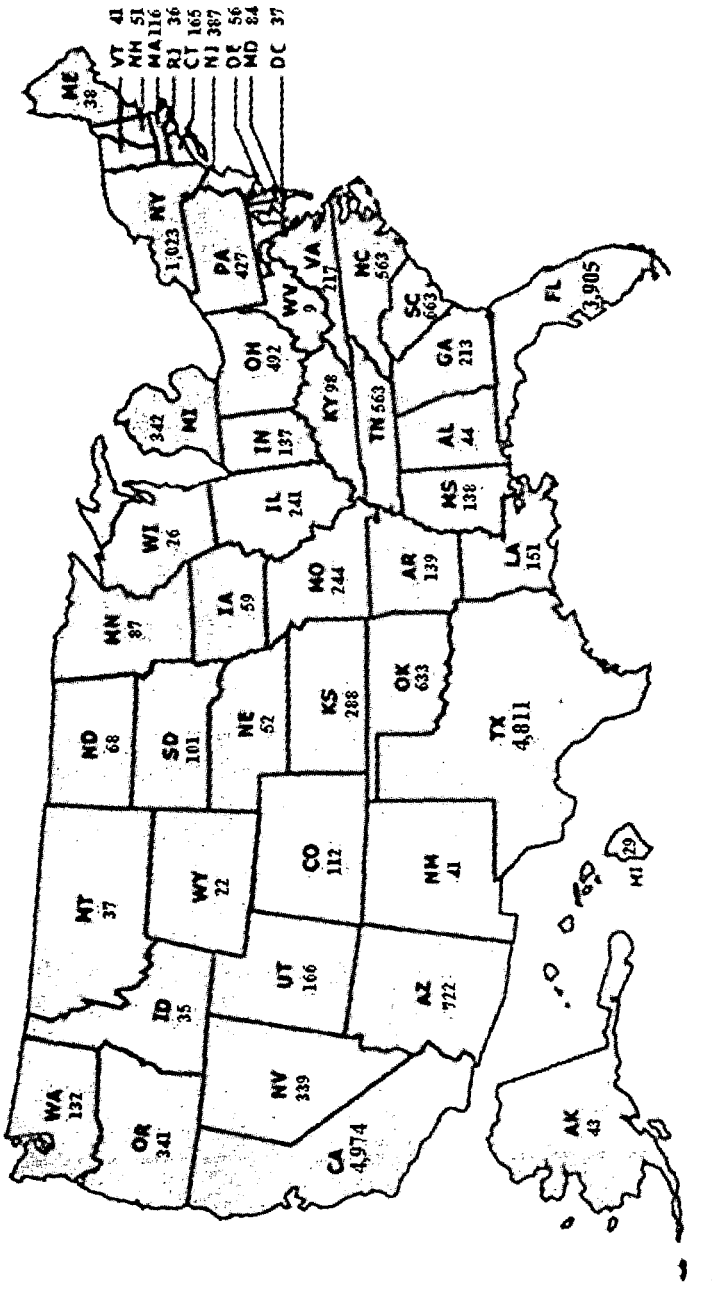


Figure 1. Total number of first party residential mold claims filed in each State between 1987 and 2002.

Table 1. Summary of Cross Sectional Pediatric Studies that Address Mold Exposure and Adverse Health Effect

| Reference | Sample size (children) | Results | Limitations |
|---|---|---|---|
| Brunekreff[49] | 4,625 | Correlation between respiratory symptoms and mold or damp homes | Exposure and symptoms were self reported |
| Cuijper[50] | 470 | No significant association between mold growth and chronic cough | Self reported (by parents); statistical association found between asthma-like symptoms and passive smoking |
| Dales[51] | 403 | 12-50% increase in symptoms prevalence associated with reported mold growth | No actual mold assessment conducted; health effects not defined |
| Dales[52] | 13,495 | Prevalence of respiratory symptoms significantly higher in homes with reported molds or dampness | Exposure and symptoms were self reported (by parents) |
| Jedrychowski[53] | 1,129 | Mold/dampness associated with respiratory symptoms | No actual mold assessment conducted; symptoms surveyed |
| Platt[54] | 1,169 | Association between respiratory health and mold growth observed in children | Presence of mold not quantified. |
| Strachan[55] | 1,000 | Onset of wheezing was significantly higher in homes reported to be moldy | Non-spore producing fungi isolated, high variability of mold levels |
| Waegemaeker[56] | 56 | Mold associated with respiratory symptoms | Symptoms self reported; other exposure agents not assessed |
| Yang [57] | 4,164 | Correlation between respiratory symptoms and dampness | Mold/dampness associated with respiratory symptoms (by parents); mold exposure indirectly assessed by dampness variable |

Amended from Fung.[58]

