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Review

Water-Damaged Buildings and Health: An Updated Review of the Evidence, Sources of Bias, and Co-Occurring Conditions

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Abstract

Background. Water damage in buildings produces a mixture that includes fungi, bacteria, endotoxin, microbial fragments, and moisture-driven chemical emissions. The respiratory evidence has been graded; evidence on the multisystem symptoms many people report has been graded limited or inadequate, or not graded. **Objectives.** To summarize the evidence as of 2026; to examine how confounding, reporting bias, the composition of the exposure mixture, and co-occurring clinical conditions bear on the observed associations; and to propose a protocol for a rigorous future review. **Methods.** Structured evidence review (PubMed, Europe PMC, agency sources) with structured supplementary searches by body system and by exposure agent in November 2026 (145 database strategies); no registered protocol or duplicate screening. **Results.** The IOM and WHO graded no outcome as causal; two later reviews judged the evidence causal for asthma exacerbation in children, whereas a 2024 GRADE review rated effects in people with asthma uncertain. Pooled respiratory odds ratios are about 1.3 to 1.8, smaller in cohorts, and change modestly with socioeconomic adjustment. Measured agents such as endotoxin show both adverse and inverse associations. No epidemiological study of metabolic, hepatic, renal, endocrine, cardiovascular, or cancer outcomes, or of occupants' gut microbiome, was identified; these gaps indicate neither presence nor absence of an effect. No identified study systematically screened exposed people for bile acid malabsorption, iron deficiency, thyroid disease, sleep apnea, perimenopause, celiac disease, or postural orthostatic tachycardia syndrome. HLA-DR typing has not been shown to provide diagnostic information. **Conclusions.** The respiratory and allergic evidence is sufficient to establish association. Beyond the airway, associations are reported for mental health, general symptoms, child neurodevelopment, skin, immune markers, and sarcoidosis, mostly in cross-sectional and self-reported designs; for metabolic, hepatic, renal, endocrine, cardiovascular, and cancer outcomes no epidemiological study was identified. The studies that would establish or exclude these associations have not been carried out, and tests used to assess exposure and susceptibility in an individual have not been validated for that purpose. The review sets out the design that research capable of answering these questions would require.

Keywords: dampness; water-damaged buildings; indoor mold; mycotoxins; indoor air quality; exposure assessment; co-occurring conditions; evidence review

1. Introduction

Persistent moisture in buildings is common. A pooled analysis of European housing surveys estimated that about one dwelling in six shows at least one indicator of dampness, mold, or water damage, with prevalence varying by region, climate, and survey method [1]. Water entering a building does not introduce a single agent. It supports the growth of fungi, bacteria including actinomycetes, and dust mites; it releases spores, cell-wall fragments, (1→3)-β-D-glucans, endotoxin, and microbial volatile organic compounds; and it accelerates chemical emissions from damp building

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materials such as plasticizer breakdown products, ammonia, and formaldehyde [2]. Human studies of these emissions concern mainly plasticizer breakdown products (Section 3.5.2); no human study of ammonia from damp materials was found, and for formaldehyde only one residential study was found [3]. Throughout this review, "water-damaged buildings" refers to that whole mixture, and "dampness" is used where the source studies use it.

Two landmark assessments framed the field. The Institute of Medicine (IOM) committee concluded in 2004 that excessive indoor dampness is a public health problem but found that no health outcome met its definition of sufficient evidence of a causal relationship [4]. The World Health Organization (WHO) guidelines of 2009 broadened the list of respiratory outcomes with sufficient evidence of association, declined to set quantitative guideline values for microbial contamination, and recommended that persistent dampness be prevented [2]. Since then, major narrative and systematic reviews [5–7] and the 2023 update of the German AWMF clinical guideline [8] have revisited the grading.

Meanwhile, people report chronic, multisystem symptoms, including fatigue, cognitive difficulty, gastrointestinal complaints, sleep disruption, and neurological symptoms, that began or worsened in association with water-damaged buildings; Nordic population surveys put self-reported intolerance to certain buildings at 4.8% of Swedish and 7.2% of Finnish adults (Section 3.2.3) [9,10]. These experiences sit largely outside what the authoritative assessments were able to grade. Diagnostic tests and treatment protocols offered for these symptoms include methods that the 2023 AWMF guideline lists as lacking sufficient scientific evidence, among them visual contrast sensitivity testing and cholestyramine "detoxification" [[8], tables 11 and 12; [11]] (Section 3.2.11).

What these assessments and reviews have not done is examine the conditions that co-occur with these presentations alongside the epidemiology, as a factor in how cases are defined and how outcomes are classified. No study of illness associated with water-damaged buildings was identified that screened for them systematically (Section 3.6.5). This review asks three questions. First, what does the current evidence show about water-damaged buildings and human health? Second, how robust are the observed associations to confounding and reporting bias, which components of the exposure mixture might account for them, and how might co-occurring clinical conditions, including bile acid malabsorption, iron deficiency, thyroid disease, sleep apnea, perimenopause, celiac disease, postural orthostatic tachycardia syndrome (POTS), mast cell activation syndrome (MCAS), myalgic encephalomyelitis/chronic fatigue syndrome (ME/CFS), long COVID, and depression, affect case definitions, outcome classification, and the interpretation of treatment response? Third, what would a rigorous future evidence review need to look like? Throughout, the question of what the observed associations represent is framed in three parts: the contribution, if any, of moisture and its microbial growth; the contribution of other agents that accompany water damage; and the contribution of other conditions in the person. Co-occurring conditions are not treated as alternative explanations by default. In a given person, such a condition may coexist with an exposure-related effect and add to symptoms, may produce symptoms that resemble one, or may go unrecognized; its presence does not by itself indicate whether the exposure contributed.

2. Methods

This is a structured evidence review of authoritative assessments, systematic reviews and meta-analyses, and primary studies, covering health outcomes by body system, the components of the exposure mixture, and co-occurring clinical conditions. The supplementary search is documented in full: its 145 database strategies, with the exact strings and the hit counts returned on the search date, are listed in the Appendix, and the query behind every figure drawn from an agency database is given below. It is not a systematic review, and that term is not used for it anywhere in this paper: no protocol was registered in advance, no formal risk-of-bias instrument was applied across studies, and study selection, data extraction, and interpretation were not performed in duplicate.

Sources were identified through searches of PubMed and Europe PMC and through agency and institutional sources (National Academies Press, WHO Institutional Repository, US Environmental

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Protection Agency, Centers for Disease Control and Prevention, National Center for Health Statistics, National Toxicology Program, and NIH RePORTER) through November 2026. NIH funding was estimated with the NIH RePORTER application programming interface (https://api.reporter.nih.gov/v2/projects/search), queried on November 8, 2026, for fiscal years 2015 to 2026 (fiscal year 2026 incomplete), using an advanced text search of project titles, abstracts, and indexing terms for any of the phrases "dampness," "water-damaged," "water damaged," "damp indoor," "indoor mold," "indoor mould," "indoor fungi," or "indoor fungal." Award amounts for all returned records, including interagency-agreement subprojects, were summed by core project number. The search returned 43 records from 13 core projects ($15.7 million); one intramural laboratory-space record that matched only on indexing terms was excluded, leaving 12 core projects ($14.3 million), whose titles were screened for relevance. Search themes covered (a) authoritative grading of health outcomes; (b) pooled effect estimates and intervention trials for respiratory and allergic outcomes; (c) non-respiratory and multisystem outcomes, including mental health, sleep, neurocognitive, and urinary mycotoxin studies; (d) exposure assessment methods; (e) proposed biological mechanisms; (f) confounding, the components of the exposure mixture, reporting bias and misclassification, and reverse causation; (g) the prevalence, presentation, and under-recognition of co-occurring clinical conditions; and (h) frameworks for environmental health evidence review.

A structured supplementary search was added in November 2026 to update the evidence by body system. It covered four areas: (1) neurological, cognitive, psychiatric, and sleep outcomes and child neurodevelopment; (2) respiratory outcomes, skin and eye outcomes, reproductive outcomes, cancer, occupational and school settings, floods, and recent systematic and umbrella reviews; (3) gastrointestinal, hepatic, metabolic, and renal outcomes and the microbiome; and (4) immune and inflammatory outcomes, fatigue and environmental intolerance, endocrine and cardiovascular outcomes, and HLA typing. Two further areas addressed components of the exposure: (5) indoor mycotoxins (occurrence, biomonitoring, guidance, and studies of measured toxins and health; purified-toxin dosing, production, and aerosolization excluded) and (6) agents other than fungi (bacterial agents, glucans, microbial volatile compounds, controlled exposures, the indoor microbiome, chemical emissions, and multi-agent studies). Each area used area-specific strategies in PubMed (E-utilities) and Europe PMC (REST interface), 145 database strategies in all (23, 31, 22, 20, 23, and 26 by area), supplemented by web searches to locate a publication recalled by the authors, professional statements on HLA typing, and agency guidance on indoor mold. All strategies were run on November 9, 2026; the exact strings and hit counts are listed in the Appendix. Records were screened in a single pass, assisted by software tools, without duplicate review. Candidate studies were checked against the existing reference list, and citation details were verified against PubMed or Europe PMC records. Effect estimates from the supplementary search were taken from abstracts unless otherwise stated; the full texts of the AWMF guideline, the 2025 position statement of the American College of Medical Toxicology, the 2016 ANSES expert report, and selected primary studies were read. Where an abstract reported no effect estimate, only the direction of the finding is described. Where several reports drew on one dataset, they are identified as such and are not counted as independent replications.

The IOM 2004 report was read from the printed page scans of the Executive Summary and Chapter 5, and the WHO 2009 guidelines from the official WHO repository PDF. Journal citations were checked against PubMed, Europe PMC, publisher, or agency records for authors, year, journal, volume, pages, DOI, and PMID. Effect estimates were taken from abstracts or, where stated, full texts. Claims that could be traced only to secondary sources are labeled as secondhand in the text or were omitted. Literature retrieval and initial extraction were assisted by software tools, including a large language model–based research assistant; the authors checked the citations as described and are responsible for the selection and interpretation of all sources.

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3. Results

3.1. Respiratory and Allergic Outcomes

3.1.1. How the Authoritative Assessments Graded the Evidence

The IOM committee used two parallel tables, one for exposure to damp indoor environments and one for the presence of mold or other agents in damp indoor environments [4]. In both, the category "sufficient evidence of a causal relationship" contained no outcomes. Sufficient evidence of an association was found for upper respiratory (nasal and throat) symptoms, cough, wheeze, and asthma symptoms in sensitized persons with asthma, with hypersensitivity pneumonitis in susceptible persons added in the mold-or-other-agents table. Dyspnea, lower respiratory illness in otherwise healthy children, and asthma development were rated limited or suggestive for dampness, but dyspnea and asthma development were rated inadequate in the mold-or-other-agents table [4]. The committee defined "sufficient evidence of an association" as evidence in which chance, bias, and confounding could be ruled out with reasonable confidence, and it stated explicitly that it was not assuming that outcomes resulted from a particular mold or from microbial agents in general [4].

WHO 2009 found sufficient epidemiological evidence of associations between dampness or mold and asthma development, asthma exacerbation, current asthma, respiratory infections (except otitis media), upper respiratory tract symptoms, cough, wheeze, and dyspnea, and it noted that four of these outcomes (asthma development, dyspnea, current asthma, and respiratory infections) had not been so classified by the IOM [2]. It rated bronchitis and allergic rhinitis as having limited or suggestive evidence of association, and altered lung function, allergy or atopy, and "asthma, ever" as inadequate or insufficient [2, pp. 71, 90]. On causality, WHO agreed with the IOM that the evidence was insufficient for any specific outcome, while judging that the evidence for asthma exacerbation was "almost" enough to meet the criteria of causality, supported by one strong intervention trial in children [2,12].

Later reviews moved further. A 2011 review of studies through late 2009 found consistent associations between evident dampness or mold and a wide range of respiratory and allergic outcomes, in both allergic and nonallergic people, and judged that the evidence strongly suggested causation of asthma exacerbation in children [5]. A 2015 update of the IOM asthma-exacerbation review, covering 69 articles, rated dampness and dampness-related agents as having sufficient evidence of a causal association in children and sufficient evidence of an association in adults, while asking that the conclusions be considered provisional [6]. The French agency ANSES, reporting 61 publications from 2006 to November 2017, concluded that visible mold and mold odor had sufficient evidence of a causal relationship with asthma development and exacerbation in children, while adult population studies were too few and divergent to support more than a limited level of evidence [7]. The 2023 AWMF guideline lists allergic respiratory diseases, asthma, bronchitis, rhinosinusitis, hypersensitivity pneumonitis, aspergillosis, and the favoring of respiratory infections under sufficient evidence for an association [8]. A 2024 systematic review for the European Academy of Allergy and Clinical Immunology applied GRADE, which assigns observational evidence a starting certainty of low [13]: it rated damp or mold exposure as increasing the risk of new-onset wheeze with moderate certainty, but judged the effect of indoor pollutants, damp or mold included, on asthma-related outcomes in people with asthma, such as exacerbation and control, to be uncertain [14]. The 2024 review applied GRADE [14], whereas the earlier reviews applied IOM-derived evidence categories [5–7], and the four reviews covered different publication windows and included different sets of studies. An umbrella review of risk factors for allergic rhinitis classed indoor dampness (OR 1.49, 1.27–1.75) and indoor mold (OR 1.66, 1.26–2.18) as "highly suggestive" rather than "convincing" evidence [15]. Each upgrade to "causal" applied to dampness or mold as a building condition, and none identified a specific organism or measured agent as the cause [5–7].

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3.1.2. Pooled Estimates and Dose-Response

Meta-analyses consistently report pooled odds ratios between about 1.3 and 1.8. Across ten respiratory and asthma outcomes, central estimates ranged from 1.34 to 1.75, with the confidence interval excluding unity in nine [16]. For respiratory infections and bronchitis, estimates restricted to confounder-adjusted studies were 1.44 (95% CI 1.31–1.59) and 1.45 (1.32–1.59), with attributable proportions of 8% to 20% if the associations are causal [17]. A 2023 meta-analysis restricted to children in high-income countries (30 studies, 22 of them cross-sectional and 24 rated poor or fair quality) gave pooled odds ratios from 1.28 (1.08–1.53) for dampness and respiratory infections to 1.76 (1.64–1.88) for mold and infection-compatible symptoms, which its authors described as compatible with a weak to moderate effect [18]. For schools, a meta-analysis of 11 cross-sectional studies found central odds ratios above 1 for all outcomes, including 1.32 for cough and 1.68 for wheeze [19]. For asthma development, a meta-analysis of 16 cohort and incident case-control studies gave summary estimates of 1.48 (1.23–1.78) using the highest and 1.27 (1.06–1.53) using the lowest study estimates; mold odor gave the largest estimate (1.73, 1.19–2.50), while water damage alone did not reach significance (1.12, 0.98–1.27) [20]. In children, visible mold was associated with asthma (1.49, 1.28–1.72) and wheeze (1.68, 1.48–1.90) [21]. For rhinitis, mold odor (2.18, 1.76–2.71) and visible mold (1.82, 1.56–2.12) were both associated [22].

The size of the association depends on study design. A 2025 meta-analysis restricted to longitudinal designs found a 53% increase in asthma risk across case-control studies, without heterogeneity, but only a 15% increase (1%–31%) across cohort studies, with substantial heterogeneity (I^2 = 60.5%) [23]. Among adults followed for nine years in the European Community Respiratory Health Survey, baseline water damage, indoor mold, and observed damp spots predicted new asthma with relative risks of about 1.3 to 1.5 [24], whereas in an eight-year Nordic cohort dampness predicted new respiratory symptoms but not asthma onset [25]. In the ten-year follow-up of 11,506 adults in the Respiratory Health in Northern Europe cohort, dampness and mold at home or at work predicted onset of respiratory symptoms and asthma (OR 1.23–2.24) and reduced remission [26]; at 20 years, cumulative exposure strengthened these associations, with the largest estimates for combined home and work exposure (doctor-diagnosed asthma OR 1.90) [27]. Both rely on self-reported exposure and outcomes. In a Swedish birth cohort followed to age 16, infant exposure was associated with persistent asthma (OR 1.73, 1.20–2.50) and with nonallergic asthma (1.80, 1.27–2.55), but not with IgE sensitization [28], and in a Finnish birth cohort with engineer inspections at about five months, moisture damage with visible mold in the child's bedroom or living room, but not in bathrooms or other spaces, predicted asthma by age 6, with wide confidence intervals [29]. In eight European birth cohorts, early mold or dampness predicted asthma symptoms in the first two years and allergic rhinitis at ages 3 to 10, but not asthma at school age [30]. In these comparisons, the cohort designs reported smaller effects than the case-control designs [23]. The cohort studies cited here assessed exposure once at baseline [24,26,27]. None of these reports tested whether the smaller cohort estimates are explained by differences in recall and selection bias, by differences in exposure misclassification, or by both.

Dose-response patterns are present but are built on observation-based scales. Across 33 multilevel observational metrics, health risks generally increased with the observed extent of dampness or mold, and simple metrics, especially mold odor, had among the strongest associations [31]. Among 28,596 French adults, reported mold area showed a graded association with current asthma and asthma symptom score, including for small areas [32]. In New Zealand children under two years, an assessor-scored damp–mold index was associated with hospitalization for acute respiratory infection (adjusted OR 1.15 per unit, 1.02–1.30), whereas a broader housing-hazard index lost significance after adjustment for tenure, socioeconomic status, and crowding [33]. Only three eligible studies used measured building moisture; two UK studies showed dose-related increases in asthma exacerbation with higher wall moisture, including an adjusted odds ratio of 7.0 for nighttime symptoms [34]. ANSES concluded that no threshold below which no effect is expected can be defined [7].

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3.1.3. Intervention and Remediation Trials

The experimental evidence consists of remediation and housing-intervention trials. The 2015 Cochrane review included 12 studies with 8,028 participants and found moderate-quality evidence that repairing mold-damaged houses reduced wheeze (OR 0.64, 0.55–0.75) and rhinitis (OR 0.57, 0.49–0.66) in adults; evidence for children, offices, and schools was limited or of very low quality [35]. In the Cleveland trial of 62 children with asthma, construction remediation of moisture sources combined with asthma education reduced symptom days, and exacerbations fell substantially in the as-treated analysis, although the intention-to-treat difference in exacerbations (28.1% versus 10.0%) did not reach significance [12]. In a Welsh trial of 164 households, removal of visible mold with fungicide and installation of a fan improved wheeze, perceived breathing, medication use, and rhinitis, but not peak flow variability; the authors noted the absence of objective benefit while judging a pure placebo effect unlikely [36]. Insulation and heating trials in New Zealand reduced wheeze, poor self-rated health, school absence, and health care use, but they also changed temperature, humidity, and nitrogen dioxide at the same time [37–39]. A systematic expert review of 11 housing interventions rated combined elimination of moisture intrusion and leaks with removal of moldy items as having sufficient evidence for implementation to reduce mold and respiratory symptoms [40]. No randomized trial of dampness or mold remediation was identified after the 2015 Cochrane review, which has not been updated. In a Quebec trial of 83 children with asthma, installing mechanical ventilation lowered airborne mold spores and formaldehyde and reduced the proportion of children with wheezing episodes but did not change symptom days, and the authors judged the trial underpowered [41]. In a New Zealand national program covering nearly one million residents, retrofitted insulation was associated with fewer respiratory (relative rate ratio 0.85, 0.81–0.90) and asthma admissions (0.80, 0.70–0.90), although dampness and mold were not measured [42]. Children with respiratory conditions attending moisture-damaged schools showed a larger fall in diary-recorded symptoms during summer holidays than children at reference schools, a within-person comparison that is parent-reported and seasonally confounded [43], whereas in an office building followed over seven years of repeated remediation attempts, severe respiratory symptoms worsened (Section 3.2.3) [44]. Across these studies, symptom outcomes improved more consistently than measures of lung function, and none of the trials blinded participants to the intervention.

3.1.4. Hypersensitivity Pneumonitis and Fungal Infection

Some conditions rest on clinical rather than epidemiological evidence. The IOM found sufficient evidence of an association between mold or bacteria in damp indoor environments and hypersensitivity pneumonitis in susceptible persons, describing it as relatively rare and immune-mediated [4]. In severely immunocompromised persons, who were excluded from its main tables, the IOM found sufficient evidence of an association between exposure to *Aspergillus* species and pulmonary aspergillosis and aspergillomas, while noting that it was less clear whether such fungal exposure related to moisture problems indoors [4]. WHO cited clinical evidence that mold and other dampness-related microbial agents increase the risks of hypersensitivity pneumonitis, allergic alveolitis, chronic rhinosinusitis, and allergic fungal sinusitis, and found sufficient clinical evidence for mold infections in susceptible individuals and for humidifier and inhalation fevers [2]. The AWMF guideline places allergic bronchopulmonary aspergillosis and mycoses under sufficient evidence [8]. After Hurricane Harvey, invasive mold infections at four Houston hospitals rose from 2.50 to 3.69 per 10,000 encounters (rate ratio 1.48, 1.10–2.00), mostly under a new surveillance case definition, and about a quarter of patients lacked classical risk factors [45]; an insurance-claims analysis over the same period found no change in coded invasive mold infections [46].

3.1.5. Floods and Occupational Settings

Six months after Hurricanes Katrina and Rita, respiratory symptom scores in a random sample of New Orleans adults rose with exposure to water-damaged homes, including among people who

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were only inside them, and respirator use was associated with less worsening of lower respiratory symptoms [47]. After Hurricane Sandy, mold or damp exposure was associated with about twofold odds of lower respiratory symptoms in a registry cohort with pre-storm data [48], and after Hurricane Harvey, flood and mold exposure were associated with upper respiratory allergic symptoms and stress [49]. These studies rely on self-report and cannot separate mold from displacement, stress, and cleanup chemicals. In occupational settings, workplace mold odor was associated with incident asthma–COPD overlap in a Finnish population-based incident case-control study with 25 overlap cases (OR 3.43, 1.04–11.29), while home exposures and other workplace dampness indicators were not [50], and in a cross-sectional survey of Veterans Administration health care workers, asthma was associated with mold and dampness as well as with construction dust and cleaning and disinfecting tasks [51].

3.2. Non-Respiratory and Multisystem Outcomes

This section is organized by body system. For each, the studies identified, including those from the supplementary search (Section 2), are described with their designs, and outcomes for which no study was identified are stated explicitly.

3.2.1. How These Outcomes Have Been Graded

The IOM placed skin symptoms, gastrointestinal problems, fatigue, neuropsychiatric symptoms, cancer, reproductive effects, and rheumatologic and other immune diseases in the category "inadequate or insufficient evidence to determine whether an association exists" [4, pp. 9-10]. That category is defined to include evidence of insufficient quality, consistency, or power and the situation in which no studies exist. The committee observed that few epidemiologic studies had examined these outcomes, that they appeared only as part of broad symptom surveys, and that little attention had been paid to evaluating alternative explanations; it called for greater research attention to the possible role of damp indoor environments in less well understood disease entities [4]. The contemporaneous press release stated that the committee found very few studies examining whether dampness or mold is linked to fatigue or neuropsychiatric disorders, that the little evidence available "does not support an association," and that "because of the dearth of well-conducted studies and reliable data, the committee could not rule out the possibility" [52]. WHO did not assign an evidence category to these outcomes; its epidemiological review excluded "outcomes on which limited research has been reported," giving as examples effects related to skin, eyes, fatigue, nausea, headache, insomnia, mucous membrane irritation, and sick-building syndrome [2, p. 64]. The AWMF guideline rates mood disorders, mucous membrane irritation, and odor effects as limited or suspected, and rates chronic fatigue syndrome, neuropsychological and neurotoxic effects, multiple chemical sensitivity, gastrointestinal effects, autoimmune diseases, and thyroid disease as inadequate [8].

In these schemes, "inadequate" means that the available research cannot answer the question; it is neither a finding of no effect nor evidence of an effect. For the fatigue, cognitive, gastrointestinal, and neurological symptoms that many people report, few studies with designs capable of separating these outcomes from alternative explanations have been published since the IOM called for that research in 2004 [4] (Sections 3.2.2 to 3.2.4).

3.2.2. Mental Health and Sleep

The searches described in Section 2 identified more studies for mental health and sleep than for the other non-respiratory outcomes in this section. A 2024 state-of-the-science review included 19 reports from 2003 to 2023 and found positive associations between residential dampness or mold and poor mental health, while noting methodological limitations in the available evidence base; most of the included studies were cross-sectional and the review performed no meta-analysis [53]. Reported associations included anxiety (OR 1.25, 1.06–1.47), 14 or more mentally unhealthy days (adjusted OR

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1.77, 1.27–2.47), and postnatal depression (OR 1.4, 1.02–1.91) [53]. In eight European cities, depression odds ratios were 1.39, 1.44, and 1.34 for minimal, moderate, and extensive dampness or mold, attenuating partially after adjustment for perceived control over the home and for physical illness [54]. That attenuation is consistent with more than one interpretation: perceived control and physical illness may lie on a pathway from exposure to depression, in which case adjustment removes part of a real effect, or they may share causes with both dampness and depression, in which case adjustment removes confounding; a cross-sectional design cannot distinguish the two.

Studies published since that review include one with a longitudinal, within-person design. In the British Household Panel Survey, individual fixed-effects models, which compare each person with themselves over time and so remove stable characteristics such as personality and long-term socioeconomic position, found that self-reported damp housing was associated with psychological distress on the General Health Questionnaire (OR 1.09, 1.05–1.14), with odds rising for each additional dampness indicator (OR 1.04, 1.02–1.07) [55]; time-varying confounders, such as job loss or a move to poorer housing, may remain. In the same data, the association was stronger among people with a chronic respiratory condition (OR 1.27 versus 1.07) [56]. In the Taiwan Birth Cohort, mold spots at home were associated with postpartum depression among 17,981 mothers (OR 1.31, 1.20–1.42), although exposure and outcome were reported on the same questionnaire and other household exposures, such as renovation and insecticide use, were also associated [57]. Several cross-sectional analyses of one wave of the Chinese Longitudinal Healthy Longevity Survey, each based on a single self-reported mold item, reported odds ratios for depressive symptoms in older adults ranging from 1.26 to 2.26 depending on the analytic approach [58,59]; because they share one dataset, they are not independent replications. A scoping review of 30 studies found that most reported associations that persisted in multivariable models, and its qualitative studies described fear about physical health effects and embarrassment about musty-smelling homes and clothing [60]. In a network analysis of pregnant women, housing problems that included leaks and condensation or mold were linked to depressive symptoms mainly through residents' feelings about their home [61]. The analysis is cross-sectional, and the authors drew no conclusion about the direction of these associations [61].

For sleep, in a ten-year cohort of 11,318 adults from five northern European countries, baseline floor dampness, visible mold, and mold odor predicted new onset of insomnia symptoms and snoring (OR 1.29–1.87), with both exposure and outcomes self-reported [62]. In German children, parent-reported visible mold or dampness was associated with sleep problems at age 10 (any sleep problem, OR 1.77, 1.21–2.60), but among children without allergic rhinoconjunctivitis only short sleep duration remained associated; the authors concluded that the influence of allergic rhinoconjunctivitis on this association requires further study [63]. In two Saskatchewan First Nations communities, dampness or water in the past 12 months and damage caused by dampness were associated with incident excessive daytime sleepiness over four years; having a house in need of repairs and having less money left at the end of the month were associated with the same outcome in that analysis [64]. These studies relied on self- or parent-reported exposure and sleep outcomes.

3.2.3. Fatigue, Headache, and Building-Related Symptoms

Population surveys report associations with general symptoms. In a Finnish study of 1,460 adults, tiredness and eye irritation remained associated with home dampness after excluding respondents who reported symptoms implausibly linked to mold, such as lumbar backache, an explicit check on reporting tendency [65]. In 4,815 Stockholm dwellings, odor combined with signs of high humidity was associated with all sick-building-type symptoms (OR 2.2–3.6), with a dose-response by the number of dampness signs [66]. In Finnish homes inspected by civil engineers, a three-level severity grade showed a dose-response for respiratory infections and for irritative and skin symptom scores [67]. Among 36,541 parents in six Chinese cities, every dampness sign examined was associated with weekly headache and fatigue, as well as with ocular, throat, and dermal symptoms (ORs 1.2–4.6), most strongly for mold odor and with a dose-response by the number of

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signs [68]; in a Swedish national sample of 5,775 adults in multi-family buildings, dampness, mold, and mold odor were risk factors particularly for headache and ocular symptoms [69]. The same Chinese survey also linked headache and fatigue to cockroaches, incense, redecoration, and gas cooking, in models adjusted for dampness and mold [70]. Inspector-scored exposure was used in one large study: in 50 schools whose rooms were scored with the NIOSH assessment tool, each one-unit increase in score was associated with a 6% to 16% higher prevalence of headache, dizziness, confusion, and difficulty remembering among 1,529 staff, an approximately log-linear exposure-response based on self-reported symptoms and reported with 90% confidence intervals [71].

In Finnish workplace and school comparisons, exposed occupants of a water-damaged police station reported more central nervous system symptoms and fatigue (relative risks about 2.8) [72], and a school study reported very large relative risks for headache, fatigue, gastrointestinal, and neurological symptoms, but with response rates of 12% and 18% and self-reported outcomes [73]. A third report from the same group, comparing 90 hospital staff in a water-damaged building with 45 unexposed staff, reported nervous system symptoms in 81% versus 11% and arrhythmias in 57% versus 2.4% (relative risk 19.75) [74]. All outcomes in that report were collected by questionnaire from occupants who knew their exposure status. Finnish research groups interpret this literature differently (Sections 3.5.4 and 3.6.5).

Nordic population surveys put self-reported intolerance to certain buildings at 4.8% of Swedish and 7.2% of Finnish adults, with a mean duration of 12 years and higher odds of somatic, psychiatric, and inflammatory airway comorbidity [9,10,75]; excess symptoms persisted after comorbid functional somatic syndromes, inflammatory disease, and mental disorders were taken into account [76]. In 83 patients with medically confirmed building-related symptoms, somatosensory amplification and modern health worries were not elevated relative to population norms and were only weakly to moderately associated with symptoms; the authors concluded that patients with building-related symptoms are not characterized by elevated modern health worries or somatosensory amplification, and that other psychosocial or environmental factors may have contributed [77]. The searches described in Section 2 identified one study of instrumented physiological outcomes in this group: in a chamber study, people with building-related symptoms and referents did not differ in autonomic responses to low-level acrolein [78].

Two longitudinal studies examined change after exposure was reduced. Over seven years of remediation attempts in a water-damaged office building with 1,175 occupants, the odds of mild non-respiratory symptoms such as headache, excessive fatigue, and fever or chills declined, while severe non-respiratory symptoms did not change and severe respiratory symptoms worsened [44]. In an uncontrolled follow-up of 45 school staff, symptoms and spirometry improved within three to six months of moving out of a moisture-damaged building [79]; regression to the mean and expectation effects cannot be excluded in that design.

Uncontrolled clinical series provide longer follow-up, with the selection and comparison-group limitations that apply to all such series. Outbreaks of severe fatigue with neurologic complaints resembling chronic fatigue syndrome were described among teachers and office workers sharing specific rooms or buildings, and at four-year follow-up, improvement in fatigue tracked improvement in nasal, sinus, and headache symptoms [80,81]. In a Swedish follow-up of 239 patients referred with building-related symptoms, nearly half reported largely unchanged symptoms after seven years or more, fatigue was the most common weekly symptom, and a delay of more than a year from onset to first clinic visit predicted loss of work capacity [82].

3.2.4. Neurocognitive Outcomes in Adults

The human neuropsychological literature consists mainly of case series from 2002 to 2004 and later work by Kilburn. A study of 182 patients with a history of mixed mold exposure reported psychological, neuropsychological, and electroencephalographic abnormalities with an internal dose-response pattern but no comparison group [83]. A study comparing 31 mold-exposed patients with people with traumatic brain injury and controls reported memory and executive dysfunction

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[84]. Kilburn compared 105 referred adults with 202 community referents [85]; the 2024 mental health review classed his studies as case-control designs with self-selected symptomatic cases [53]. These studies drew on referred patients, and their designs (self-selected or referred cases and, in some studies, no comparison group) limit what they can show: they can neither establish nor exclude an effect of exposure on cognition. A clinical article from 2009 set out the neurologic and neuropsychiatric features its author considered characteristic of exposure to mold and mycotoxins, observing that they can resemble recognized conditions including pain syndromes, movement disorders, delirium, dementia, and disorders of balance and coordination, and argued that separating them neurodiagnostically from those conditions is a precondition for studying them properly [86]. The article reports neither a comparison group nor systematic case ascertainment, so it describes a proposed syndrome rather than testing one; its argument for formal differential diagnosis is the same consideration developed in Section 3.6.

Controlled work since then is limited. A 2026 Colombian case-control study of 60 older adults with amnestic mild cognitive impairment and 60 controls matched for sex, age, and neighborhood found no association with a structured home inspection score (OR 0.99, 0.91–1.08), while self-reported nasal irritation at home was associated with mild cognitive impairment (OR 5.84, 1.20–28.37), an imprecise estimate based on self-report [87]. The study's outcome was amnestic mild cognitive impairment in participants with a median age of 69 [87], not the difficulty remembering, confusion, and dizziness recorded in the symptom surveys in Section 3.2.3. By contrast, among 11,888 Chinese adults with a median age of 82, self-reported musty odor was associated with cognitive impairment on the administered Mini-Mental State Examination (OR 1.40, 1.25–1.57) [88]; in this cross-sectional design, reverse causation, in which cognitive decline reduces the ability to maintain a home, cannot be excluded. The two studies differed in exposure assessment (structured home inspection versus self-reported musty odor), in outcome (amnestic mild cognitive impairment versus score on an administered Mini-Mental State Examination), and in population (median age 69 in Colombia versus median age 82 in China) [87,88]. The only brain-imaging study identified compared volumetric MRI in 17 clinic patients and 18 controls and reported caudate atrophy and pallidal enlargement [89]; it came from the group that proposed CIRS, compared many brain regions in a small sample, and no independent replication was identified. A 2017 narrative review from clinical immunology concluded that there was no scientific evidence that exposure to visible mold causes memory loss, inability to focus, fatigue, or headache [90]; it predates most of the studies summarized in this section. No epidemiological study was identified of incident dementia, measured olfactory function, or peripheral neuropathy in relation to damp or water-damaged buildings. No study was identified that combined objective exposure assessment, a population-based comparison group, and exposure-blinded neuropsychological testing in adults; the closest are inspector-scored exposure with self-reported cognitive symptoms [71] and self-reported exposure with an administered cognitive screen [88].

3.2.5. Child Neurodevelopment

Several birth cohorts have examined early-life dampness and child neurodevelopment. In Krakow, visible mold recorded at intervals for more than two years of early life was associated with an adjusted IQ deficit of 9.2 points (95% CI 3.1 to 15.2) at age 6 among 277 children of non-smoking mothers [91]; the confidence interval spans 3.1 to 15.2 points and the estimate has not been replicated. In a Spanish cohort, persistent early-life dampness was associated with a 4.9-point lower general cognitive score at age 4, but endotoxin and fungal extracellular polysaccharides measured in house dust were not associated with test scores [92]. In two German cohorts, mold and dampness were associated with emotional symptoms on the parent-reported Strengths and Difficulties Questionnaire at age 10 (abnormal total difficulties with dampness, adjusted OR 1.51, 1.10–2.07) but not with the hyperactivity/inattention subscale [93], and in the UK Millennium Cohort, damp or condensation at 9 months was associated with emotional and conduct problems, but not hyperactivity, at age 3 [94]. No study was identified that used diagnosed attention-deficit/hyperactivity disorder as an outcome.

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Reports linking dampness or condensation to autism-spectrum outcomes come from a survey analysis of 72 parent-reported cases, in which the authors interpreted window condensation as a proxy for a low ventilation rate in the home [95], and from a birth cohort in which dampness or mold at age 2 was one of many exposures associated with autism-spectrum symptoms, rather than diagnosis, at that age [96]. A 2026 PROSPERO-registered systematic review of 25 studies in high-income countries graded the evidence linking mold, damp, and condensation, together with other substandard housing conditions, to poorer early child development as of moderate certainty, while rating every included study at high or very high risk of bias [97]. Composite housing measures that combine mold with pests and peeling paint, a marker of lead hazard, show similar associations but cannot isolate mold [98].

3.2.6. Skin, Eye, Reproductive Outcomes, and Cancer

Skin outcomes show the same contrast between reported and examined findings that appears for respiratory outcomes (Section 3.5.4). In ISAAC Phase 2, reported in a meeting abstract, current dampness or mold among 46,051 children in 20 countries was associated with parent-reported flexural eczema (adjusted OR 1.96 in non-affluent and 1.34 in affluent countries) but not with eczema found on examination (OR 0.93, 0.76–1.13) [99]. Visible mold was associated with reported atopic dermatitis among 17,881 northern European offspring aged 30 or younger (OR 1.35, 1.12–1.62) [100]. For the eye, fungal DNA in classroom dust was associated with shorter tear-film break-up time, measured instrumentally as well as by self-assessment, among 368 Malaysian students [101].

Evidence on birth outcomes is sparse and inconsistent. A British cohort reported associations of retrospectively reported serious mold problems with low birthweight (OR 1.98, 1.13–3.47) and small-for-gestational-age birth [102], and a Chinese study reported an association of perinatal mold or damp stains with parent-reported preterm birth (OR 1.73, 1.04–2.90) [103], while a Shanghai study of 13,335 children found no association between household dampness and preterm birth, low birthweight, or small-for-gestational-age birth [104]. All three relied on retrospective report. A retrospective case series from one clinical practice reported complications in 19 of 21 pregnancies among eight women from seven homes, most with documented macrocyclic trichothecene contamination, but it had no comparison group, selected patients by clinical presentation, and used a commercial ELISA that detects these toxins only as a group [105]; it can generate hypotheses but not estimate risk.

WHO noted that the IOM had found no evidence that cancer or reproductive outcomes were altered by exposure to indoor air and that a search of the preceding five years found no studies on these subjects [2, p. 84], and it stated that there is no epidemiological evidence for an association between exposure in damp buildings and cancer [2, p. 88]. The supplementary search for this review identified no epidemiological study of residential dampness or mold and cancer.

3.2.7. Gastrointestinal, Hepatic, Metabolic, and Renal Outcomes, and the Microbiome

Gastrointestinal complaints appear in this literature mainly as items within broad symptom surveys [73]. The IOM and the AWMF guideline place these outcomes in the inadequate category [4,8]. The supplementary search identified no epidemiological study of incident diabetes, adiposity or glycemic measures, liver disease, or kidney disease in relation to building dampness or mold, including in large cohorts in which dampness has been studied for other outcomes. The human literature on hepatic, renal, and metabolic effects of mycotoxins concerns ingested exposure: a 2026 systematic review of 268 studies found, on meta-analysis, associations of aflatoxin exposure with hepatocellular carcinoma, cirrhosis, elevated alanine aminotransferase, and gallbladder cancer, and graded the certainty of evidence as very low for all 21 outcomes assessed [106]. A systematic review of occupational aflatoxin exposure in grain, milling, baking, and feed work, where exposure occurs by inhalation of organic dust and by dermal contact as well as by ingestion, reports elevated hepatic and renal enzymes [107]; a review of airborne mycotoxin measurements found the highest exposures in agricultural work such as animal feeding and harvester cleaning [108].

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No study was identified that characterized the gut microbiome of occupants of damp or water-damaged buildings. The nearest human evidence concerns the upper airway. In 58 nasal swab samples from infants in San Juan, nasal mycobiome composition differed between those born in the first and second years after Hurricane Maria (beta diversity $p < 0.01$; *Alternaria* prevalence 42% versus 9%), although alpha diversity did not differ, exposure was defined only by birth period, no home fungal measurements were made, and respiratory outcomes were not followed [109]. In a pilot study of 56 children in Shanghai, nasal and oral microbiota composition was associated with two mycotoxins detected in indoor dust, deoxynivalenol and nivalenol, and with tobacco smoke exposure [110]; the alimentary route is the main source of internal exposure to these compounds [111]. Animal studies show that inhaled or instilled microbial material can alter gut communities, under conditions unlike residential exposure. Intratracheal *Aspergillus fumigatus* produced fecal dysbiosis in one rat strain and intestinal inflammation without a change in fecal composition in another, with no rise in plasma cytokines [112]; in immunosuppressed mice, *A. fumigatus* infection enriched the lung microbiome for anaerobic bacteria, and the authors propose that the gut microbiome influences the lung through the plasma metabolome, a gut-to-lung direction rather than a fungal effect on the gut [113]; and intranasal endotoxin-rich swine-barn dust extract reduced some gut commensal species in mice without robustly altering overall community structure [114]. Two of these are fungal infection models, one of them in immunosuppressed mice, and the third delivered a concentrated dust extract intranasally; none permits a dose comparison with residential inhalation.

3.2.8. Immune, Inflammatory, and Autoimmune Outcomes

Direct measurements of immune markers in exposed people are few and point in different directions. In a Finnish birth cohort with engineer home inspections, moisture damage in the main living areas was associated cross-sectionally with higher C-reactive protein and greater stimulated pro-inflammatory cytokine production at age 6 [115], but in the same cohort, damage inspected in infancy did not predict C-reactive protein or leukocyte counts at age 6 [116]. Small studies have reported higher monocyte toll-like receptor and dectin-1 expression in occupants of damp homes [117] and higher mold-reactive T-cell frequencies with greater reported exposure [118]; these are markers of exposure or immune engagement rather than of disease. In 99 patients referred to secondary care with symptoms that they associated with workplace moisture damage, C-reactive protein, sedimentation rate, total IgE, exhaled nitric oxide, and skin-prick results were not higher than in 48 controls, although elevated blood neutrophils were more common among those with asthma (19% versus 2% of patients without asthma) [119]. A transcriptomic study found airway and systemic inflammatory signatures in adult-onset asthma unrelated to dampness but not in dampness-related asthma [120]. No study was identified that measured mast cell mediators, such as tryptase, histamine, or prostaglandin D2 metabolites, in people exposed to damp buildings, so this pathway is untested in this population rather than excluded. A review of mold, mycotoxins, and immune function concluded that the literature gives a heterogeneous picture, that allergic responses to molds are generally accepted while other direct effects remain disputed, and that exposure may matter most in people with pre-existing dysregulation of the immune system, including allergic and non-allergic chronic inflammatory disease and autoimmune disorders [121].