[49] B. Brunekreef et al., *Home Dampness and Respiratory Morbidity in Children*, 140 AM. REV. RESPIR. DIS. 1363, 1363-67 (1989).
[50] C. E. J. Cuijpers et al., *Adverse Effects of the Indoor Environment on Respiratory Health in Primary School Children*, 68 ENVIRON. RES. 11, 11-23 (1995).
[51] R. E. Dales & D. Miller, *Residential Fungal Contamination and Health: Microbial Cohabitants as Covariates*, 107 ENVIRON. HEALTH PERSPECT. 481, 481-83 (1999).
[52] R. E. Dales et al., *Respiratory Health Effects of Home Dampness and Molds among Canadian Children*, 134 AM. J. EPIDEMIOL. 196, 196-203 (1991).
[53] W. Jedrychowski & E. Flak, *Separate and Combined Effects of the Outdoor and Indoor Air Quality on Chronic Respiratory Symptoms Adjusted for Allergy among Preadolescent Children*, 11 INT. J. OCCUP. MED. ENVIRON. HEALTH. 19, 19-35 (1988).
[54] S. D. Platt et al., *Damp Housing, Mold Growth, and Symptomatic Health State*, 298 BR. MED. J. 1673, 1673-78 (1989).
[55] D. P. Strachan et al., *Quantification of Airborne Moulds in the Homes of Children with and without Wheeze*, 45 THORAX 382, 382-87 (1990).
[56] M. Waegemaekers et al., *Respiratory Symptoms in Damp Homes. A Pilot Study*, 44 ALLERGY 192, 192-98 (1989).
[57] C.-Y. Yang et al., *Damp Housing Conditions and Respiratory Symptoms in Primary School Children*, 24 PEDIATR. PULMONOL. 73, 73-77 (1997).
[58] F. Fung & W. G. Hughson, *Health Effects of Indoor Fungal Bioaerosol Exposure*, 18 APPL. OCCUP. ENVIRON. HYG. 535, 535–44 (2003).

Table 2. Summary of Cross Sectional Adult Studies that Address Mold Exposure and Adverse Health Effect

| Reference | Sample size | Results | Limitations |
|---|---|---|---|
| Bholah[59] | 23 office buildings | Prevalence of fungi was higher in naturally ventilated areas | Self reported, health effects not clearly defined |
| Dales[60] | 14,799 adults | Mold/dampness associated with respiratory symptoms | Mold/dampness associated with respiratory symptoms |
| Hodgson [61] | 14 adults | Increase in symptoms prevalence associated with potential mold exposure | Self reported; symptoms not well defined |
| Jarvis[62] | 488 adults | Respiratory symptoms associated with mold exposure | Self reported; symptoms not well defined |
| Johanning [63] | 53 adults | Respiratory symptoms associated with mold exposure | Control environment not sampled; symptoms not well defined |
| Li[64] | 264 adults | Significant association between mold and eye irritation, pharyngeal symptoms, and difficulties in concentration not respiratory symptoms | Self reported; other exposures not assessed |
| Ross [65] | 57 adults | Mold spore abundance only associated with emergency room visits for asthma | Little evidence for relationship between asthma severity and indoor mold levels |
| Ruotsalainen[66] | 268 workers | Eye and respiratory symptoms associated to exposure to water damage and mold odor | Self reported |
| Sudakin[67] | 37 adults | Prevalence of respiratory symptoms higher before relocation from water damaged building; extensive mold assessment | No controls |

Amended from Fung.[68]

[59] R. Bholah & A. H. Subratty, *Indoor Biological Contaminants and Symptoms of Sick Building Syndrome in Office Buildings in Mauritius*, 12 INT. J. ENVIRON. HEALTH RES. 93, 93-98 (2002).
[60] R. E. Dales et al., *Respiratory Health Effects of Home Dampness and Molds among Canadian Children*, 134 AM. J. EPIDEMIOL. 196, 196-203 (1991).
[61] M. J. Hodgson et al., *Building-Associated Pulmonary Disease from Exposure to Stachybotrys Chartarum and Aspergillus Versicolor*, 40 J. OCCUP. ENVIRON. MED. 241, 241-49 (1998).
[62] J. Q. Jarvis & P. R. Morey, *Allergic Respiratory Disease and Fungal Remediation in a Building in a Subtropical Climate*, 16 APPL. OCCUP. ENVIRON. HYG. 380, 380-88 (2001).
[63] E. Johanning et al., *Health and Immunology Study Following Exposure to Toxigenic Fungi (Stachybotrys Chartarum) in a Water-Damaged Office Environment*, 68 INT. ARCH. OCCUP. ENVIRON. HEALTH 207, 207-18 (1996).
[64] C.-S. Li et al., *Indoor Pollution and Sick Building Syndrome Symptoms among Workers in Day-Care Centers*, 52 ARCH. ENVIRON. HEALTH 200, 200-07 (1997).
[65] M. A. Ross et al., *Association of Asthma Symptoms and Severity with Indoor Bioaerosols*, 55 ALLERGY 705, 705-11 (2000).
[66] R. Ruotsalainen et al., *Dampness and Molds in Day-Care Centers as an Occupational Health Problem*, 66 INT. ARCH. OCCUP. ENVIRON. HEALTH. 369, 369-74 (1995).
[67] D. L. Sudakin, *Toxigenic Fungi in a Water-Damaged Building: An Intervention Study*, 34 AM. J. INDUS. MED. 183, 183-90 (1998).
[68] F. Fung & W. G. Hughson, *Health Effects of Indoor Fungal Bioaerosol Exposure*, 18 APPL. OCCUP. ENVIRON. HYG. 535, 535–44 (2003).