Evidence on autoimmune disease comes mainly from clusters. Finnish investigators described inflammatory rheumatic disease in a moisture-damaged office (incidence density ratio 6.8, 95% CI 3.6–13.0) and joint disease in a health center [122,123]; the AWMF guideline does not regard such findings as sufficient evidence while studies from other centers are lacking [8]. No study since 2015 was identified that measured autoantibodies or incident autoimmune disease in a defined exposed population. The evidence on sarcoidosis comes mainly from occupational case-control studies and two meta-analyses of them. In the 706-pair ACCESS study, work in areas with musty odors was associated with sarcoidosis (adjusted OR 1.62, 1.24–2.11) [124], a cluster occurred in a historically water-damaged office building [125], and a 2026 meta-analysis whose quantitative synthesis covered 13 studies in all found occupational mold or mildew exposure associated with pulmonary sarcoidosis

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(pooled OR 1.59, 1.33–1.90; I^2 = 0%), consistent in direction with an earlier meta-analysis [126,127]. The studies its supplementary material lists for the mold and mildew exposure include the ACCESS study described above, so that pooled estimate and the ACCESS result are not independent of one another [126]. Exposure in these studies was assessed by questionnaire rather than by measurement [124,126,127].

3.2.9. Endocrine, Cardiovascular, and Autonomic Outcomes

The supplementary search identified no epidemiological study of thyroid function, cortisol or other hypothalamic–pituitary–adrenal measures, sex hormones or menstrual function, blood pressure, heart rate variability, cardiovascular disease, or postural orthostatic tachycardia syndrome in relation to dampness or mold exposure. The AWMF guideline rates thyroid disease as inadequate and notes the absence of systematic studies or case reports establishing a link with thyroid disease or other endocrinopathies [8]. The available material consists of a nine-patient uncontrolled treatment series [128] and the chamber study of autonomic responses described above [78]. Thyroid disease and POTS share symptoms with those reported in this literature and are discussed in Section 3.6.

3.2.10. Animal and Laboratory Evidence

Animal studies indicate that effects beyond the airway are biologically possible, at doses whose relation to human indoor exposure is unknown. Intranasal satratoxin G caused olfactory sensory neuron loss and inflammation extending to the olfactory bulb in mice, with a no-effect level of 5 μg/kg and a lowest-effect level of 25 μg/kg at 24 hours [129]. In the same model, satratoxin G at 100 μg/kg by intranasal instillation produced olfactory sensory neuron apoptosis and atrophy of the olfactory epithelium, while its biosynthetic precursor roridin L2 at the equivalent dose did not; in cultured PC-12 neuronal cells satratoxin G was cytotoxic at 10 to 25 ng/ml, whereas roridin L2 was without effect at concentrations up to 1000 ng/ml [130]. Following intranasal instillation of 500 μg/kg in mice, satratoxin G was cleared from plasma with a half-life of 20 minutes and reached its highest tissue concentrations 15 to 30 minutes after dosing, in the nasal turbinates (480 ng/g), kidney (280 ng/g), and lung (250 ng/g), with 14 ng/g in the olfactory bulb and 3 ng/g in the brain; the half-life in the nasal turbinate was 7.6 hours, and cumulative excretion in feces and urine over five days accounted for less than 0.3 percent of the administered dose [131]. In rhesus macaques, selected by the investigators because their nasal airways more closely resemble those of humans, a single intranasal instillation of 20 μg of satratoxin G into one nasal passage, or 5 μg daily for four days, produced acute rhinitis, atrophy of the olfactory epithelium, and olfactory sensory neuron apoptosis; the single higher dose reduced olfactory sensory neuron volume density by 13 percent and the repeated lower dose by 66 percent, with 14-fold and 24-fold increases in apoptotic cells of the olfactory epithelium respectively, in three animals per group [132]. Repeated intranasal exposure to intact *Stachybotrys* spores or to toxin-extracted spores reduced hippocampal neurogenesis and impaired contextual memory in mice [133]. In a follow-up study in male mice, toxin-extracted spores impaired long-term spatial memory in the Morris water maze, and hippocampal interleukin-1β immunoreactivity correlated with performance during training [134]. Together these findings suggest that, in mice, innate immune activation by fungal cell-wall material, and not only mycotoxins, can produce such effects, although the authors note that their purified spore preparations do not represent typical human exposure [134]. Nose-only inhalation of viable *Aspergillus versicolor* spores for up to four weeks increased pro-inflammatory and glial gene expression in several mouse brain regions [135], whereas 96 hours of continuous inhalation of ultrafine *Alternaria* particles caused lung inflammation without detectable brain inflammation and reduced innate immune markers in the brainstem [136]. The fungal volatile 1-octen-3-ol, a contributor to musty odor, reduced dopamine and caused dopaminergic neuron loss in *Drosophila* [137] and worsened depressive-like behavior in a rat depression model [138]; these studies used invertebrate or disease-primed models, and their relevance to residential exposure has not been established. In inhalation work by NIOSH, repeated exposure to viable *S. chartarum* conidia caused pulmonary arterial remodeling, which progressed faster with the strain that produced more

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respirable fragments [139]. In the National Toxicology Program's three-month study, in which mice inhaled viable conidia, heat-inactivated conidia, or filtered air, the target organs identified after viable conidia were the larynx, the lung, and, to a lesser extent, the bronchial lymph nodes, and the histopathological changes seen after viable conidia were not seen in the heat-inactivated particle control or the air control [140]. Spore preparations from a satratoxin-producing strain and from a strain that does not produce satratoxin caused comparable inflammatory cell recruitment in mouse lungs, indicating that the toxin was not required for that response [141]. Sterigmatocystin and 5-methoxysterigmatocystin, produced by *Aspergillus* series *Versicolores* and both detected in dust from damp indoor occupational and living areas, were instilled intratracheally in rats at doses the investigators selected to correspond to the concentrations measured in that dust; at 24 hours neither toxin alone nor the two combined significantly altered lactate dehydrogenase, albumin, interleukin-6, tumor necrosis factor-α, or macrophage inflammatory protein-1α in bronchoalveolar lavage fluid, while both produced DNA damage of similar intensity in an alkaline comet assay, only 5-methoxysterigmatocystin produced significant damage in a neutral comet assay, and the combination produced less DNA damage than either toxin alone [142]. Reviews of this literature describe inhibition of protein synthesis and activation of cellular stress responses as the best-characterized actions of macrocyclic trichothecenes, note that other constituents of the fungus, including atranones and cell-wall material, also provoke inflammation, and identify chronic low-level inhalation and the measurement of human exposure as the principal unresolved questions [143,144]. One review separates evidence of lung injury in animal models from evidence of established fungal infection and revisits the reports of acute pulmonary hemorrhage in infants, for which a causal link remains unresolved [144]. A review of inhaled toxicants situates these findings among the inflammatory pathways shared with other inhaled agents [145], and a review of neurological effects describes oxidative stress, inflammation, and the olfactory route as candidate mechanisms without establishing that indoor exposure causes a specific neurological disorder in people [146]. A 2026 review of occupational settings compiles airborne measurements from agriculture and food processing and notes the scarcity of human outcome data and the absence of health-based occupational limits specific to airborne mycotoxins [147].

Two features of this body of work limit what can be inferred from it for building occupants. Material is generally delivered directly to the nose or trachea, or as concentrated aerosols generated under laboratory conditions, rather than breathed from the air of an occupied building; and the preparations are purified compounds or cultivated spore suspensions rather than the mixture that accompanies water damage indoors. One study set its doses by reference to concentrations measured in dust from damp indoor areas [142], although it too delivered them by direct instillation. Acute respiratory illness following inhalation of large quantities of mold-laden agricultural dust has been described in people [148], but that setting differs substantially from residential conditions and the responsible dust constituent was not identified. The IOM noted that toxicologic studies in animal and cellular models cannot by themselves support conclusions about human health effects and that the doses required to cause effects in humans have not been determined [4, p. 7].

3.2.11. The CIRS Case-Series Literature

A distinct clinical literature describes a "chronic inflammatory response syndrome" (CIRS) associated with water-damaged buildings. Its foundation is a time-series study in which 28 patients were enrolled and 13 entered a double-blind arm (7 cholestyramine, 6 placebo), reporting symptom reduction with cholestyramine, recurrence on re-exposure, and improvement in visual contrast sensitivity [149]. A 2024 review of CIRS treatment identified 13 articles referencing treatment for CIRS, of which 11 described the Shoemaker Protocol; two of its authors disclose providing expert witness testimony in CIRS cases for both plaintiffs and defense [150]. The main methodological concerns are that most data come from one group; samples are small; the foundational study interpreted improvement on cholestyramine as support for an exposure-related cause [149], although cholestyramine also binds bile acids (Section 3.6.1); visual contrast sensitivity and the proposed

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biomarkers are not specific to water-damaged-building exposure; and there are no multicenter blinded trials. The AWMF guideline lists the visual contrast sensitivity test among diagnostic methods without sufficient scientific evidence and cholestyramine "detoxification" among treatments without sufficient evidence [8, tables 11 and 12]. A 2026 comparison of 28 people described by the investigators as biotoxin-exposed, with clinical signs of biotoxicity, and 30 controls found that the handheld visual contrast sensitivity chart did not distinguish between the groups, while digital contrast tests did [151].

3.2.12. Laboratory Tests for Mycotoxins in People

Two kinds of laboratory test are offered to people concerned about mycotoxin exposure, and they face different problems. Urinary tests aim to detect the compounds themselves or their metabolites, and the central difficulty is attributing a detected compound to a building rather than to diet or another source. Serum antibody tests aim to detect an immune response to mycotoxins, and the central difficulty is whether the assay reflects a response to the toxin at all.

Urinary methods also differ from one another. Mass spectrometry identifies individual compounds and underlies validated population biomonitoring [152], whereas ELISA infers a compound from antibody binding. A 2013 study found at least one mycotoxin by ELISA in 93% of 112 patients with chronic fatigue syndrome, compared with no positive results among 55 people with no reported history of water-damaged-building exposure, tested previously by the same laboratory using the same methods [153]. A published comment noted that the controls were selected for having no history of exposure to water-damaged buildings, a selection method that, the comment argued, undermines the comparison between groups [154]. A later series of 236 patients with ME/CFS who reported mold exposure found urinary evidence of mycotoxin exposure in 92.4%, without a control group [155]. The immunoassay methods underlying this testing — competitive ELISA for trichothecenes and immunoaffinity columns with fluorometry for aflatoxins and ochratoxin A — were described earlier by authors affiliated with the commercial laboratory offering the test, who detected mycotoxins in specimens from mold-exposed patients but not from comparison patients with no known exposure, whose selection was not described [156]. In a NIOSH evaluation prompted by an office worker's positive urine results from a direct-to-consumer laboratory, destructive inspection of the office found no water damage or significant fungal growth; CDC noted that mycotoxins occur in the urine of healthy people, that no disease-predictive levels have been established, and that no such test is approved by the Food and Drug Administration, and it does not recommend biological testing of people in water-damaged buildings [157]. General-population biomonitoring shows routine dietary exposure; in the European HBM4EU aligned studies, total deoxynivalenol was measured in the urine of 1,270 adults from six countries, median concentrations ranged from 0.41 to 10.16 µg/L by country, and 12.3% exceeded the health-based guidance value [152]. A single-case investigation prompted by an elevated urinary result in an atopic child found no fungal source in his home, culturable fungi only in a previously water-damaged, since-repaired classroom, and normal results in household members and a classmate; the authors concluded that mold in the school could not be ruled out as the source of the child's elevated result and that the thresholds at which effects occur may be lower in sensitive individuals [158]. Apart from a pilot study in which LC-MS/MS detected mycophenolic acid in repeated urine samples from two symptomatic workers in one building, neither of whom took immunosuppressive medication and whose urinary profiles differed from those of household and unrelated controls [159], no peer-reviewed study was identified that compared urinary mycotoxins by mass spectrometry between symptomatic people and controls drawn from the same population. For urinary testing by mass spectrometry, CDC states that no mycotoxin levels predicting disease have been established [157], and the American College of Medical Toxicology states that it is inappropriate to attribute mycotoxins identified in urine to postulated remote inhalation exposure [11, p. 3]. A separate question is whether material entering the body by inhalation appears in urine in measurable amounts. In the mouse study described in Section 3.2.10, cumulative excretion of satratoxin G in feces and urine over five days accounted for

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less than 0.3 percent of an intranasally administered dose [131]. That result comes from one compound, one species, and a single instilled dose, and satratoxin G is not among the compounds that these panels principally report, so it does not establish what urinary recovery would be after residential exposure. The published studies are further limited by control selection and dietary background; they show neither that people lack the compounds nor that compounds detected came from a building.

Serum IgG tests marketed for mycotoxins raise a separate question, about what the assay detects. Mycotoxins are low-molecular-weight compounds that act as haptens: unless they become covalently bound to a carrier protein, they are not expected to provoke a class-switched IgG response, so it is unclear what a positive result reflects [160]. The authors of that analysis, who work within the CIRS framework, describe both mycotoxin antibody testing and urinary ELISA as resting on flawed science and state that no study of exposure to mycotoxins alone has withstood peer review [160]. The American College of Medical Toxicology likewise does not accept mycotoxin antibody testing as a method of exposure assessment [11, p. 3]. No controlled study was identified that related serum antibody results to measured building exposure or to clinical outcomes. Protein adducts are a different kind of marker, since they detect the toxin bound to a host protein rather than an antibody against it: satratoxin G–albumin adducts were reported in three exposed patients and not in controls, but the finding has not been replicated [161].

3.3. Exposure Assessment

A 2011 review of this literature reported consistent associations for observed dampness or mold — visible dampness, visible mold, water damage, and musty odor — and only limited, partly equivocal associations for measured microbiologic agents in dust [5]. The NIOSH Dampness and Mold Assessment Tool scores these four factors semi-quantitatively without environmental sampling; its scores have shown exposure-response patterns with respiratory symptoms in schools, a hospital, and a college, but when three teams independently assessed the same rooms in the study that led to the tool's development, concordance between them was 100% for moisture and 88% for water stains, against 63% for visible mold and 75% for mold odor, and its developers note that the assessment is somewhat subjective and can vary from person to person [162]. The same authors write that there are no standard or consensus methods for quantifying microbial agents and that associations between quantitative microbial measurements and respiratory health have been inconsistent [162]. Measured moisture correlates with observational indicators, most strongly with musty odor and visible mold [163].

Studies of quantitative microbial measures have reported associations in inconsistent directions. A 2011 review concluded that current evidence did not support measuring specific indoor microbial factors to guide health-protective actions [5]. In German birth cohorts, extracellular polysaccharides and endotoxin were inversely associated with asthma [164], but across three European birth cohorts, early-life endotoxin was positively associated with asthma in one, inversely in another, and not in the third, and glucans and extracellular polysaccharides were not significantly associated with asthma in any [165]. In a Swedish case-control study, culturable fungi, glucan, and ergosterol in bedroom dust were associated neither with childhood asthma, rhinitis, or eczema nor with parent- or inspector-reported dampness [166]. The Environmental Relative Moldiness Index (ERMI), a dust qPCR panel of 36 fungal species developed at the EPA [167], has been linked to childhood asthma in a Cincinnati cohort [168], and its developer's 2025 review describes consistent associations with asthma outcomes [169]. ERMI values are reasonably repeatable over months in the same home, but bedroom and living-room values correlated at r = 0.69 and vacuum-bag dust at r = 0.58 with floor dust, which the authors describe as supporting use of the index for exposure classification [170]; values in 40 French dwellings ranged from -2.7 to 28.8 [171], above the upper bound of 20 on the US scale as the index developer describes it [169]. The evaluations located were authored or coauthored by the index developer. In 2013 the EPA Office of Inspector General reported that the EPA "readily acknowledged" that ERMI and the underlying mold-specific qPCR method had not been validated

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or peer reviewed by the agency for public use, and that the EPA considered both to be "research tools not intended for public use" [172] (p. 4). No PubMed-indexed validation of the HERTSMI-2 index was found.

Mycotoxins and other microbial secondary metabolites are present in water-damaged buildings. Mass spectrometry detected at least one target mycotoxin in 66% of building-material samples from Swedish water-damaged buildings [173]. The exposure is a mixture: every one of 69 samples from severely moisture-damaged Finnish homes contained at least one of 186 targeted fungal or bacterial metabolites [174]. In schools in three European countries, 42% to 58% of dust and swab samples contained at least one metabolite but most individual compounds occurred in 10% or fewer, and moisture-damaged schools had more metabolites at higher levels than reference schools, significantly so only when the countries were pooled [175]; after a flood in Croatia, airborne *Aspergillus* section *Versicolores* levels were three times as high in the flooded village as in a control village, while dustborne isolates were equally present in both; sterigmatocystin and 5-methoxysterigmatocystin were among 75 metabolites detected in house dust [176]. In air, ELISA detected macrocyclic trichothecenes at concentrations from below 10 to above 1,300 pg/m³ in contaminated buildings, higher than in controls and rising with air disturbance [177], and LC-MS/MS measured satratoxins G and H at 0.25 and 0.43 ng/m³ in one water-damaged dwelling [178]. Validated mass spectrometry methods can now quantify airborne mycotoxins in mold-infested workplaces, although very low sampled masses and trace concentrations remain the main obstacles [179]. Screening of airborne *Penicillium* and *Aspergillus* isolates from occupied rooms at a zoological garden combined species identification, mycotoxin analysis by liquid chromatography–mass spectrometry, and a cytotoxicity assay, an approach that characterizes what airborne isolates from an occupied building can produce, although it does not measure what occupants receive [180]. Exposure assessment for these metabolites is described as challenging [181]; an overview concluded that diet is the main source of human mycotoxin exposure, although residential and occupational pathways should also be considered [111], and no study was identified that apportioned an occupant's intake between building and diet. Detection of these compounds in building materials, dust, and air does not, however, establish an inhaled dose. The IOM stated that there are no generally accepted health-based standards for fungal spores, hyphae, or metabolites, and that the number of samples needed for an accurate assessment of risk-relevant exposure cannot be specified [4, p. 12]. WHO concluded that relationships between dampness, microbial exposure, and health could not be quantified precisely enough to recommend guideline values [2, p. 95], and a 2018 review reached the same conclusion about health-based limits [34]. No validated measure of personal dose for specific agents was identified, and the assessments cited above report that health-based values could not be derived from the available exposure data [2,4,34].

3.4. Mechanisms

The mechanisms can be grouped by strength of human evidence. Established mechanisms include IgE-mediated allergy and asthma, hypersensitivity pneumonitis, and infection in immunocompromised people [2,4,8]. A mechanism with partial human support is inflammation arising from non-allergic innate immune activation by fungal fragments, glucans, and endotoxin: associations appear in nonallergic as well as allergic people [5,28,182], and animal inhalation studies implicate fungal fragments and show shifts in immune response with repeated exposure [139,183]. In vitro, low doses of valinomycin and other microbial toxins combined with β-glucan produced synergistic proinflammatory responses in human macrophages, while some combinations with endotoxin were inhibitory [184], and dust from moisture-damaged schools was more inflammatory than dust from reference schools in two of three countries, with muramic acid, a Gram-positive bacterial marker, the strongest predictor [185]. WHO likewise noted that both allergic and non-allergic mechanisms may be involved and that adverse effects occur in nonatopic populations [2]. Direct measurements of immune markers in exposed people are few and mixed (Section 3.2.8).

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The toxicology of mycotoxins as compounds is well characterized, with described effects on protein synthesis, oxidative stress, and immune function, although that literature rests largely on dietary and occupational exposure rather than on the indoor environment [186]. Mycotoxin-driven systemic toxicity at realistic indoor inhaled doses remains contested. WHO described the evidence that mycotoxins play a role in indoor-air health problems as extremely weak and noted that no study had shown that people in damp buildings who report nervous system symptoms are exposed to effective levels of mycotoxins [2, p. 82]. Two dose-modeling papers from overlapping authors, whose corresponding affiliations were the consulting firms GlobalTox and Veritox, concluded that toxic doses are implausible by indoor inhalation: the first modeled a maximum inhalable 24-hour dose from mold spores and compared it with effects data [187], and the second compared reported airborne mycotoxin levels with a generic concentration of no toxicologic concern of 30 ng/m^3 [188]; members of the same group wrote the 2003 American College of Occupational and Environmental Medicine position statement on molds [189]. The first model is built on inhalation of intact spores, and both address toxic thresholds rather than chronic effects; neither models chronic low-dose immune effects, mixtures, or toxin carried on particles smaller than spores, which has been detected in laboratory air sampling over contaminated building material [190]. The field studies measured concentrations in building air and materials; the dose models estimated inhaled doses against toxic thresholds. Neither addressed chronic low-dose immune effects. A 2025 position statement of the American College of Medical Toxicology concluded that no diagnostic laboratory or treatment methods grounded in medical toxicology support systemic mycotoxin toxicity from mold-related inhalation exposures [11, p. 5]; like the 2003 ACOEM statement, it is a professional society position rather than a systematic evidence review. Earlier statements differ in emphasis: a 2006 report of the American Academy of Pediatrics called the health effects of inhaled mold toxins, beyond allergy and infection, controversial and in need of further investigation [191]; a 2006 position paper of the American Academy of Allergy, Asthma and Immunology, one of whose authors also coauthored the 2003 ACOEM statement, regarded many proposed mold-related illnesses as largely or completely unproven [192]; and the French agency ANSES rated the evidence for health effects of indoor mycotoxins as very low and their inhaled toxicity as controversial [[193], p. 67]. Olfactory-limbic, neuroinflammatory, and central sensitization pathways rest mainly on animal and hypothesis-level evidence, in which inhalation studies have reported both activation and suppression of brain innate immune markers [129,133,135,136]. A proposed “dampness and mold hypersensitivity syndrome” includes five clinical criteria but, by its author’s account, no accepted laboratory tests [194].

3.5. Confounding, Exposure Components, and Bias

This section separates four kinds of process that can affect the observed associations and that are often grouped together as confounding. Socioeconomic and housing conditions that influence both dampness and health are confounders in the strict sense (Section 3.5.1). Agents produced by or accompanying water damage, such as endotoxin, mites, and chemical emissions from damp materials, are components of the exposure or mediators of its effects, so adjusting for them would remove part of what the exposure does; household exposures that are not products of water damage, such as gas cooking, are potential confounders (Sections 3.5.2 and 3.5.3). Reporting bias, misclassification of exposure or outcome, and selection are forms of bias rather than confounding (Sections 3.5.4 and 3.5.5). Co-occurring clinical conditions (Section 3.6) mainly raise questions of outcome misclassification, differential diagnosis, and the interpretation of treatment response; they are confounders of the exposure–outcome association only where they are associated with exposure, and conditions that the exposure might itself cause would be mediators rather than confounders.

3.5.1. Confounding by Socioeconomic and Housing Conditions

Where crude and adjusted estimates can be compared, adjustment for deprivation changes the associations only modestly. In Edinburgh children, adjusting the mold–wheeze association for tenure, crowding, smokers, and gas cooking moved the odds ratio from 3.70 to 3.00 [195]; with

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surveyor-confirmed dampness and physician-diagnosed asthma, adjustment slightly increased the estimate from 2.62 to 3.03 [196]; and restricting a meta-analysis of respiratory infections to studies controlling for age, sex, smoking, and socioeconomic status changed findings only slightly [17]. Associations persisted across 20 countries in both affluent and non-affluent settings and among atopic and non-atopic children (wheeze OR 1.58, 1.40–1.79) [182] and across 12 pooled studies of more than 58,000 children [197]. The WHO and Mendell reviews already required control for sex, smoking, and socioeconomic status for inclusion, so their pooled evidence is adjusted by construction [2,5], although adjustment for coarsely measured socioeconomic status can leave residual confounding (Section 3.5.5).

Cold and fuel poverty have been examined in fewer studies than the other factors in this section, and the available studies do not separate their effects from those of dampness. Inadequate heating predicted mold contamination in UK social housing (OR 3.4, 2.0–5.8) [198], only social tenants showed raised asthma despite mold being common in private rentals [199], and lower bedroom temperature over the preceding days was associated with small short-term reductions in lung function in children with asthma [200]. These findings place cold both upstream of dampness and alongside it as a respiratory risk, so adjustment for heating removes part of the dampness effect if heating acts partly by reducing dampness.

3.5.2. Components of the Exposure Mixture and Accompanying Household Exposures

Water-damaged buildings carry a mixture of agents, several of which are independently associated with the same respiratory outcomes. House dust mite allergen rises with humidity [201]; endotoxin has sufficient evidence of association with asthma exacerbation in preschool children, and nitrogen dioxide, rodent and cockroach allergen, and dust mite have limited or suggestive evidence [6]; gas cooking, which is not a product of water damage, is associated with childhood asthma (OR 1.32) [202] and is a potential confounder to the extent that it is more common in damp housing; and degradation of PVC flooring on damp concrete releases 2-ethyl-1-hexanol, which was associated with asthma symptoms in hospital staff (adjusted OR 8.6) [203]. 2-Ethyl-1-hexanol arises indoors mainly from hydrolysis of plasticizers and flooring adhesives on moist, alkaline concrete rather than from the flooring materials themselves [204]. Removing degraded flooring from a Finnish office reduced volatile compounds, symptoms, and medication need [205], and plastic wall materials and recent floor-leveling compounds predicted adult-onset asthma [206].

Studies that measured or adjusted for individual components have not removed the dampness association. Mite sensitization was more common in damp homes but did not explain wheeze, and the authors concluded that the link was principally non-atopic [182,207]. In a New Zealand case-control study with assessor, researcher, and parent assessment, visible mold and mold odor were dose-dependently associated with new wheeze regardless of atopic status, while qPCR microbial levels, temperature, and humidity were not [208]. Associations were comparable for atopic and non-atopic asthma in an engineer-inspected Finnish study [209], and fitted carpets, a mite reservoir, were associated with fewer symptoms while reported mold was associated with more [210]. In Finnish homes, 333 measured microbial metabolites did not explain the moisture-damage association with asthma [211], and in Danish classrooms, inspector-assessed dampness predicted lower lung function and wheeze while seven measured microbial components did not [212]. WHO observed that unless many samples are taken per house, sampling of culturable organisms will probably give a poor quantitative measure of exposure, leading to a nonspecific bias towards the null [2, p. 29], and that quantitative measurement is not inherently more accurate if the microbes measured are not the causal factors [2, p. 66].

Few studies have related measured indoor mycotoxins, rather than dampness or mold, to health outcomes. In the Finnish homes study, higher metabolite loads in floor dust were associated with less active asthma at age 6 [211]. Two reports from one study of 462 pupils in Malaysian schools found the *Stachybotrys* marker verrucarol in classroom dust inversely associated with daytime breathlessness [213] and positively associated with weekly tiredness ($p = 0.04$, among many

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comparisons) [214]. In an untargeted case-control analysis, homes of Shanghai children with asthma or allergic rhinitis were enriched in mycotoxins mainly of *Fusarium* and *Alternaria* origin [215]. These studies are too few and inconsistent to support or exclude an association with any specific outcome.

3.5.3. Agents Other than Fungi

Endotoxin, from Gram-negative bacteria, is the non-fungal agent with the largest body of epidemiological studies located by this review, and its associations differ in direction by age and setting. In adults, house-dust endotoxin was associated with diagnosed asthma, asthma symptoms, and wheeze in a nationally representative US housing survey, regardless of allergic status [216], with wheeze and asthma medication use in a larger national survey [217], and with atopic and nonatopic asthma in a farming population [218]. In children, a meta-analysis found endotoxin positively associated with wheeze in infants and toddlers (OR 1.48) but inversely with asthma at school age (OR 0.82) [219]; in a Dutch birth cohort, an inverse association with asthma was confined to the preschool years in children of allergic mothers, and later endotoxin was associated with more allergic sensitization [220]; and European birth cohorts show associations in both directions (Section 3.3). Dampness is only a modest determinant of house-dust endotoxin: it was one of several determinants in four European birth cohorts, with heterogeneity between cohorts [221], and it was not among the predictors reported from the US national survey, in which income, pets, cockroaches, smoking, and carpeting were prominent [217].

Actinomycetes have been detected specifically in moisture-damaged structures: *Streptomyces* metabolites, including valinomycin, were found only in building materials from moist structures in severely damaged Finnish homes [174], and the house-dust bacteria that declined after renovation were mostly Actinomycetales, although building-material growth had only a small effect on house-dust communities overall [222]. In a water-damaged office building with a sarcoidosis cluster, thermophilic actinomycetes in dust were associated with lower lung function and granulomatous disease-like symptoms and nontuberculous mycobacteria borderline with asthma symptoms, whereas endotoxin was inversely associated with asthma and fungi and glucan with no outcome [223]. In 39 severely damaged homes, the highest actinomycete counts in building materials were associated with general symptoms and peak-flow variability in adults and with lower respiratory symptoms in children, with wide confidence intervals and no undamaged reference homes [224].

The composition of the indoor microbiota appears to matter in both directions. In a case-control study of 41 children, lower rather than higher fungal diversity in house dust at 12 months predicted asthma at age 7, and elevated moisture was associated with greater diversity [225]. Amish children, whose house dust held several times more endotoxin than that of Hutterite children, had much less asthma and allergic sensitization [226]; in non-farm Finnish and German homes, bacterial communities resembling those of farm homes predicted lower asthma risk [227].

In the only double-blind, placebo-controlled challenge located with spores aerosolized from wet building materials, eight school employees with prior building-related symptoms reacted no more to high spore concentrations than to placebo, although the study was too small to exclude an effect [228]. Microbial volatile organic compounds are not specific markers, since none of more than 200 identified is exclusively microbial, and irritation in chamber studies has appeared at concentrations several orders of magnitude above indoor levels, although toxicological data are sparse [229]. Two hours of 1-octen-3-ol at 10 mg/m³ produced mucosal irritation, nasal inflammatory markers, headache, and nausea [230], whereas 2-ethyl-1-hexanol at 1 mg/m³ produced only minimal odor and eye discomfort and no objective changes [231]. In northern European homes, several microbial volatile compounds, formaldehyde, and a plasticizer were associated with sick building syndrome symptoms, in an analysis with many comparisons [3].

Studies that measured several agents together point in different directions. In a water-damaged office building, hydrophilic fungi remained associated with post-occupancy asthma after adjustment for ergosterol and endotoxin [232]. In 25 European schools selected by moisture-damage status, symptoms were more prevalent and 14 microbial markers generally higher in damaged schools, yet

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wheeze tended to be inversely associated with marker levels and no other symptom consistently with any marker [233]; in the Finnish and Dutch schools of the same project, one bacterial genus and endotoxin partly explained the damage–symptom association in Finland but not in the Netherlands, and the authors judged the explanatory role of individual taxa to be limited [234].

Taken together, these studies indicate that "dampness" marks a mixture, that the association has not been explained by any single measured co-exposure, and that the causal agent or agents remain unidentified. Chemical emissions produced by water in building materials are products of the exposure rather than independent causes, so adjusting for them would remove part of the exposure's effect. Endotoxin and the microbiota as a whole have shown both risk and protective associations with the same outcomes [165,219,225,227], so the agents accompanying water damage should not be assumed to act in the same direction or to add their effects.

3.5.4. Reporting Bias, Misclassification, and Selection

Reporting bias is documented. At every level of measured bronchial lability, parents of children in moldy homes reported more wheeze, leading Strachan to suggest that awareness of mold might account for much of the symptom association [195]. In Canadian homes, reported mold corresponded to higher dust fungi but with high inaccuracy and systematic reporting differences by allergy and smoking status [235]. Agreement between occupants and inspectors can be poor, yet in some studies occupant reports correlated better with case status or with fungal measures, since occupants see intermittent dampness that a single inspection misses [2,236].

For respiratory outcomes, reporting bias cannot account for associations observed with engineer-inspected exposure and clinically determined incident asthma [209], observed damp spots in a prospective cohort [24], moisture meters and surveyor assessment [34,196], or randomized remediation [12,36]. Center-level asthma prevalence also correlated with reported mold among nonasthmatic respondents, which argues against over-reporting by asthmatics alone [210]. Objective outcomes are, however, weaker than symptom outcomes: dampness was associated with reported but not examined eczema and not with bronchial hyperresponsiveness [182], and lung function associations were weak in the US Six Cities study [237]. In a Dutch birth cohort, early-life dampness or mold was associated with slower lung-function growth between ages 12 and 16 [238], but no association was found with maximal lung function at a mean age of 25.7 years [239], and the reported-versus-examined contrast recurs for eczema (Section 3.2.6).

For non-specific symptoms, the available designs cannot separate these processes from a biological effect. In Finnish schools, observed indoor air problems were associated with greater parental worry and with higher child symptom reports, and in cross-sectional mediation analysis worry might account for 67% to 84% of the association [240], although in cross-sectional data parental worry may also be a response to children's symptoms or to the indoor air problems themselves; in schools without indoor air problems, socioemotional difficulties predicted indoor-air-related symptoms [241]; and school-level symptom reporting mediated the link between observed moisture problems and perceived air quality [242]. WHO noted that unpleasant odors can themselves cause stress responses and non-specific symptoms such as headache and nausea [2, p. 89]. To the extent that musty odor is part of the exposure, such responses would be effects of the building rather than reporting artifacts, although they would point to a different mechanism than toxicity. These findings describe limitations of self-report designs. They do not show whether the symptoms are related to the exposure; they show that same-respondent questionnaires cannot distinguish between explanations.

3.5.5. Reverse Causation, Avoidance, and Publication Bias

Reverse causation is addressed by incident and prospective designs, which still show associations, though smaller ones (Section 3.1.2). WHO noted that people with asthma who change their environment to reduce exposure would cause cross-sectional studies to underestimate effects [2, p. 76]. Mendell and colleagues judged that publication bias was likely to have inflated associations,

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while most remaining biases would underestimate them [5]; a meta-analysis of respiratory infections found little effect of publication bias [17].

No E-value or formal quantitative bias analysis was found in this literature. As an illustrative calculation for this review, the E-value formula of VanderWeele and Ding [243] was applied to the incident-asthma summary estimates of 1.27 to 1.48 [20]. Treating the odds ratios as approximate risk ratios, which is reasonable when the outcome is uncommon, gives E-values of about 1.9 to 2.3 for the point estimates and about 1.3 to 1.8 for the lower confidence limits. Under the more conservative conversion recommended for common outcomes (risk ratio approximated by the square root of the odds ratio), the corresponding values are about 1.5 to 1.7 and 1.2 to 1.5. An unmeasured confounder would therefore need to be associated with both dampness and incident asthma by a risk ratio of roughly 1.5 to 2.3 each, conditional on the measured covariates, to explain the point estimates fully.

3.5.6. Bradford Hill Considerations

For respiratory outcomes, most Hill considerations are met to some degree: consistency across settings, dose-response with observed severity, temporality in incident designs, coherence with plausible mechanisms, and experimental evidence from remediation. Strength is modest and specificity is weak, and the agent has not been identified. For these outcomes the IOM concluded that the evidence was sufficient for association but not for a causal relationship, and WHO did not conclude causation [2,4]. Randomized remediation and housing trials balance socioeconomic factors by design and therefore argue against the associations being wholly due to deprivation, but because each intervention changes microbes, mites, humidity, temperature, nitrogen dioxide, and material emissions at once, they test the water-damaged-building complex rather than mold alone [12,36,38]. For non-respiratory outcomes, the associations are of similar magnitude, but temporality has been examined in few studies (Sections 3.2.2 and 3.2.5), no remediation trial has measured fatigue or cognition (one longitudinal building study recorded fatigue and headache during remediation attempts [44]), plausibility rests on animal and in vitro work, and vulnerability to reporting effects is highest. No intervention study has provided the confounding check for these outcomes that remediation trials provide for respiratory outcomes.

3.6. Co-Occurring Clinical Conditions

Several conditions overlap the multisystem symptoms reported in association with water-damaged buildings, are common in women in midlife, and are frequently unrecognized for years. They are relevant in two ways. Clinically, they can coexist with an exposure and add to a person's symptom burden. In research, they can lead to outcome misclassification, blur case definitions, and complicate the interpretation of treatment response; they act as confounders of the exposure–outcome association only where they are themselves associated with exposure. Their presence does not imply that an exposure is unimportant, and a history of exposure does not make them less likely; Table 1 summarizes the evidence reviewed.

Table 1. Co-occurring conditions that overlap symptoms reported in association with water-damaged buildings.

Condition	Burden and under-recognition	Overlapping features	Diagnostic approaches described in the literature
Bile acid malabsorption	About 28% of people meeting IBS-D criteria (SeHCAT <10%)	Chronic diarrhea, urgency, abdominal pain	SeHCAT (outside US), serum C4, fecal bile acids [246,247]

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	[244]; SeHCAT unavailable in the US [245]		
Iron deficiency without anemia	27.4% of US women aged 18–49 by inflammation-adjusted ferritin [248]; hemoglobin and MCV miss much of it [249]	Fatigue, reduced exercise tolerance	Ferritin interpreted with an inflammatory marker [250]
Hypothyroidism	4.6% of the US population, mostly subclinical; TPO antibodies in 11.3% [251]	Fatigue, cognitive slowing, mood, weight, myalgia	TSH and free T4, with thyroid peroxidase antibodies (TPOAb) [252]
Obstructive sleep apnea	93% of moderate-to-severe cases in women undiagnosed in one cohort [253]	Insomnia, depressed mood, nighttime palpitations [254]	Home sleep apnea testing or polysomnography [255]
Perimenopause	Menopausal status carried the largest adjusted symptom odds among 16,065 women aged 40–55 [256]	Sleep disruption, forgetfulness, mood change, stiffness	Menstrual and symptom history [257]
Celiac disease	0.71% of the US population; 29 of 35 cases unaware [258]	Fatigue, neurologic and joint symptoms, anemia [259]	tTG-IgA with total IgA while consuming gluten [260]
POTS	94% female; median diagnostic delay 24 months [261]	Lightheadedness, headache, difficulty concentrating	Active stand or tilt testing [262,263]
ME/CFS	1.3% of US adults, 1.7% of women [264]; most undiagnosed [265]	Post-exertional malaise, unrefreshing sleep, cognitive impairment	Clinical criteria [266,267]
Long COVID	3.4% of US adults currently affected in 2022, highest at ages 35–49 [268]	Fatigue, cognitive symptoms, dysautonomia	Clinical history of infection and symptom onset [269]

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Depression	16.0% of US females in a two-week period [270]	Fatigue, sleep, concentration	Validated screening instruments such as the PHQ-9 [271,272]

3.6.1. Bile Acid Malabsorption and the Interpretation of Binder Response

Bile acid malabsorption is relevant to this review because it bears on how treatment response is interpreted. In a systematic review of 18 studies, 32% of patients with diarrhea-predominant irritable bowel syndrome (IBS-D) had moderate bile acid malabsorption on SeHCAT (7-day retention below 10%), and response to cholestyramine increased with severity [273]. A meta-analysis of six studies estimated a pooled prevalence of 28.1% at the same threshold [244], and a meta-analysis of consecutive secondary-care IBS patients found bile acid diarrhea in 41% [274]. In a Mayo Clinic cohort of 219 patients with IBS-D, 79% of them women, 20% had bile acid diarrhea by serum C4, and those patients had more severe diarrhea and worse quality of life [275]. SeHCAT is not available in the United States, C4 and fecal bile acid testing are not routinely available, and response to a bile acid binder is therefore often used as a surrogate for diagnosis [245,276]. In a prospective Danish diagnostic accuracy study, an empirical treatment trial had only 63% sensitivity and 65% specificity against SeHCAT, and the authors concluded that diagnosis by empirical treatment was inadequate [247].

The agents commonly given as binders to people with illness associated with water-damaged buildings also act on bile acids. Activated charcoal lowered cholesterol in a dose-dependent manner comparable to cholestyramine and raised markers of cholesterol synthesis consistent with interrupted bile acid recirculation [277,278]; activated carbon removed conjugated and unconjugated bile acids from solution in vitro [279]; and cholestyramine binds both ochratoxin A and bile salts in vitro [280]. A hypothesis paper by the present authors, posted as a preprint and not yet peer reviewed, argues that in people with chronic diarrhea after exposure, improvement on a binder cannot by itself distinguish clearance of a toxin from treatment of unscreened bile acid malabsorption, and that this requires only that bile acid malabsorption occur at its usual rate and go untested, not that the exposure cause it [281]. The paper offers testable predictions, including that C4, FGF19, or fecal bile acids should separate binder responders from non-responders, and notes its own limits: it applies only to the diarrheal subset, removal from the building confounds every binder account, and SeHCAT prevalence does not transfer directly to a C4-screened US population [281]. No study has measured bile acid malabsorption by any assay in people exposed to water-damaged buildings.

3.6.2. Iron, Thyroid, Sleep, and Hormonal Transition

Iron deficiency without anemia is common in US women. In NHANES data for 2015 to 2023, 27.4% of non-pregnant women aged 18 to 49 had iron deficiency by inflammation-adjusted ferritin thresholds, compared with 8% who had iron-deficiency anemia [248], and standard hemoglobin and red cell indices miss much of it [249]. Two randomized trials in non-anemic women with fatigue found that iron reduced fatigue in those with ferritin at or below 50 μg/L [282,283]. Biochemical hypothyroidism affects about one in twenty people in the United States, mostly in mild form and often without a prior diagnosis, and thyroid antibodies are more common in women and rise with age [251]. Obstructive sleep apnea in women is common in midlife (moderate-to-severe in 9% of women aged 50 to 70) [284], was undiagnosed in an estimated 93% of moderate-to-severe cases in women in the Wisconsin cohort [253], and in a referred clinical population women reported insomnia, depression, and nighttime palpitations more often than men, while men more often reported witnessed apneas [254]. Peri- and postmenopausal status carried the largest adjusted symptom odds ratios in a cross-sectional survey of 16,065 women aged 40 to 55, including difficulty sleeping, forgetfulness, mood symptoms, and stiffness [256,285].

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3.6.3. Celiac Disease, Vitamins, POTS, and MCAS

Celiac disease affects 0.71% of Americans, most of them unaware of it [258], with higher prevalence in women [286], and extraintestinal manifestations can misdirect diagnosis [259]; gastroenterology guidance suggests celiac serology in IBS with diarrhea [287]. Vitamin D deficiency as defined by a 25-hydroxyvitamin D concentration at or below 20 ng/mL was found in 41.6% of US adults in NHANES 2005–2006 [288], and low vitamin B12 is proportionally more common after age 50 and less common among supplement users [289]. POTS mainly affects women of childbearing age, carries a median diagnostic delay of two years, and causes lightheadedness, headache, and difficulty concentrating in more than 90% of patients [261]; onset can follow immunological stressors such as viral infection [263], and 31% of a highly symptomatic Swedish long COVID cohort met POTS criteria [290]. Mast cell activation occurs in allergic and inflammatory reactions and in clonal mast cell disease. The consensus classification was developed because increasing numbers of patients were being told that their findings reflected mast cell activation in the absence of either, and it defines three mast cell activation syndromes, primary, secondary, and idiopathic [291]. Under those criteria, mast cell activation is identified by typical clinical symptoms, a substantial transient increase in serum total tryptase or in other mast cell mediators such as histamine or prostaglandin D2 or their urinary metabolites, and a response of symptoms to agents that attenuate mediator production or activity [291]; the tryptase increase is assessed during an episode against the person's own baseline [292]. A critical review reported that increasing numbers of patients are given a diagnosis of MCAS on the basis of alternative criteria that often lack specificity and validation, and warned that misdiagnosis may delay recognition of the correct conditions [292]. The frequently quoted estimate that 17% of people are affected derives from symptom self-report rather than mediator confirmation [293]. No study was identified that measured mast cell mediators in people exposed to water-damaged buildings (Section 3.2.8), so whether such exposure produces measurable mast cell activation is untested rather than settled in either direction. For research purposes, studies should record which diagnostic criteria for MCAS were applied, because broader criteria overlap the same nonspecific symptoms while consensus criteria require documented mediator elevation.

3.6.4. ME/CFS, Long COVID, Depression, Fibromyalgia, and Upper Airway Disease

ME/CFS affects about 1.3% of US adults, 1.7% of women, and peaks in midlife [264], and its hallmark is post-exertional malaise [266]; the building-associated fatigue outbreaks described above closely resembled it [80]. For symptoms that began in 2020 or later, long COVID is a possible additional contributor to fatigue, cognitive symptoms, and dysautonomia [268]. Depression affects 16% of US females in any two-week period [270], and because dampness is itself associated with depression partly through physical illness and loss of control over one's home [54], mood symptoms can be a consequence of an exposure and its illness, an independent contributor, or both. About 1.75% of US adults meet survey criteria for fibromyalgia, yet 73% of them report a different diagnosis, while 73.5% of those carrying the diagnosis do not meet criteria, a two-way misclassification that complicates the classification of widespread pain and fatigue [294,295]. Allergic rhinitis and chronic rhinosinusitis, by contrast, have themselves been associated with home dampness in meta-analysis and in population samples, so they are part of the exposure picture rather than alternatives to it: dampness and mold are associated with rhinitis risk in meta-analysis [22], and in a Swedish population sample of 26,577 adults, reported home dampness was associated with symptom-defined chronic rhinosinusitis (adjusted OR 1.71), with risk rising with the number of dampness signs [296]; chronic rhinosinusitis affects about one in ten European adults [297], and its symptom burden overlaps the systemic symptoms reported in association with water-damaged buildings.

3.6.5. Has Any Study Screened for These Conditions?

No study of illness associated with water-damaged buildings was identified that systematically screened for the conditions above. The closest evidence comes from small clinic series with strong

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selection effects. Among 65 patients presenting to an allergy center with concerns about mold exposure, rhinitis and cough were most common and 53% had mold skin-test reactivity [298]. A retrospective review of 50 of 82 consecutive medical evaluations for suspected mold-related toxicity reported that 30 patients (60%) had evidence of non-mold-related conditions, many of them preexisting, that could explain their presenting complaints; two had mold allergy, and the authors considered the symptoms of 17 others to reflect transient aeroirritation [299]. The paper's abstract does not describe how patients were referred, but a news report at the time of publication stated that they had been referred by a defense attorney in civil litigation or by insurance adjusters for workers' compensation carriers [300]. That setting limits generalizability, and the presence of other conditions does not establish that the exposure was irrelevant. A Finnish series of 12 patients with indoor-air-related work disability found that nearly all had co-occurring somatic and psychiatric diagnoses [301], and subsequent Finnish trials of psychological interventions reported mixed results, with an underpowered trial of cognitive behavioral therapy missing its primary endpoint and internet-delivered acceptance and commitment therapy improving quality of life and work ability [302–304]. In the one of these reports whose examinations are described in detail, the assessment covered somatic, psychological, and psychiatric evaluation, autonomic function including an active orthostatic test, cortisol measurements, lung function, and allergy tests; no screening for iron deficiency, thyroid disease, sleep apnea, celiac disease, bile acid malabsorption, or perimenopause is reported [301].

3.7. Susceptibility

Why symptoms differ among occupants of the same building is a recurring clinical and research question. Established modifiers are atopy and pre-existing asthma for allergic outcomes and immunocompromise for infection [4,8,189]. Before differences between occupants are interpreted as individual susceptibility, exposure heterogeneity must be considered: risk in an engineer-inspected study rose with moisture damage in the main living quarters but not elsewhere in the house [209], and ERMI values from bedroom and living room dust in the same home were correlated but not identical ($r = 0.69$) [170].

A genetic explanation that has commonly been offered in the CIRS literature holds that particular HLA-DR haplotypes mark susceptibility, with a reported population carrier frequency of about 24%. A preprint by one of the present authors (DS), using National Marrow Donor Program haplotype frequencies, estimated that 88.4% of European Americans carry at least one of the listed haplotypes under a broad reading and 59.1% under a strict reading, with all 28 of the 64 world populations with two-locus data that passed data-quality screens exceeding 24% under the broad reading [305]. The 24% figure could not be reproduced from any dataset analyzed, and its originator attributes it to an unpublished clinical series; the proposed relative risks rest on a comparison group of 111 attendees of the same specialty clinic [305]. No study was identified that evaluated HLA-DR typing as a diagnostic or predictive test for illness associated with water-damaged buildings, whether as a case-control study with independent population controls, as a prospective study of typed occupants, or as a study reporting sensitivity and specificity against a reference standard. A 2025 review written from within the CIRS framework states, in its account of the 2005–2010 period, that "no peer-reviewed stratification of haplotype-specific risk has yet been published" [306]. Neither the AWMF guideline nor the 2025 position statement of the American College of Medical Toxicology addresses HLA typing [8,11]. Independent studies show that HLA class II genes can shape responses to fungal antigens in other conditions: lung T cells from patients with Löfgren's syndrome recognized an *Aspergillus* peptide in an HLA-DR3-restricted manner [307], HLA-DR and -DQ alleles differed by mold sensitization in children with asthma [308], and the ACCESS sarcoidosis study found only a suggestive interaction between HLA-DRB1*1101 and exposure to molds and musty odors [309]. In ME/CFS, HLA associations found against 4,511 population controls were modest (odds ratios 1.5 and 2.1) and were carried by 19% of patients and 12% of controls, without exposure data [310]. These studies address other questions; none tests the haplotype panel used in CIRS practice. If the

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sensitivities reported for the classification are taken at face value, the preprint's carrier-frequency estimates imply positive likelihood ratios of 0.85 to 1.07 under the broad reading and 1.3 to 1.6 under the strict reading, so a positive result would change the probability of the condition little or not at all [305]. On current evidence, HLA-DR typing has not been shown to provide diagnostic information for this purpose. The preprint notes that two-locus data may overstate carriage where historical serological distinctions cannot be resolved, and it has not been peer reviewed. Whether the co-occurring conditions in Section 3.6, allergic sensitization, or prior viral illness account for part of the variation between occupants has not been tested; unlike HLA haplotype, each of them can be measured directly.

4. Discussion

The evidence supports several conclusions with differing degrees of confidence. Occupants of water-damaged buildings consistently have higher rates of respiratory and allergic symptoms, respiratory infections, and asthma, with odds ratios of about 1.3 to 1.8, and two later reviews have judged the evidence causal for asthma exacerbation in children, one of them also for asthma development, while a third found causation strongly suggested [5–7]. A 2024 GRADE assessment rated the evidence for new-onset wheeze as of moderate certainty but the evidence for asthma-related outcomes in people who already have asthma as uncertain [14], so the strength of the causal judgment for exacerbation depends on the framework applied. Confounding by socioeconomic deprivation does not appear to explain these associations, given modest shifts on adjustment, persistence across very different housing contexts, and benefit in randomized trials. Reporting bias inflates some symptom estimates, but associations persist with inspected exposure, incident clinical outcomes, and randomized remediation. What the evidence cannot yet do is separate the contribution of fungi from that of bacteria, endotoxin, mites, chemical emissions, and the moisture itself, because the exposure metric that best predicts health is observational and nonspecific. Studies measuring several agents together identify different candidate agents in different settings, and endotoxin has shown protective as well as adverse associations [219,232–234].

For the chronic, multisystem symptoms that many people report, few studies have used designs capable of answering the question. The available studies are mostly cross-sectional and self-reported, with associations of similar magnitude to the respiratory findings. Clinic series and the early neurocognitive studies were drawn from referred populations. The strongest newer designs give mixed signals: a within-person panel analysis found a small association with psychological distress [55], inspector-scored exposure showed an exposure-response with self-reported cognitive symptoms [71], birth cohorts report associations with emotional symptoms and, in two cohorts that administered tests, with lower cognitive scores, but not with hyperactivity [91–93], and objective immune and laboratory measures have been mixed or null [116,119]. No prospective cohort has followed exposed people for clinically adjudicated multisystem outcomes, no remediation trial has measured fatigue or cognition, and no study has paired objective exposure with exposure-blinded neuropsychological testing. For metabolic, hepatic, renal, endocrine, cardiovascular, and autonomic outcomes, for cancer, and for the gut microbiome of occupants, the supplementary search identified no epidemiological study at all. The IOM asked for this research in 2004 [4]. Under the search used for this review (Section 2), NIH funding for projects mentioning indoor dampness, water damage, or indoor mold totaled roughly $14.3 million across 12 core projects from fiscal years 2015 to 2026, several of them animal or immunology studies, and none of the projects identified by title studies chronic multisystem symptoms in occupants [311]. A September 18, 2026 HHS roundtable on mold "focused on invasive mold disease surveillance and diagnosis," according to the HHS event summary [312]. This is a gap in research; it is evidence neither for nor against an effect, and it has persisted for more than two decades since the IOM's recommendation.

The question of what the observed associations represent therefore has three parts. The first is whether, and how much, moisture and its microbial growth contribute to symptoms. The second is how much other agents that accompany water damage contribute, including endotoxin, mites, pest

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allergens, and plasticizer breakdown products, along with cold and poor ventilation, which are upstream causes of dampness as well as independent risks. The third is what else is going on in the person. On the third part, the evidence reviewed here shows that several conditions overlapping the reported symptom picture are common in women in midlife, are frequently missed, and have never been systematically measured in exposed cohorts. That absence affects clinical care, because a person may have an exposure-associated illness together with an unrecognized condition that adds to it, or an unrecognized condition whose symptoms resemble an exposure-associated illness, and identifying the condition matters in either case. It also affects research, because case definitions and treatment-response studies that do not record these conditions cannot estimate whether, or how much, the exposure contributes. Bile acid malabsorption is a specific instance in which the interpretation of treatment response depends on a condition that is rarely tested for in the United States.

The evidence reviewed here does not yet distinguish, for most non-respiratory outcomes, between a causal effect of water-damaged buildings and associations produced by bias, confounding, or co-occurring conditions. Table 2 sets out, for each body system, the kinds of findings that would strengthen or weaken a causal interpretation. The same standard applies in both directions: a null result weakens a causal interpretation only when the study had adequate power, an objectively measured exposure, and an outcome measure suited to the symptoms people report, and a positive result strengthens it only when exposure and outcome were not assessed by the same respondent. Co-occurring conditions weaken a causal interpretation only when they preceded the exposure and are not plausibly caused by it; a condition that the exposure could produce would be a mediator rather than an alternative explanation.

Table 2. Findings that would strengthen or weaken a causal interpretation, by body system.

Body system	Current state of evidence	Findings that would strengthen a causal interpretation	Findings that would weaken a causal interpretation
Respiratory and allergic	Consistent pooled odds ratios of about 1.3 to 1.8, smaller in cohorts; causal judgments for asthma exacerbation in children in two reviews, rated uncertain for people with asthma under GRADE; remediation improves symptoms more consistently than lung function [6,7,14,16,23,35]; measured microbial components associated with outcomes in both directions [165,233]	Dose-response with measured building moisture [34]; improvement in objective outcomes (exacerbations requiring care, lung function) after remediation, assessed by evaluators blind to allocation; identification of a measured agent whose level predicts incident asthma	Adequately powered prospective cohorts with measured moisture showing no excess incident asthma; remediation trials with objective outcomes and attention-control arms showing no benefit; associations confined to self-reported outcomes with no objective counterpart

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		independently of observed dampness	
Neurological and cognitive	Referred case series without exposure-blinded testing [83–85]; one null case-control study of mild cognitive impairment in older adults [87] and one positive cross-sectional study with an administered screen [88]; inspector-scored exposure-response with self-reported cognitive symptoms [71]; child cohorts with lower tested scores but no hyperactivity association [91–93]; mouse studies at doses of unknown human relevance [129,133,134]	Deficits on exposure-blinded neuropsychological testing in population-sampled exposed occupants compared with unexposed controls, with dose-response by measured exposure; deficits in incident cases absent at a pre-exposure baseline; improvement after remediation or relocation exceeding that in a comparison group; replication by independent groups; human olfactory or neuroinflammatory findings paralleling the animal work	No difference on blinded testing in adequately powered population-based studies with objective exposure; deficits fully accounted for by co-occurring conditions that preceded exposure (sleep apnea, thyroid disease, iron deficiency, depression); deficits seen in referred samples that do not replicate in population samples
Psychiatric (mood, anxiety, sleep)	Mostly cross-sectional studies with self-reported exposure; odds ratios of about 1.2 to 1.9; partial attenuation with perceived control and physical illness; a small within-person association in panel data [53–55,62]	Prospective association with inspected or measured dampness in people without baseline symptoms; dose-response by objective exposure; improvement after remediation compared with control housing; persistence after adjustment for	Associations that disappear when exposure is assessed by inspection rather than self-report; associations fully explained by housing insecurity, financial strain, or depression measured before exposure; no change in mood outcomes after effective remediation

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		socioeconomic status and housing insecurity (without adjusting for plausible mediators)	
Gastrointestinal	Graded inadequate [4,8]; few studies, mostly self-reported with low response rates [73]; no study has measured bile acid malabsorption in exposed people; empirical binder response is not diagnostic [247,281]; no study of hepatic, metabolic, or renal outcomes or of the occupant gut microbiome	Higher incidence of objectively characterized gastrointestinal findings in exposed occupants than in population controls; symptom improvement after remediation exceeding that in a comparison group; benefit from binders in randomized trials among people without bile acid malabsorption on C4 or fecal bile acid testing	Gastrointestinal excess confined to people with bile acid malabsorption, celiac disease, or other conditions that preceded exposure; binder response confined to people with bile acid malabsorption [281]; no gastrointestinal excess in population cohorts with measured exposure
Immune and inflammatory (non-allergic, systemic)	Non-allergic associations for respiratory outcomes [5,182]; immune shifts with repeated exposure in animals [139,183]; proposed syndromes without accepted laboratory tests [194]; contested mast cell criteria [291,292,313]; a cross-sectional C-reactive protein association not seen prospectively [115,116]; routine laboratory results in	Prespecified markers, with standardized specimen handling, that differ between exposed occupants and population controls, track measured exposure, and normalize after remediation, replicated by independent laboratories; mediator elevations meeting consensus criteria	Marker differences that disappear with population-based controls or standardized specimen handling; no difference in markers measured blind to exposure status; inflammatory findings fully explained by co-occurring conditions that preceded exposure

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	referred patients similar to controls [119]; occupational association with sarcoidosis [126]	more often in exposed people, which would require episode-timed sampling against individual baselines	
Fatigue and multisystem symptoms	Survey associations, some with checks on reporting tendency [65,66]; outbreak reports and clinic follow-up [80,82]; no prospective cohort with adjudicated outcomes; no systematic screening for co-occurring conditions [299,301]; symptom amplification not elevated in one clinic sample [77]; a blinded spore challenge in eight symptomatic employees found no excess reactions but was too small to exclude an effect [228]; measured verrucarol associated with tiredness in one school study [214]	Prospective cohorts enrolling both ill and well occupants, with objective exposure, in which incidence rises with exposure after co-occurring conditions are measured; differences in post-exertional malaise or objective function by exposure, using established criteria [266]; improvement after remediation or relocation exceeding that in a comparison group with similar expectations; clusters in buildings whose occupants were unaware of the damage	Excess confined to settings where occupants knew of the damage; incidence unrelated to objective exposure in adequately powered cohorts; symptoms fully explained by co-occurring conditions that preceded exposure and are not plausibly caused by it; no change after effective remediation

Urinary mycotoxin panels cannot link a result to a building and have no established disease thresholds, dust indices have not been validated for clinical decisions, the HLA classification used to explain susceptibility has not been validated and, under its reported sensitivities, would carry little diagnostic information, the CIRS literature rests largely on a single group's case series, and the dose models used to argue that toxic indoor doses are implausible come from a single group of authors and do not address fragment-borne, chronic low-dose, or mixed exposures (Section 3.4). These limitations fall on tests, protocols, and study designs. None of them bears on whether people are ill, and none settles whether the exposure contributes to their illness. The persistence of disability in long-term follow-up [82] and the association between delayed evaluation and worse work outcomes in that cohort are consistent with a benefit from earlier, broader clinical assessment, although that association is observational.

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5. Recommendations for a Rigorous Future Review

A future evidence review capable of resolving these questions would need the following features.

Governance and conflicts of interest. The review should be convened by a body such as the National Academies or conducted under the NTP Office of Health Assessment and Translation (OHAT) framework, which was designed for environmental exposures and integrates human, animal, and mechanistic evidence [314]. Panelists and authors should be screened for financial conflicts, including litigation consulting, test or product sales, and clinical services tied to particular diagnoses, a standard that would apply to the present authors. Foundational papers in this field include dose models by authors affiliated with consulting firms, index evaluations published by the index developer, and clinical case series from groups that provide the associated diagnostic or treatment services (Sections 3.2.11, 3.2.12, and 3.4), and conflicts should be recorded at the level of individual included studies.

Protocol and patient partnership. The protocol should be preregistered (for example in PROSPERO or the Open Science Framework), with population, exposure, comparator, and outcome questions developed with patients. Structured patient partnership should occur at question setting, protocol review, and interpretation, drawing on established engagement models such as priority-setting partnerships and patient-centered outcomes research principles.

Methods. Screening, data extraction, and risk-of-bias assessment should be performed in duplicate. Evidence should be organized into separate streams (human observational, intervention, animal inhalation, and mechanistic) and integrated using OHAT, the Navigation Guide, or GRADE adapted for environmental health [13,314,315], with starting confidence for observational studies based on design features rather than automatic downgrading. The divergence between the 2024 GRADE assessment and earlier causal judgments for asthma exacerbation [6,7,14] shows how much conclusions can depend on the framework chosen, so the choice should be justified in the protocol. Evidence should be graded separately by exposure metric (observed dampness, measured moisture, measured microbial agents, and specific co-exposures), because the metrics differ in what they measure and in how well they predict health outcomes (Sections 3.3 and 3.5.2). Outcomes should distinguish clinically adjudicated endpoints from self-reported symptoms. Quantitative bias analysis, including E-values, should be reported. Conclusions should state explicitly whether evidence is inadequate because it is absent or because adequate studies found no effect.

Co-occurring conditions as data. Included studies should be coded for whether they measured or adjusted for co-occurring conditions, and the review should report this as a dimension of study quality for multisystem outcomes. Where condition data exist, they should be analyzed according to their likely role (as confounders where they are associated with exposure, as effect modifiers, as additive contributors, or as sources of outcome misclassification), not as exclusions; conditions that could themselves be consequences of exposure should not be adjusted for as confounders.

Primary research agenda. The review should specify the research needed to close the gaps it identifies: a research case definition for multisystem illness associated with water-damaged buildings, developed by a process similar to the IOM's for ME/CFS [266], together with standardized symptom and outcome measures that remain usable if no distinct syndrome emerges; validated personal exposure measures, including measured building moisture and water activity [34]; prospective cohorts of occupants that enroll both ill and well people, use objective exposure assessment, and record co-occurring conditions including bile acid malabsorption (by C4 or fecal bile acids), ferritin, thyroid function, sleep-disordered breathing, menopausal status, celiac serology, orthostatic testing, and post-viral history; remediation trials with non-respiratory outcomes and dismantling designs that separate moisture repair, material removal, and ventilation; urinary mycotoxin studies by mass spectrometry with population-based controls and dietary assessment; binder trials that stratify by bile acid status and compare bile acid sequestrants with different toxin-binding properties [281]; and susceptibility studies with population-based controls that account for within-building differences in exposure.

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6. Limitations

This review was conducted without a registered protocol, a formal risk-of-bias instrument, or duplicate screening and extraction, so selection and interpretation may reflect the authors' judgment and prior views. Searches were limited mainly to PubMed, Europe PMC, and agency sources, largely in English. The supplementary search was structured and its strings are reported, but it was run once, screened in a single pass without duplicate review, and did not include Embase, Web of Science, or gray literature beyond agency sources; several strategies returned many irrelevant records, of which only the first were screened, so relevant studies may have been missed. Many effect estimates, including most from the supplementary search, were taken from abstracts rather than full texts, and several numbers await full-text confirmation. Several secondary characterizations could not be confirmed against primary documents and are labeled as such. Some newer evidence comes from multiple analyses of single datasets, which were identified where recognized but may not have been in every case. The NIH funding figure is a lower bound from one search strategy, excludes other federal agencies, and includes NIH-funded interagency agreements. The two preprints by the authors that are cited here have not been peer reviewed. Literature retrieval was assisted by software tools, which may have influenced which sources were found. Finally, the authors have financial and professional interests related to this topic, described below.

Author Contributions: DS conceived the review, designed and ran the searches, selected and appraised the sources, and wrote the manuscript. Both authors developed the review questions and the framing, including the decision to examine co-occurring clinical conditions alongside the epidemiology. AS read the complete draft and revised the manuscript critically for intellectual content; that reading took place after the draft was complete and did not constitute duplicate screening or duplicate data extraction (Section 2). Both authors approved the final version and accept accountability for the accuracy and integrity of the work.

Funding: No external funding was received for this review.

Data Availability: No new data were generated. The search strategy for the NIH RePORTER query is described in Section 2, and the supplementary search strings and hit counts are listed in the Appendix; extraction notes are available from the corresponding author on request.

Ethics Statement: Not applicable; this review analyzed published literature and public records only.

Conflicts of Interest Statement: DS is a physician whose consulting work includes people with illness associated with water-damaged buildings, and she derives income from that work and from associated educational programs. DS recommends oral binders and supplements to clients through Fullscript, a practitioner dispensary platform, and receives a commission on non-prescription purchases made through it, including binder products of the classes discussed in this review. DS has a promotional agreement with BCL Wellness, which manufactures a line of non-prescription binder products. DS and AS are co-authors of *Conquering Mold* (2024), a book on illness following exposure in water-damaged buildings, receive royalties from its sale, and are revising it in light of evidence discussed here. DS and AS are planning a telehealth service for bile acid malabsorption screening, which is not yet operating. DS and AS are co-authors of the bile acid malabsorption hypothesis preprint (doi:10.20944/preprints202609.2001.v1), and DS is the sole author of the HLA carrier-frequency preprint (doi:10.20944/preprints202610.2763.v1); both are cited in this review. AS is the spouse of DS. Neither author has received payment, commission, or other benefit from any laboratory or manufacturer in connection with urine mycotoxin panels or mycotoxin antibody testing, and neither holds a financial interest in their sale. No commercial entity had any role in the conception, drafting, or decision to publish this review.

Appendix A. Supplementary Search Log

The supplementary search (Section 2) was run on November 9, 2026, in four body-system areas and two exposure-agent areas. The table compiles the exact search strings and hit counts recorded for each strategy. Hit counts are as returned on the search date. Several strategies returned large numbers of irrelevant records, mainly because "dampness" is also a diagnostic term in traditional

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Chinese medicine; in those cases the first records sorted by relevance or citation count were screened, as noted. A few entries were recorded in summarized rather than verbatim form in the original logs (N1, N16, N18, G15, IE4, and the food-term exclusion and abbreviated outcome blocks in M2, M5, and M7) and are reproduced as recorded; within a string, "#n" refers to strategy n in the same area. Strategy identifiers are prefixed by area (N, neurological and related; R, respiratory and other; G, gastrointestinal and related; I, immune and related; M, mycotoxins as an indoor exposure; A, agents other than fungi, in which the letters after the A are the original log labels). Three web rows in area M record agency guidance pages that were read (M19 to M21); they are not counted among the database strategies. Repeated exposure blocks are abbreviated as follows; the block names D_R, D_I, and X_I were renamed from the original logs (where each was called D or X) to keep the areas distinct.

- EXP_E (Europe PMC exposure block, area N): `TITLE_ABS:("indoor dampness" OR "residential dampness" OR "home dampness" OR "household dampness" OR "damp housing" OR "damp homes" OR "damp home" OR "damp building" OR "damp buildings" OR "mold exposure" OR "mould exposure" OR "visible mold" OR "visible mould" OR "indoor mold" OR "indoor mould" OR "household mold" OR "household mould" OR "water damage" OR "water-damaged" OR "moisture damage" OR "dampness and mold" OR "dampness and mould" OR "mold odor" OR "mould odour")`, with `NOT TITLE_ABS:("Chinese medicine" OR TCM OR acupuncture OR decoction)`. A first attempt that used bare "dampness" returned mostly traditional Chinese medicine "dampness syndrome" papers, so those terms were excluded.
- EXP_P (PubMed exposure block, area N): `(dampness[tiab] OR "damp housing"[tiab] OR "damp homes"[tiab] OR "damp home"[tiab] OR "damp buildings"[tiab] OR mould[tiab] OR mold[tiab] OR moldy[tiab] OR mouldy[tiab] OR "water damage"[tiab] OR "water-damaged"[tiab] OR "moisture damage"[tiab] OR "moisture-damaged"[tiab]) AND (home[tiab] OR homes[tiab] OR housing[tiab] OR residential[tiab] OR indoor[tiab] OR building[tiab] OR buildings[tiab] OR dwelling*[tiab] OR household*[tiab])`, with `NOT ("medicine, chinese traditional"[mh] OR TCM[tiab] OR acupuncture[tiab])`.
- D_R (Europe PMC exposure block, area R): `(TITLE_ABS:dampness OR TITLE_ABS:"damp housing" OR TITLE_ABS:"indoor mold" OR TITLE_ABS:"indoor mould" OR TITLE_ABS:"visible mold" OR TITLE_ABS:"visible mould" OR TITLE_ABS:"water damage" OR TITLE_ABS:"moisture damage" OR TITLE_ABS:"household mold" OR TITLE_ABS:"household mould" OR TITLE_ABS:"mold exposure" OR TITLE_ABS:"mould exposure")`.
- D_I (PubMed exposure block, area I): `(dampness[tiab] OR "moisture damage*"[tiab] OR "moisture-damage*"[tiab] OR "water-damaged"[tiab] OR "water damage"[tiab] OR "indoor mold"[tiab] OR "indoor mould"[tiab] OR "damp building*"[tiab] OR "mold exposure"[tiab] OR "mould exposure"[tiab] OR "moldy"[tiab] OR "mouldy"[tiab])`.
- X_I (PubMed exclusion block, area I): `NOT (chinese[tiab] OR TCM[tiab] OR syndrome[ti] OR decoction[tiab] OR acupuncture[tiab] OR moxibustion[tiab] OR "dampness-heat"[tiab] OR "cold-dampness"[tiab] OR "dampness syndrome"[tiab] OR "damp-heat"[tiab] OR "Medicine"[ti] OR rats[tiab] OR mice[tiab])`.

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Area	ID	Database	Search string	Date	Hits
Neurological, cognitive, psychiatric, sleep, child development	N1	Europe PMC	EXP (broad, incl. bare "dampness") AND TITLE_ABS:(depression OR anxiety OR "mental health" OR psychological OR psychiatric OR "psychological distress") (years: 2022–2026)	Nov 9, 2026	70 (mostly TCM noise)
	N2	Europe PMC	EXP_E AND TITLE_ABS:(depression OR depressive OR anxiety OR "mental health" OR psychiatric OR "psychological distress" OR suicide OR wellbeing OR "well-being") (years: 2023–2026)	Nov 9, 2026	14
	N3	Europe PMC	EXP_E AND TITLE_ABS:(sleep OR insomnia OR snoring OR "sleep quality") (years: 2015–2026)	Nov 9, 2026	12
	N4	Europe PMC	EXP_E AND TITLE_ABS:(cognitive OR cognition OR dementia OR "mild cognitive impairment" OR memory OR neuropsych* OR neurocognitive OR "executive function") (years: 2015–2026)	Nov 9, 2026	14
	N5	Europe PMC	EXP_E AND TITLE_ABS:(ADHD OR "attention deficit" OR autism OR autistic OR neurodevelopment* OR "behavioral problems" OR "behavioural problems" OR "child development" OR hyperactivity) (years: 2015–2026)	Nov 9, 2026	2
	N6	Europe PMC	EXP_E AND TITLE_ABS:(headache OR migraine OR neuropathy OR neurological OR olfactory OR smell OR neuroinflammation OR brain OR MRI OR "nervous system") (years: 2015–2026)	Nov 9, 2026	30
	N7	PubMed	EXP_P AND (ADHD OR "attention deficit" OR autism OR autistic OR neurodevelopment* OR "child behavior" OR "behavioral problems" OR "behavioural problems" OR hyperactivity OR "cognitive development" OR IQ)[tiab] (years: all years)	Nov 9, 2026	15

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	N8	PubMed	EXP_P AND (cognitive OR cognition OR dementia OR Alzheimer* OR neuropsycholog* OR memory OR brain OR neuroimaging OR MRI)[tiab] (years: 2015–2026)	Nov 9, 2026	37
	N9	PubMed	EXP_P AND (depress* OR anxiety OR "mental health" OR psychiatric OR "psychological distress" OR suicid* OR schizophren* OR psychosis OR wellbeing)[tiab] (years: 2023–2026)	Nov 9, 2026	32
	N10	PubMed	EXP_P AND (sleep* OR insomnia OR snoring OR apnea)[tiab] (years: 2015–2026)	Nov 9, 2026	28
	N11	PubMed	EXP_P AND (headache* OR migraine OR neuropath* OR neurolog* OR olfact* OR smell OR neuroinflamm* OR "nervous system" OR tremor OR paresthes*)[tiab] (years: 2015–2026)	Nov 9, 2026	55
	N12	Europe PMC	(NeuroQuant OR "volumetric MRI" OR "diffusion tensor" OR "brain imaging" OR "brain MRI" OR SPECT OR qEEG) AND ("water-damaged building*" OR "chronic inflammatory response syndrome" OR "mold exposure" OR "mould exposure" OR "mold illness" OR "toxic mold") (years: all)	Nov 9, 2026	25
	N13	PubMed	(Stachybotrys OR satratoxin* OR "macrocyclic trichothecene*" OR roridin OR verrucarin)[tiab] AND (brain OR neur* OR olfactory OR cognit* OR microglia* OR hippocamp* OR astrocyt*)[tiab] (years: 2015–2026)	Nov 9, 2026	21 (mostly SMTP drug-development papers)
	N14	PubMed	("1-octen-3-ol" OR "fungal volatile organic" OR "microbial volatile organic" OR "mold volatile*")[tiab] AND (neurotox* OR dopamin* OR neuro* OR brain OR Drosophila)[tiab] NOT (insect* OR mosquito* OR attract* OR oviposit*)[tiab] (years: 2013–2026)	Nov 9, 2026	23

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	N15	PubMed	(mold OR mould OR fungal OR Aspergillus OR Penicillium OR Alternaria)[tiab] AND (inhal* OR intranasal* OR "spore exposure")[tiab] AND (brain OR hippocamp* OR neuroinflamm* OR microglia* OR cognit* OR "anxiety-like" OR "depressive-like")[tiab] AND (mice OR mouse OR rats OR murine)[tiab] (years: 2015–2026)	Nov 9, 2026	40 (mostly *Cryptococcus* infection models)
	N16	PubMed	broad fungal-spore/VOC terms AND brain/behavior terms (years: 2015–2026)	Nov 9, 2026	316 (too noisy; replaced by #13–15)
	N17	Europe PMC	ABSTRACT:(mold OR mould OR dampness OR damp) AND ABSTRACT:(home OR housing OR residential OR dwelling OR indoor) AND TITLE:(ADHD OR autism OR neurodevelopment* OR "cognitive development" OR "behavioral problems" OR "child development" …) (years: all)	Nov 9, 2026	6
	N18	Europe PMC	child behavior/development terms AND (mold OR mould OR dampness OR "home environment") AND child terms (years: 2010–2026)	Nov 9, 2026	210 (mostly irrelevant "home environment" hits; screened)
	N19	PubMed	EXP_P AND ("olfactory dysfunction" OR anosmia OR hyposmia OR "smell identification" OR "peripheral neuropathy" OR "small fiber" OR "nerve conduction" OR "postural sway" OR balance OR "reaction time" OR electroencephalogra*)[tiab] (years: all)	Nov 9, 2026	27
	N20	PubMed	EXP_P AND (ADHD OR "attention deficit" OR "strengths and difficulties" OR "emotional problems" OR "conduct problems" OR "developmental delay" OR	Nov 9, 2026	3

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			"Ages and Stages" OR Bayley OR "mental development")[tiab] (years: 2014–2026)		
	N21	PubMed	EXP_P AND (dementia OR Alzheimer* OR Parkinson* OR "multiple sclerosis" OR amyotrophic OR neurodegenerat*)[tiab] (years: all)	Nov 9, 2026	12
	N22	PubMed	NeuroQuant[tiab] AND (inflammat* OR mold OR mould OR CIRS OR "water-damaged" OR biotoxin*)[tiab] (years: all)	Nov 9, 2026	5
	N23	PubMed	"chronic inflammatory response syndrome"[tiab] AND (brain OR cognit* OR neuro*)[tiab] (years: all)	Nov 9, 2026	8
Respiratory, skin and eye, reproductive, cancer, occupational, floods, reviews	RE1	Europe PMC	TITLE:(dampness OR mould OR mold OR "water damage" OR "moisture damage") AND (TITLE:"meta-analysis" OR TITLE:"systematic review" OR TITLE:"review") AND PUB_YEAR:[2019 TO 2026] AND SRC:MED	Nov 9, 2026	49
	RE2	Europe PMC	(TITLE_ABS:"dampness" OR TITLE_ABS:"mould" OR TITLE_ABS:"mold") AND (TITLE_ABS:"meta-analysis" OR TITLE_ABS:"systematic review") AND PUB_YEAR:[2019 TO 2026] AND (TITLE_ABS:"indoor" OR TITLE_ABS:"home" OR TITLE_ABS:"house" OR TITLE_ABS:"building")	Nov 9, 2026	9
	RE3	Europe PMC	(TITLE_ABS:dampness OR "damp housing" OR "indoor mould" OR "indoor mold" OR "water-damaged" OR "damp and mould" OR "damp and mold") AND (PUB_TYPE:review OR TITLE:review OR TITLE:umbrella OR TITLE:"state of the") AND PUB_YEAR:[2022 TO 2026] AND SRC:MED	Nov 9, 2026	184 (mostly TCM "dampness" noise)
	RE4	Europe PMC	(dampness OR mould OR mold OR "water damage") AND TITLE:"meta-analysis" AND (asthma OR wheeze	Nov 9, 2026	216

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			OR rhinitis OR respiratory OR eczema) AND PUB_YEAR:[2016 TO 2026] AND SRC:MED		
	RE5	Europe PMC	D_R AND (TITLE:COPD OR TITLE:"chronic obstructive" OR TITLE:"lung function" OR TITLE:spirometr* OR TITLE:FEV1) AND PUB_YEAR:[2015 TO 2026] AND SRC:MED	Nov 9, 2026	17
	RE6	Europe PMC	D_R AND (TITLE:rhinosinusitis OR TITLE:sinusitis OR TITLE:"nasal polyp*") AND PUB_YEAR:[2010 TO 2026] AND SRC:MED	Nov 9, 2026	4
	RE7	Europe PMC	D_R AND (TITLE:"respiratory infection*" OR pneumonia OR bronchiolitis OR otitis OR "common cold") AND PUB_YEAR:[2015 TO 2026] AND SRC:MED	Nov 9, 2026	22
	RE8	Europe PMC	(dampness OR "visible mold" OR "visible mould" OR "mould odour" OR "mold odor" OR "water damage") AND TITLE_ABS:adult* AND TITLE:asthma AND PUB_YEAR:[2017 TO 2026] AND SRC:MED	Nov 9, 2026	17
	RE9	Europe PMC	(dampness OR "moisture damage" OR "water damage" OR mould OR mold) AND (remediation OR renovation OR intervention OR repair*) AND (randomi* OR trial OR "follow-up" OR "before and after" OR controlled) AND (asthma OR respiratory OR symptoms) AND PUB_YEAR:[2015 TO 2026] AND SRC:MED	Nov 9, 2026	26
	RE10	Europe PMC	TITLE:"remediating buildings damaged" (Cochrane update check)	Nov 9, 2026	4 (no update after 2015)
	RE11	Europe PMC	("moisture damage" OR "moisture-damaged" OR "mould damage" OR "mold damage" OR "indoor air problem*") AND (renovation OR remediation OR	Nov 9, 2026	15

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			repair* OR relocation) AND PUB_YEAR:[2012 TO 2026] AND SRC:MED		
	RE12	Europe PMC	("Healthy Homes Initiative" OR "Warm Up New Zealand" OR "Arbed" OR "home improvement" OR "housing intervention*") AND (mould OR mold OR damp*) AND (hospitali* OR asthma OR respiratory) AND PUB_YEAR:[2015 TO 2026] AND SRC:MED	Nov 9, 2026	73
	RE13	Europe PMC	(flood* OR hurricane OR Katrina OR Harvey OR "Hurricane Maria" OR "Hurricane Sandy" OR "Hurricane Ian") AND (mold OR mould OR dampness OR "water damage" OR "water-damaged") AND (respiratory OR asthma OR health OR symptoms) AND SRC:MED	Nov 9, 2026	103
	RE14	Europe PMC	same, PUB_YEAR:[2016 TO 2026], sorted by date	Nov 9, 2026	47
	RE15	Europe PMC	(dampness OR "moisture damage" OR "water damage" OR "water-damaged" OR mould OR mold) AND (TITLE:school* OR teacher* OR office* OR hospital OR workplace OR occupational OR "day care" OR kindergarten) AND (symptom* OR asthma OR respiratory) AND PUB_YEAR:[2015 TO 2026] AND SRC:MED	Nov 9, 2026	94
	RE16	Europe PMC	("water damage" OR "water leak*" OR "water-damaged" OR flood*) AND hospital AND (aspergill* OR "mold infection*" OR "mould infection*" OR outbreak OR mucor*) AND SRC:MED	Nov 9, 2026	46 (mostly unrelated)
	RE17	Europe PMC	D_R AND (TITLE:eczema OR TITLE:"atopic dermatitis") AND PUB_YEAR:[2015 TO 2026] AND SRC:MED	Nov 9, 2026	33

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	RE18	Europe PMC	(eczema OR "atopic dermatitis") AND (mould OR mold OR dampness) AND (TITLE:"meta-analysis" OR TITLE:"systematic review") AND SRC:MED	Nov 9, 2026	102 (no damp-specific eczema meta-analysis found)
	RE19	Europe PMC	(dampness OR mould OR mold OR "water damage") AND (TITLE:eye OR ocular OR "dry eye" OR conjunctiv* OR "tear film") AND SRC:MED	Nov 9, 2026	70
	RE20	Europe PMC	D_R AND (TITLE:pregnancy OR birth OR prenatal OR preterm OR fertility OR gestational) AND PUB_YEAR:[2010 TO 2026] AND SRC:MED	Nov 9, 2026	38
	RE21	Europe PMC	("indoor mold" OR "indoor mould" OR "home dampness" OR "residential dampness" OR dampness OR "visible mould" OR "visible mold") AND (TITLE:"birth weight" OR preterm OR miscarriage OR "pregnancy outcome*" OR "birth outcome*" OR preeclampsia OR infertility OR semen OR sperm OR menstrua*) AND SRC:MED	Nov 9, 2026	21 (3 relevant)
	RE22	Europe PMC	("indoor mold" OR "indoor mould" OR "home dampness" OR "residential dampness" OR "damp housing" OR "water-damaged building" OR "moisture damage") AND (cancer OR leukemia OR leukaemia OR carcinogen*) AND SRC:MED	Nov 9, 2026	4 (none epidemiological)
	RP1	PubMed	(dampness[tiab] OR "indoor mold"[tiab] OR "indoor mould"[tiab] OR "water damage"[tiab] OR "moisture damage"[tiab] OR "visible mold"[tiab] OR "visible mould"[tiab]) AND (systematic review[pt] OR meta-analysis[pt] OR "systematic review"[ti] OR "meta-analysis"[ti]) AND 2019:2026[dp]	Nov 9, 2026	55

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	RP2	PubMed	(dampness[tiab] OR "indoor mold"[tiab] OR "indoor mould"[tiab] OR "water damage"[tiab] OR "moisture damage"[tiab]) AND (eczema[ti] OR "atopic dermatitis"[ti]) AND 2015:2026[dp]	Nov 9, 2026	26
	RP3	PubMed	(dampness[tiab] OR "indoor mold"[tiab] OR "indoor mould"[tiab] OR "visible mold"[tiab] OR "visible mould"[tiab]) AND (pregnancy[ti] OR "birth weight"[ti] OR preterm[ti] OR "birth outcomes"[ti])	Nov 9, 2026	6
	RP4	PubMed	("home dampness"[tiab] OR "residential dampness"[tiab] OR "indoor mold"[tiab] OR "indoor mould"[tiab] OR "damp housing"[tiab] OR "moisture damage"[tiab] OR "household mold"[tiab] OR "residential mold"[tiab] OR "water-damaged"[tiab]) AND (cancer[tiab] OR leukemia[tiab] OR leukaemia[tiab] OR neoplasm*[tiab] OR lymphoma[tiab])	Nov 9, 2026	4 (3 AWMF guideline versions; 1 Arno-flood restoration-worker mortality cohort)
	RP5	PubMed	(hurricane[tiab] OR flood*[tiab]) AND (mold[tiab] OR mould[tiab]) AND (respiratory[tiab] OR asthma[tiab] OR symptoms[tiab]) AND 2005:2026[dp]	Nov 9, 2026	58
	RP6	PubMed	(dampness[tiab] OR "moisture damage"[tiab] OR "water damage"[tiab] OR mold[tiab] OR mould[tiab]) AND (school*[ti] OR teacher*[ti] OR office[ti] OR hospital[ti] OR workplace[ti]) AND (symptom*[tiab] OR asthma[tiab]) AND 2015:2026[dp]	Nov 9, 2026	69
	RP7	PubMed	(dampness[tiab] OR "moisture damage"[tiab] OR mold[tiab] OR mould[tiab]) AND (remediation[tiab] OR renovation[tiab]) AND (asthma[tiab] OR respiratory[tiab] OR symptoms[tiab]) AND 2015:2026[dp]	Nov 9, 2026	36

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	RP8	PubMed	(dampness[tiab] OR "indoor mold"[tiab] OR "indoor mould"[tiab] OR "water damage"[tiab]) AND (rhinosinusitis[ti] OR COPD[ti] OR "lung function"[ti] OR "respiratory infection"[ti] OR "respiratory infections"[ti]) AND 2015:2026[dp]	Nov 9, 2026	18
	RP9	PubMed	(dampness[tiab] OR mould[tiab] OR mold[tiab]) AND (eye[ti] OR ocular[ti] OR "tear film"[ti])	Nov 9, 2026	61
Gastrointestinal, microbiome, hepatic, metabolic, renal	G1	Europe PMC	(mold OR mould OR damp* OR "water-damaged" OR fungal OR Stachybotrys OR Aspergillus OR mycotoxin*) AND (microbiome OR microbiota) AND (indoor OR building OR home OR inhal* OR dampness) AND PUB_YEAR:[2024 TO 2026]	Nov 9, 2026	12,839 (Too broad; first 60 screened)
	G2	Europe PMC	ABSTRACT:("gut microbiota" OR "gut microbiome" OR "intestinal microbiota" OR "fecal microbiota" OR "faecal microbiota") AND ABSTRACT:(mold OR mould OR dampness OR "water-damaged" OR "water damaged" OR Stachybotrys) AND (indoor OR home OR house OR building OR inhal* OR dampness) AND PUB_YEAR:[2015 TO 2026]	Nov 9, 2026	73
	G3	Europe PMC	(TITLE:mold OR TITLE:mould OR TITLE:"water-damaged" OR TITLE:"damp" OR TITLE:dampness OR TITLE:Stachybotrys OR TITLE:"fungal spores" OR TITLE:"fungal exposure" OR TITLE:"indoor fungi") AND (TITLE:microbiome OR TITLE:microbiota OR TITLE:gut OR TITLE:intestinal OR TITLE:mycobiome) NOT TITLE:(phlegm OR spleen OR "dampness-heat" OR "damp-heat")	Nov 9, 2026	34
	G4	Europe PMC	("gut microbiota" OR "gut microbiome" OR "intestinal microbiota" OR "infant gut" OR "fecal microbiome" OR "stool microbiome") AND ("household mold" OR "household mould" OR "home dampness" OR "visible mold" OR "visible mould" OR "mold exposure" OR "mould exposure" OR "moisture damage" OR "water	Nov 9, 2026	154

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			damage" OR "damp housing" OR "indoor mold" OR "indoor mould" OR "mold odor" OR "dampness and mold")		
	G5	Europe PMC	(Stachybotrys OR "Aspergillus versicolor" OR "Penicillium chrysogenum" OR "fungal spores" OR "mold spores" OR "mould spores" OR "conidia" OR "fungal fragments" OR "fungal aerosol") AND (inhal* OR intranasal OR "intratracheal" OR aspiration OR aerosol) AND ("gut microbiota" OR "gut microbiome" OR "intestinal microbiota" OR "cecal" OR "fecal microbiota" OR "lung microbiome" OR "lung microbiota" OR "airway microbiome" OR "gut-lung") AND PUB_YEAR:[2015 TO 2026]	Nov 9, 2026	500
	G6	Europe PMC	(mold OR mould OR "water-damaged" OR mycotoxin*) AND (microbiome OR microbiota OR dysbiosis) AND (gut OR intestinal) AND (indoor OR building OR inhal* OR "airborne") AND PUB_YEAR:[2024 TO 2026] NOT (poultry OR broiler OR pig OR piglets OR hens OR feed OR cattle OR fish OR dampness-heat OR phlegm OR spleen)	Nov 9, 2026	372
	G7	Europe PMC	("chronic inflammatory response syndrome" OR biotoxin OR "mold-related illness" OR "mould-related illness" OR "mold illness" OR "sick building") AND (microbiome OR microbiota OR dysbiosis OR mycobiome)	Nov 9, 2026	217
	G8	Europe PMC	(AUTH:"Godoy-Vitorino F" OR AUTH:"Bolaños-Rosero B") AND (fung* OR mold OR mould OR hurricane) AND PUB_YEAR:[2019 TO 2026]	Nov 9, 2026	41
	G9	Europe PMC	(hurricane OR flood* OR flooding) AND (mold OR mould OR fung*) AND (microbiome OR microbiota OR mycobiome) AND PUB_YEAR:[2015 TO 2026]	Nov 9, 2026	3,658

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	G10	Europe PMC	(bioaerosol* OR "fungal aerosol" OR "airborne fungi" OR "fungal spores" OR "mold spores" OR "mould spores" OR Stachybotrys OR Alternaria OR Cladosporium OR "Aspergillus versicolor" OR "house dust") AND (mice OR mouse OR rats) AND ("gut microbiota" OR "gut microbiome" OR "intestinal microbiota" OR "fecal microbiota" OR "gut-lung axis") AND (inhal* OR intranasal OR exposure) AND PUB_YEAR:[2015 TO 2026]	Nov 9, 2026	2,111
	G11	Europe PMC	(TITLE:Alternaria OR TITLE:Aspergillus OR TITLE:Penicillium OR TITLE:Cladosporium OR TITLE:Stachybotrys OR TITLE:"fungal allergen" OR TITLE:"mold allergen" OR TITLE:"fungal spore") AND (TITLE:gut OR TITLE:intestin* OR TITLE:"gut-lung" OR TITLE:"fecal" OR TITLE:"lung microbiome" OR TITLE:"airway microbiome" OR TITLE:"microbiota") AND PUB_YEAR:[2018 TO 2026] NOT TITLE:(oryzae OR niger OR fermentation OR fermented OR broiler OR poultry OR plant OR soil OR rhizosphere OR invasive OR aspergillosis OR Candida)	Nov 9, 2026	55
	G12	Europe PMC	(mycotoxin* OR aflatoxin OR deoxynivalenol OR ochratoxin OR zearalenone OR fumonisin) AND ("gut microbiota" OR "gut microbiome" OR "fecal microbiota" OR "faecal microbiota" OR "stool") AND (human OR humans OR adults OR children OR infants OR participants OR volunteers OR cohort) AND (urinary OR biomarker* OR serum OR "aflatoxin-albumin") AND PUB_YEAR:[2015 TO 2026] NOT (pig* OR broiler* OR poultry OR chicken* OR hens OR cattle OR cow* OR fish OR rats OR mice)	Nov 9, 2026	311
	G13	Europe PMC	TITLE:(mycotoxin* AND ("gut microbiota" OR "gut health" OR "intestinal microbiota" OR microbiome)) AND PUB_TYPE:review AND PUB_YEAR:[2017 TO 2026]	Nov 9, 2026	10

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	G14	Europe PMC	(“household mold” OR “household mould” OR “home dampness” OR “residential dampness” OR “indoor dampness” OR “visible mold” OR “visible mould” OR “mold exposure” OR “mould exposure” OR “dampness and mold” OR “dampness and mould” OR “water-damaged” OR “water damage” OR “moisture damage” OR “damp housing” OR “mold odor” OR “mouldy”) AND (diabetes OR obesity OR “body mass index” OR overweight OR “metabolic syndrome” OR glucose OR insulin OR “kidney disease” OR renal OR “liver” OR hepatic OR “gastrointestinal” OR diarrhea OR diarrhoea OR “abdominal pain” OR nausea OR “irritable bowel” OR “inflammatory bowel” OR dyslipidemia OR cholesterol) AND PUB_YEAR:[2015 TO 2026] NOT (phlegm OR spleen OR “damp-heat” OR “dampness-heat” OR “TCM”)	Nov 9, 2026	1,662
	G15	Europe PMC	Title-restricted version of #14 (mold/damp/water damage/moisture/musty in title × diabetes/obesity/metabolic/glucose/kidney/renal/liver/ hepatic/gastrointestinal/bowel/diarrhea/inflammation in title)	Nov 9, 2026	173
	G16	Europe PMC	(“mold” OR “mould” OR “musty” OR dampness) AND (CLHLS OR CHARLS OR “Healthy Longevity” OR “NHANES” OR “UK Biobank” OR “Health and Retirement”) AND (diabetes OR hypertension OR “kidney” OR “liver” OR “multimorbidity” OR “chronic disease” OR “frailty” OR obesity OR “cardiometabolic” OR gastrointestinal OR “digestive”) AND PUB_YEAR:[2015 TO 2026]	Nov 9, 2026	394
	G17	Europe PMC	(dampness OR mold OR mould OR “moisture damage” OR “water damage” OR “indoor air”) AND (“gastrointestinal symptoms” OR “gastrointestinal complaints” OR “abdominal pain” OR “stomach” OR diarrh* OR nausea) AND (school* OR home* OR office OR building* OR occupant*) AND PUB_YEAR:[2015 TO	Nov 9, 2026	2,777

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			2026] NOT (phlegm OR spleen OR “damp-heat” OR “dampness-heat” OR food OR norovirus)		
	G18	Europe PMC	(inhal* OR intranasal OR intratracheal OR “nose-only”) AND (ochratoxin OR aflatoxin OR trichothecene* OR satratoxin OR gliotoxin OR sterigmatocystin OR deoxynivalenol OR “T-2 toxin” OR roridin OR verrucarin) AND (liver OR hepat* OR kidney OR renal OR nephro* OR systemic OR toxicokinetic* OR bioavailab* OR absorption) AND PUB_YEAR:[2010 TO 2026]	Nov 9, 2026	2,508
	G19	Europe PMC	(“house dust” OR “settled dust” OR “indoor dust” OR “indoor air” OR “building materials” OR “water-damaged”) AND (mycotoxin* OR ochratoxin OR aflatoxin OR sterigmatocystin OR trichothecene*) AND (“exposure assessment” OR “daily intake” OR “estimated daily” OR “dietary” OR “risk assessment” OR “contribution” OR “biomonitoring” OR urine OR urinary) AND PUB_YEAR:[2010 TO 2026]	Nov 9, 2026	795
	G20	Europe PMC	(Stachybotrys OR satratoxin OR “macrocyclic trichothecene*”) AND (inhal* OR intranasal OR intratracheal) AND (liver OR kidney OR serum OR plasma OR systemic OR distribution OR spleen OR thymus) AND PUB_YEAR:[2005 TO 2026]	Nov 9, 2026	326
	G21	PubMed	(damp*[tiab] OR mold[tiab] OR mould[tiab] OR “water-damaged”[tiab] OR “water damage”[tiab] OR Stachybotrys[tiab] OR mycotoxin*[tiab]) AND (microbiome[tiab] OR microbiota[tiab] OR gut[tiab] OR intestinal[tiab] OR liver[tiab] OR hepatic[tiab] OR metabolic[tiab] OR kidney[tiab]) AND (indoor[tiab] OR building*[tiab] OR home*[tiab] OR inhal*[tiab]) AND 2015:2026[dp]	Nov 9, 2026	893
	G22	PubMed	(mold[tiab] OR mould[tiab] OR “water-damaged”[tiab] OR dampness[tiab] OR Stachybotrys[tiab] OR “fungal spores”[tiab]) AND (microbiome[tiab] OR	Nov 9, 2026	29

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			microbiota[tiab] OR mycobiome[tiab]) AND (gut[tiab] OR intestinal[tiab] OR fecal[tiab] OR nasal[tiab] OR airway[tiab] OR lung[tiab]) AND (indoor[tiab] OR home*[tiab] OR house*[tiab] OR inhal*[tiab] OR hurricane[tiab] OR flood*[tiab]) AND 2015:2026[dp]		
	G23	Web search	“mold exposure gut microbiome study 2025 mice inhalation Stachybotrys OR Aspergillus”; ““mold” exposure home “gut microbiome” human study 2025 OR 2026”; ““water-damaged” building exposure microbiome dysbiosis study 2026”	Nov 9, 2026	n/a (Surfaced Nikitashina 2025 (Leibniz-HKI press release) and the Shoemaker 2025 MRA review; confirmed there is no human gut study)
Immune and inflammatory, fatigue and intolerance, endocrine and cardiovascular, HLA	IH1	PubMed	(HLA[tiab] OR HLA-DR[tiab] OR haplotype*[tiab]) AND (mold[tiab] OR mould[tiab] OR “water-damaged”[tiab] OR biotoxin*[tiab] OR “chronic inflammatory response syndrome”[tiab] OR dampness[tiab])	Nov 9, 2026	79 (all screened; mostly plant/fungal genetics, TCM, invasive mycoses)
	IH2	Europe PMC	(ABSTRACT:“HLA” OR TITLE:“HLA”) AND (ABSTRACT:“water-damaged” OR ABSTRACT:“sick building” OR ABSTRACT:“biotoxin” OR ABSTRACT:“indoor mold” OR ABSTRACT:“indoor	Nov 9, 2026	15

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			mould" OR ABSTRACT:"dampness" OR ABSTRACT:"mold exposure" OR ABSTRACT:"mould exposure" OR ABSTRACT:"chronic inflammatory response syndrome")		
	IH3	Europe PMC (full text)	"water-damaged buildings" AND ("HLA-DR" OR "HLA DR")	Nov 9, 2026	10
	IH4	Europe PMC (full text)	"chronic inflammatory response syndrome" AND ("HLA-DR" OR "HLA DR" OR "HLA")	Nov 9, 2026	10
	IH5	PubMed	("Shoemaker"[tiab] OR "chronic inflammatory response syndrome"[tiab] OR CIRS[tiab]) AND (HLA[tiab] OR haplotype[tiab]) AND (replicat*[tiab] OR validat*[tiab] OR sensitivity[tiab] OR specificity[tiab])	Nov 9, 2026	2 (neither relevant)
	IH6	PubMed	Shoemaker RC[au] OR Hudnell HK[au]	Nov 9, 2026	132 (screened; most are soybean genomics by a different R.C. Shoemaker)
	IH7	PubMed	("chronic inflammatory response syndrome"[tiab] OR "biotoxin illness"[tiab] OR "biotoxin-associated"[tiab])	Nov 9, 2026	21
	IH8	PubMed	(HLA[tiab]) AND ("multiple chemical sensitivity"[tiab] OR "environmental intolerance"[tiab] OR "sick building"[tiab] OR "building-related"[tiab])	Nov 9, 2026	2

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	IH9	PubMed	("HLA-DR"[tiab] OR "HLA-DRB1"[tiab] OR "HLA class II"[tiab]) AND ("chronic fatigue"[tiab] OR "myalgic encephalomyelitis"[tiab])	Nov 9, 2026	29
	II1	PubMed	D_I AND ("C-reactive protein"[tiab] OR CRP[tiab] OR interleukin*[tiab] OR cytokine*[tiab] OR "inflammatory marker*"[tiab] OR TNF[tiab] OR "nitric oxide"[tiab]) AND humans[mh] AND 2000:2026[dp]	Nov 9, 2026	193 (TCM-heavy)
	II2	PubMed	D_I AND ("C-reactive protein"[tiab] OR CRP[tiab] OR interleukin*[tiab] OR cytokine*[tiab] OR "inflammatory marker*"[tiab] OR TNF[tiab] OR leukocyte*[tiab] OR immunoglobulin*[tiab] OR lymphocyte*[tiab] OR "immune response*"[tiab]) AND humans[mh] AND 2010:2026[dp] X_I	Nov 9, 2026	56
	II3	PubMed	D_I AND (sarcoidosis[tiab] OR rheumat*[tiab] OR autoimmun*[tiab] OR arthritis[tiab] OR lupus[tiab] OR "multiple sclerosis"[tiab] OR "inflammatory bowel"[tiab] OR autoantibod*[tiab] OR "connective tissue"[tiab] OR vasculitis[tiab]) X_I	Nov 9, 2026	54
	II4	PubMed	sarcoidosis[ti] AND (mold[tiab] OR mould[tiab] OR mildew[tiab] OR fungal[tiab] OR fungi[tiab] OR "water damage*"[tiab] OR "water-damaged"[tiab] OR moisture[tiab] OR dampness[tiab] OR beta-glucan[tiab] OR glucan[tiab]) AND (exposure*[tiab] OR environment*[tiab])	Nov 9, 2026	32
	II5	PubMed	("mast cell*"[tiab] OR tryptase[tiab] OR histamine[tiab]) AND (dampness[tiab] OR "water-damaged"[tiab] OR "moisture damage*"[tiab] OR "indoor mold"[tiab] OR "indoor mould"[tiab] OR "mold exposure"[tiab] OR "mould exposure"[tiab] OR mycotoxin*[tiab]) AND 2010:2026[dp]	Nov 9, 2026	20 (no human exposure study)
	II6	PubMed	("beta-glucan"[tiab] OR "β-glucan"[tiab] OR "(1,3)-beta-D-glucan"[tiab] OR "1,3-β-D-glucan"[tiab] OR	Nov 9, 2026	130 (dominat

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			glucan[tiab] OR "toll-like"[tiab] OR TLR*[tiab] OR dectin*[tiab]) AND (dampness[tiab] OR "water-damaged"[tiab] OR "moisture damage*"[tiab] OR "moisture-damaged"[tiab] OR "indoor mold"[tiab] OR "indoor mould"[tiab] OR "mold exposure"[tiab] OR "mould exposure"[tiab] OR "damp"[tiab]) AND (human*[tiab] OR adult*[tiab] OR children[tiab] OR occupant*[tiab] OR worker*[tiab] OR volunteer*[tiab]) AND 2010:2026[dp] NOT (chinese[tiab] OR TCM[tiab])		ed by "DAMP" /"dampening"; the relevant human study, Punsman n 2013, came from I2)
	IF1	PubMed	("indoor air"[tiab] OR "building-related"[tiab] OR "sick building"[tiab] OR "moisture damage*"[tiab] OR dampness[tiab] OR "water-damaged"[tiab] OR "moldy building*"[tiab] OR "mouldy building*"[tiab]) AND ("idiopathic environmental intolerance"[tiab] OR "environmental intolerance"[tiab] OR "chemical intolerance"[tiab] OR "multiple chemical sensitivity"[tiab] OR "chronic fatigue"[tiab] OR "myalgic"[tiab] OR fatigue[ti] OR "persistent physical symptoms"[tiab] OR "functional somatic"[tiab] OR "nonspecific symptoms"[tiab] OR "non-specific symptoms"[tiab]) AND 2015:2026[dp] NOT (chinese[tiab] OR TCM[tiab])	Nov 9, 2026	61
	IE1	PubMed	D_I AND (thyroid[tiab] OR cortisol[tiab] OR adrenal[tiab] OR testosterone[tiab] OR estrogen*[tiab] OR oestrogen*[tiab] OR menstrua*[tiab] OR endocrin*[tiab] OR diabetes[tiab] OR insulin[tiab] OR "blood pressure"[tiab] OR hypertension[tiab] OR "heart rate"[tiab] OR cardiovascular[tiab] OR autonomic[tiab] OR orthostatic[tiab] OR POTS[tiab] OR "heart disease"[tiab] OR "coronary"[tiab]) AND humans[mh] X_I	Nov 9, 2026	32 (no epidemiologic study of these outcomes)
	IE2	PubMed	(dampness OR damp OR mold OR mould OR moisture OR "water damage*") AND (housing OR home* OR indoor OR building* OR residential) AND ("blood	Nov 9, 2026	15 (none relevant)

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			pressure" OR hypertension OR "heart rate variability" OR "cardiovascular disease*" OR "postural orthostatic" OR dysautonomia OR thyroid OR cortisol OR "sex hormone*" OR diabetes[ti] OR "metabolic syndrome") AND 2010:2026[dp] X_I NOT (food OR grain* OR feed OR maize OR zearalenone OR aflatoxin* OR fungicide* OR plant*[ti] OR "moisture content" OR skin[ti] OR wound* OR dental OR mattress*) (all [tiab])		
	IE3	Europe PMC	(ABSTRACT:"dampness" OR ABSTRACT:"water-damaged" OR ABSTRACT:"moisture damage" OR ABSTRACT:"indoor mold" OR ABSTRACT:"indoor mould" OR ABSTRACT:"visible mold" OR ABSTRACT:"visible mould") AND (ABSTRACT:"blood pressure" OR ABSTRACT:"hypertension" OR ABSTRACT:"heart rate variability" OR ABSTRACT:"cardiovascular" OR ABSTRACT:"orthostatic" OR ABSTRACT:"autonomic") AND PUB_YEAR:[2010 TO 2026] NOT ABSTRACT:"Chinese medicine"	Nov 9, 2026	40 (none relevant)
	IE4	Europe PMC	same exposure block + ABSTRACT:"mold exposure" AND (ABSTRACT:"thyroid" OR ABSTRACT:"cortisol" OR ABSTRACT:"testosterone" OR ABSTRACT:"estradiol" OR ABSTRACT:"menstrual" OR ABSTRACT:"endocrine" OR ABSTRACT:"HPA axis") AND PUB_YEAR:[2005 TO 2026] NOT ABSTRACT:"Chinese medicine"	Nov 9, 2026	36 (only Somppi 2017 and Kramer 2021 case material)
	IG1	Web	HLA-DR testing mold illness guideline position statement "not recommended" OR "no evidence" water-damaged buildings	Nov 9, 2026	led to the ACMT 2025 statement (read in full)
Mycotoxins as an indoor exposure (occurrence,	M1	Europe PMC	TITLE:(mycotoxin* OR trichothecene* OR sterigmatocystin OR "secondary metabolite*") AND TITLE:("water-damaged" OR "water damaged" OR "moisture damage*" OR damp* OR indoor OR	Nov 9, 2026	41

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biomonitoring, guidance, measured-toxin outcomes)			building* OR dust OR "house dust") AND PUB_YEAR:[2005 TO 2026] NOT TITLE:(food OR feed OR cereal OR grain OR maize OR wheat OR milk OR herbal OR "traditional chinese" OR rice OR coffee OR wine OR beer OR spice)		
	M2	Europe PMC	ABSTRACT:(mycotoxin* OR trichothecene* OR sterigmatocystin) AND ABSTRACT:("settled dust" OR "house dust" OR "indoor air" OR "building material*" OR "moisture damage*" OR "water-damaged") AND PUB_YEAR:[2010 TO 2026] NOT TITLE:(food/feed/livestock terms)	Nov 9, 2026	81
	M3	Europe PMC	(mycotoxin* OR trichothecene* OR sterigmatocystin OR ochratoxin) AND (urine OR urinary OR serum OR biomarker* OR biomonitoring) AND ("water-damaged" OR "moisture damage*" OR "damp building*" OR "mold-damaged" OR "mould-damaged" OR "indoor mold" OR "indoor mould" OR "moldy building*" OR "mouldy building*") AND PUB_YEAR:[2005 TO 2026]	Nov 9, 2026	179 (mostly full-text noise)
	M4	PubMed	(mycotoxin*[tiab] OR trichothecene*[tiab] OR ochratoxin[tiab] OR sterigmatocystin[tiab]) AND (urine[tiab] OR urinary[tiab] OR serum[tiab] OR biomonitoring[tiab] OR biomarker*[tiab]) AND ("water-damaged"[tiab] OR "moisture damage"[tiab] OR "moisture-damaged"[tiab] OR damp*[tiab] OR "indoor"[tiab]) AND 2005:2026[dp]	Nov 9, 2026	29
	M5	Europe PMC	(mycotoxin* OR sterigmatocystin OR verrucarol OR trichothecene*) AND (asthma OR symptom* ...) AND (dust OR "indoor air" OR classroom* OR school* OR home*) AND (measured OR LC-MS ...)	Nov 9, 2026	5,210 (too broad; discarded)
	M6	PubMed	(mycotoxin*[tiab] OR sterigmatocystin[tiab] OR verrucarol[tiab] OR trichothecene*[tiab] OR "secondary metabolite*"[tiab]) AND (asthma[tiab] OR symptom*[tiab] OR wheez*[tiab] OR rhinitis[tiab] OR	Nov 9, 2026	69

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			"sick building"[tiab] OR health[ti]) AND (dust[tiab] OR "indoor air"[tiab] OR classroom*[tiab] OR school*[tiab] OR dwelling*[tiab] OR "water-damaged"[tiab] OR "moisture damage*"[tiab]) AND 2005:2026[dp] NOT (food/feed/grain terms [ti])		
	M7	PubMed	(sterigmatocystin[tiab] OR verrucarol[tiab] OR mycotoxin*[tiab] OR "fungal metabolite*"[tiab] OR "microbial metabolite*"[tiab]) AND dust[tiab] AND (asthma[tiab] OR wheez*[tiab] OR rhinitis[tiab] OR symptom*[tiab] OR eczema[tiab] OR "lung function"[tiab]) AND (children[tiab] OR pupils[tiab] OR students[tiab] OR adults[tiab] OR occupants[tiab]) AND 2005:2026[dp]	Nov 9, 2026	7
	M8	PubMed	Peitzsch M[au] AND (mycotoxin* OR metabolite*)	Nov 9, 2026	42
	M9	PubMed	Vishwanath V[au] AND metabolite*	Nov 9, 2026	4
	M10	PubMed	Hintikka EL[au]	Nov 9, 2026	15
	M11	PubMed	Nevalainen A[au] AND "Indoor Air"[ta] AND 2015[dp]	Nov 9, 2026	n/a (rate-limit error; record located by author search)
	M12	PubMed	Sulyok M[au] AND (dust[tiab] OR indoor[tiab] OR building*[tiab])	Nov 9, 2026	16
	M13	PubMed	Täubel M[au] AND (metabolite* OR mycotoxin*)	Nov 9, 2026	11
	M14	PubMed	(mold[ti] OR mould[ti] OR molds[ti] OR fungi[ti] OR fungal[ti]) AND ("practice parameter"[ti] OR position[ti] OR statement[ti] OR "work group report"[ti] OR guideline*[ti] OR "technical report"[ti] OR "clinical	Nov 9, 2026	7

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			report"[ti]) AND (indoor[tiab] OR damp*[tiab] OR building*[tiab] OR home*[tiab]) AND 2005:2026[dp]		
	M15	PubMed	Bush RK[au] AND Portnoy JM[au] AND mold	Nov 9, 2026	2
	M16	PubMed	"spectrum of noninfectious health effects from molds"	Nov 9, 2026	4
	M17	Europe PMC	TITLE:("practice parameter" OR "position paper" OR "position statement" OR "work group report" OR "clinical report") AND TITLE:(mold OR mould OR molds OR fungal OR mycotoxin*) AND PUB_YEAR:[2005 TO 2026]	Nov 9, 2026	10
	M18	Europe PMC	TITLE:"mold prevention strategies" AND hurricanes	Nov 9, 2026	1
	M19	Web (canada.ca)	Health Canada Residential Indoor Air Quality Guideline: Moulds	Nov 9, 2026	1 page
	M20	Web (anses.fr + report PDF)	ANSES "Moisissures dans le bâti" 2016 (saisine 2014-SA-0016)	Nov 9, 2026	1 report
	M21	Web (cdc.gov)	CDC mold "About" page	Nov 9, 2026	1 page
	M22	Europe PMC	(mycotoxin* OR "secondary metabolite*") AND ("house dust" OR "settled dust" OR "indoor dust") AND ("daily intake" OR "exposure assessment" OR "risk assessment" OR dietary OR tolerable) AND PUB_YEAR:[2008 TO 2026]	Nov 9, 2026	223
	M23	Europe PMC	TITLE:(mycotoxin* OR ochratoxin OR trichothecene* OR sterigmatocystin OR roridin OR satratoxin) AND TITLE:(urine OR urinary OR serum OR blood OR "human samples" OR biomarker* OR "breast milk" OR tissue* OR patients OR residents OR occupants) AND	Nov 9, 2026	40

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			(mold OR mould OR "water-damaged" OR indoor OR damp*) AND PUB_YEAR:[2005 TO 2026]		
	M24	Europe PMC	TITLE:(mycotoxin* OR "fungal toxins" OR "fungal metabolites" OR "secondary metabolites") AND TITLE:(indoor OR building* OR dwelling* OR damp* OR "water-damaged" OR inhal* OR airborne) AND (review) AND PUB_YEAR:[2012 TO 2026]	Nov 9, 2026	7
	M25	PubMed	(mycotoxin*[tiab]) AND (urine[tiab] OR urinary[tiab]) AND (commercial[tiab] OR "direct-to-consumer"[tiab] OR unvalidated[tiab] OR "clinical laborator*"[tiab] OR "functional medicine"[tiab] OR "mold illness"[tiab] OR "mould illness"[tiab] OR "chronic fatigue"[tiab] OR "mold toxicity"[tiab]) AND 2010:2026[dp]	Nov 9, 2026	13
	M26	PubMed	(satratoxin*[tiab] OR "macrocyclic trichothecene*"[tiab] OR roridin*[tiab] OR phenylspirodrimane*[tiab] OR sterigmatocystin[tiab] OR "mycophenolic acid"[tiab] OR chaetoglobosin*[tiab]) AND (urine[tiab] OR urinary[tiab] OR serum[tiab] OR plasma[tiab]) AND human*[tiab] AND 2005:2026[dp]	Nov 9, 2026	161
Agents other than fungi (endotoxin, bacteria, glucans and fragments, MVOCs, microbiome, chemical emissions, multi-agent studies)	AEN 1	PubMed	endotoxin[tiab] AND (dampness[tiab] OR "water damage*"[tiab] OR "moisture damage*"[tiab] OR mold[tiab] OR mould[tiab]) AND (home*[tiab] OR house*[tiab] OR school*[tiab] OR building*[tiab] OR dwelling*[tiab]) AND 2005:2026[dp]	Nov 9, 2026	68 (top 30 screened)

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	AEN 2	PubMed	endotoxin[tiab] AND (asthma[tiab] OR wheez*[tiab]) AND ("house dust"[tiab] OR household[tiab] OR home*[tiab] OR school*[tiab]) AND humans[mh] AND 2005:2026[dp]	Nov 9, 2026	178 (top 30 screened)
	AEN 3	PubMed	endotoxin[ti] AND (asthma[tiab] OR wheez*[tiab]) AND (meta-analysis[pt] OR review[pt] OR "systematic review"[tiab])	Nov 9, 2026	24 (all screened)
	AEN 4	Europe PMC	TITLE:"endotoxin" AND (ABSTRACT:"asthma" OR ABSTRACT:"wheeze") AND (ABSTRACT:"house dust" OR ABSTRACT:"homes" OR ABSTRACT:"school") AND PUB_YEAR:[2005 TO 2026]	Nov 9, 2026	71 (top 30 screened)
	AM U1	PubMed	("muramic acid"[tiab] OR "peptidoglycan"[tiab]) AND ("house dust"[tiab] OR school*[tiab] OR home*[tiab]) AND (asthma[tiab] OR wheez*[tiab] OR atopy[tiab] OR symptom*[tiab])	Nov 9, 2026	31 (all screened)
	AA C1	PubMed	(actinomycet*[tiab] OR streptomyces[tiab] OR mycobacteri*[tiab]) AND ("moisture damage*"[tiab] OR "moisture-damaged"[tiab] OR "water-damaged"[tiab] OR "water damage*"[tiab] OR dampness[tiab] OR "indoor air"[tiab] OR "house dust"[tiab]) AND 2005:2026[dp]	Nov 9, 2026	84 (top 30 screened)
	AA C2	Europe PMC	(ABSTRACT:"valinomycin" OR ABSTRACT:"ionophore" OR ABSTRACT:"streptomyces" OR ABSTRACT:"actinomycetes") AND (ABSTRACT:"indoor" OR ABSTRACT:"moisture-damaged" OR ABSTRACT:"water-damaged" OR ABSTRACT:"building") AND PUB_YEAR:[2005 TO 2026]	Nov 9, 2026	263 (top 30 screened) (too broad)
	AA C3	PubMed	(valinomycin[tiab] OR ionophor*[tiab] OR "Streptomyces"[tiab]) AND (indoor[tiab] OR building*[tiab] OR "moisture"[tiab]) AND (toxic*[tiab]	Nov 9, 2026	31 (all screened)

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			OR cytotox*[tiab] OR inflammat*[tiab]) AND 2005:2026[dp]		
	AAC4	PubMed	("Streptomyces"[tiab] OR "actinobacteria"[tiab] OR mycobacteri*[tiab]) AND ("moisture damage*"[tiab] OR "moisture-damaged"[tiab] OR "water-damaged"[tiab]) AND (asthma[tiab] OR symptom*[tiab] OR health[tiab] OR inflammat*[tiab]) AND 2005:2026[dp]	Nov 9, 2026	18 (all screened)
	AGL1	PubMed	("glucan*"[tiab]) AND ("house dust"[tiab] OR "indoor"[tiab] OR school*[tiab] OR office*[tiab] OR dwelling*[tiab]) AND (asthma[tiab] OR wheez*[tiab] OR symptom*[tiab] OR atopy[tiab] OR "lung function"[tiab] OR inflammat*[tiab]) AND humans[mh] AND 2005:2026[dp]	Nov 9, 2026	58 (top 30 screened)
	AFR1	PubMed	("fungal fragment*"[tiab] OR "submicron*"[tiab] OR "hyphal fragment*"[tiab]) AND (fung*[tiab] OR mold[tiab] OR mould[tiab]) AND (indoor[tiab] OR building*[tiab] OR home*[tiab]) AND 2005:2026[dp]	Nov 9, 2026	36 (all screened)
	AMV1	PubMed	("microbial volatile organic compound*"[tiab] OR MVOC*[tiab] OR "1-octen-3-ol"[tiab]) AND (human*[tiab] OR symptom*[tiab] OR asthma[tiab] OR exposure[tiab]) AND 2005:2026[dp]	Nov 9, 2026	247 (top 30 screened) (insect-olfaction noise; replaced by AMV2 and AX2)
	AMV2	Europe PMC	(ABSTRACT:"microbial volatile organic" OR ABSTRACT:"MVOC" OR ABSTRACT:"1-octen-3-ol") AND (ABSTRACT:"indoor" OR ABSTRACT:"dwelling" OR ABSTRACT:"building") AND (ABSTRACT:"symptom" OR ABSTRACT:"asthma" OR ABSTRACT:"exposure	Nov 9, 2026	13 (all screened)

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			chamber" OR ABSTRACT:"volunteers") AND PUB_YEAR:[2005 TO 2026]		
	AC H1	PubMed	("2-ethyl-1-hexanol"[tiab] OR "2-ethylhexanol"[tiab] OR "2-ethyl hexanol"[tiab]) AND (indoor[tiab] OR building*[tiab] OR floor*[tiab] OR symptom*[tiab]) AND 2005:2026[dp]	Nov 9, 2026	51 (top 30 screened)
	AC H2	PubMed	(dampness[tiab] OR "moisture damage*"[tiab] OR "water damage*"[tiab] OR "damp concrete"[tiab] OR "floor dampness"[tiab]) AND (plasticizer*[tiab] OR phthalate*[tiab] OR DEHP[tiab] OR formaldehyde[tiab] OR ammonia[tiab] OR "volatile organic"[tiab] OR VOC*[tiab]) AND (asthma[tiab] OR symptom*[tiab] OR "sick building"[tiab] OR health[tiab]) AND 2005:2026[dp]	Nov 9, 2026	53 (top 30 screened)
	AM B1	PubMed	(microbiota[tiab] OR microbiome[tiab] OR "microbial diversity"[tiab] OR "bacterial diversity"[tiab] OR "fungal diversity"[tiab]) AND ("house dust"[tiab] OR "indoor dust"[tiab] OR "floor dust"[tiab] OR classroom*[tiab] OR "indoor microbi*"[tiab]) AND (asthma[tiab] OR wheez*[tiab] OR atopy[tiab] OR allerg*[tiab]) AND 2005:2026[dp]	Nov 9, 2026	194 (top 25 screened)
	AM B2	PubMed	(microbiota[tiab] OR microbiome[tiab] OR "microbial communit*"[tiab] OR sequencing[tiab]) AND ("moisture damage*"[tiab] OR "moisture-damaged"[tiab] OR dampness[tiab] OR "water-damaged"[tiab] OR "water damage*"[tiab]) AND (home*[tiab] OR house*[tiab] OR school*[tiab] OR building*[tiab]) AND 2005:2026[dp] NOT (chinese[tiab] OR TCM[tiab])	Nov 9, 2026	45 (top 25 screened)
	AFA 1	Europe PMC	(TITLE:"farm" OR TITLE:"Amish" OR TITLE:"microbiota" OR TITLE:"microbial exposure" OR TITLE:"microbial diversity") AND (TITLE:"asthma" OR TITLE:"allergy" OR	Nov 9, 2026	23 (all screened)

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			TITLE:“atopy”) AND (ABSTRACT:“house dust” OR ABSTRACT:“indoor”) AND PUB_YEAR:[2010 TO 2026]		
	ACC 1	PubMed	(“chamber”[tiab] OR “controlled exposure”[tiab] OR “double-blind”[tiab] OR “experimental exposure”[tiab]) AND (mold[tiab] OR mould[tiab] OR fungal[tiab] OR glucan[tiab] OR “microbial volatile”[tiab] OR “1-octen-3-ol”[tiab] OR “damp building material*”[tiab]) AND (volunteer*[tiab] OR subjects[tiab] OR human*[tiab]) AND (symptom*[tiab] OR “nasal”[tiab] OR “lung function”[tiab]) AND 2005:2026[dp]	Nov 9, 2026	35 (all screened)
	AX2	PubMed	(“1-octen-3-ol”[tiab] OR “2-ethyl-1-hexanol”[tiab] OR “3-methyl-1-butanol”[tiab]) AND (chamber[tiab] OR “experimental”[tiab] OR volunteers[tiab]) AND humans[mh]	Nov 9, 2026	22 (all screened)
	AX4	PubMed	Claeson AS[au] AND (damp[tiab] OR “building material*”[tiab] OR microbial[tiab]) AND 2005:2026[dp]	Nov 9, 2026	2
	AS M1	PubMed	(endotoxin[tiab] OR glucan[tiab] OR ergosterol[tiab] OR “muramic acid”[tiab] OR “extracellular polysaccharide*”[tiab]) AND (fung*[tiab] OR mold[tiab] OR mould[tiab]) AND (dampness[tiab] OR “moisture damage*”[tiab] OR “water-damaged”[tiab] OR “water damage*”[tiab] OR “damp”[tiab]) AND (symptom*[tiab] OR asthma[tiab] OR wheez*[tiab] OR “lung function”[tiab]) AND 2005:2026[dp]	Nov 9, 2026	32 (all screened)
	AX5	PubMed	(HITEA[tiab]) AND (endotoxin[tiab] OR microbial[tiab] OR glucan[tiab] OR schools[tiab])	Nov 9, 2026	11 (all screened)
	AX6	PubMed	(endotoxin[tiab] OR “Gram-negative”[tiab]) AND (dampness[tiab] OR “moisture damage*”[tiab] OR “water damage*”[tiab]) AND (adult*[tiab] OR worker*[tiab] OR occupant*[tiab] OR office*[tiab]) AND 2005:2026[dp]	Nov 9, 2026	15 (all screened)

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	AX1	PubMed	Dannemiller KC[au] AND (asthma[tiab] OR diversity[tiab])	Nov 9, 2026	20
	AX3	PubMed	Sahlberg B[au] AND (formaldehyde[tiab] OR "sick building"[tiab])	Nov 9, 2026	5

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