



## **Defeating Class Certification In Single-Building / Multiple-Occupant Mold Exposure Cases**

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**Commentary*****Defeating Class Certification In Single-Building / Multiple-Occupant Mold Exposure Cases***

By  
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People have lived with mold as long as they have lived indoors. Until the mid-1990's, the close and constant association between people and indoor mold spawned only a small number of lawsuits.<sup>1</sup> In the late 1990's, however, Americans lost their ability to live non-litigiously with this ubiquitous organism. Although there has been neither a demonstrable increase in the quantity of mold,<sup>2</sup> nor any significant change in the scientific evidence related to the health effects of mold, there *has* been an explosion of mold litigation.<sup>3</sup> The plaintiff's bar has recently, and unsurprisingly, attempted to push mold litigation into the class action context by suing on behalf of all residents or tenants of multiple-occupant buildings that have a pervasive mold problem. Given the ubiquity of mold and the enormous number of large multiple-occupant buildings in the United States, the potential scope of single-building/multiple-occupant mold exposure litigation is breathtaking.

This article details some of the arguments that defense counsel should raise against certification of single-building/multiple-occupant mold exposure class actions. Part A gives a brief account of the history of mold litigation, explaining why it has become so pervasive. Part B provides an introduction to the science of mold and its potential health effects. Part C expands on the particular aspects of that science which raise individualized issues of proof and thus defeat the predominance and superiority requirements for certification of a class. Part D argues that, independent of the rules governing class actions, a mold-exposure class would violate the due process clause. Finally, Part E suggests certain analogies from other "toxic exposure" cases that may be useful in defeating class certification.

**A. *The Growth Of Mold Litigation***

The recent explosion in mold litigation was caused by a combination of misleading medical reports, media hype, and the possibility of large verdicts in what promised to be an enormous number of mold cases. "The current concern about toxic mold really started several years ago after federal health officials blamed mold in dilapidated homes in Cleveland for a cluster of 10 cases of *pulmonary hemorrhage* (bleeding in the lungs). But the report was later retracted when outside reviewers found critical mistakes in the investigation."<sup>4</sup> Although the Cleveland report

was retracted, the popular media became infatuated with the possibility that mold, an organism we all live with, might be dangerous or even deadly.<sup>5</sup> This media attention — along with a few large high-profile verdicts — sparked an epidemic of mold-litigation that has swept across the country.<sup>6</sup> Today, “it is almost impossible to follow local or national news without learning about new mold claims being made by building occupants including employees, homeowners, apartment tenants, parents, and school children.”<sup>7</sup>

This public hysteria has also caused insurance claims relating to indoor mold to skyrocket from \$200,000,000 in 2000 to \$2,500,000,000 in 2002.<sup>8</sup> This precipitous rise in the number of claims is “a key reason [that insurers] are charging more for insurance policies and halting coverage of homeowners who have filed water damage claims.”<sup>9</sup> Indeed, in thirty-five states insurers have successfully lobbied to exclude mold claims from insurance policies unless the damage results from another covered loss.<sup>10</sup> The situation is so bad that many building owners are finding it difficult to obtain any insurance for mold exposure claims.<sup>11</sup>

Until now, courts have confined the epidemic of mold litigation to traditional single-plaintiff lawsuits. Attempts to certify single-building/multiple-occupant classes have been rejected by several courts. For example, a New York trial court refused to certify a class of “[a]ll . . . residents . . . at Phipps Plazas, \* \* \* who have suffered personal injuries and/or emotional distress as a result of exposure to various chemicals, fungi, mycotoxins, bacteria, construction debris and other toxic substances due to the defendants’ negligence.”<sup>12</sup> The court held that “[w]hile some factual issues could perhaps be resolved in a class action format, these issues are thoroughly intertwined with those that must be determined individually,” such as “[t]he specific conditions which subjected individual Plaintiffs to mold exposure,” “[d]efendants’ repair and remediation efforts,” and “causation and \* \* \* comparative negligence.”<sup>13</sup>

The California Court of Appeals has reached the same conclusion.<sup>14</sup> Plaintiffs sought certification of a class of “all persons who were, or are, residents of the apartment units owned by AVALONBAY COMMUNITIES, INC. \* \* \* who have been exposed to toxic materials in the apartments including, without limitation, to bioaerosols emanating from excessive levels of mold, mildew and fungus and/or pesticides and/or other chemicals.”<sup>15</sup> The appellate court quoted the trial court’s order denying class certification with approval, finding that “each purported class member would have to prove exposure to a particular toxic substance, the timing of such exposure, and the effect of such exposure; Defendants would legitimately seek to present evidence of exposure other than at the apartment complex for each class member.”<sup>16</sup>

The reasoning and conclusions of these decisions are clearly correct. Due to the nature of such claims and the characteristics of mold, individual issues must predominate over any potential common issues in a purported single-building/multiple-occupant class action seeking damages for exposure to mold. These cases also show that courts are able to see through the superficial appeal of a class based on “common” exposure to mold in a single building and recognize that, taking into account the actual science of mold, mold-exposure claims simply cannot be litigated on a class wide basis.

## **B. The Growth Of Mold**

Molds are naturally occurring members of the fungi kingdom. They reproduce and spread through the distribution of spores. An individual mold spore can enter a building in many ways, including through open doors or windows or by becoming attached to pets, clothes, or other personal items that enter the building.<sup>17</sup> In order to begin germinating, a mold spore needs only an appropriate nutrient base (*e.g.*, wood, paper, cloth, or dust) and moisture.<sup>18</sup> Some species of mold require a constant source of standing water while others can thrive on just the humidity in the air.<sup>19</sup> Once they have a nutrient base and water, the spores of some species of mold can produce a mold colony, growing at an exponential rate, within 24 to 48 hours.<sup>20</sup>

Because molds spread so rapidly and require so little to thrive, “[they] are ubiquitous in nature and grow almost anywhere indoors and outdoors.”<sup>21</sup> “In the natural environment man is exposed to more than 100 species of airborne or dust-bound microfungi. Fungal spore counts frequently exceed pollen counts in the atmosphere by 1000-fold.”<sup>22</sup> Not only are they ubiquitous in nature generally, but “[m]olds are very common in buildings and homes and will grow anywhere indoors where there is moisture.”<sup>23</sup>

“Exposure to molds \* \* \* and their spores is unavoidable except when the most stringent of air filtration, isolation, and environmental sanitation measures are observed, eg, [sic] in organ transplant isolation units.”<sup>24</sup> Exposure to mold can occur through physical contact with mold growth on a surface, inhalation of mold spores and airborne fragments, or ingestion of mold on food. In addition, some molds produce microbial volatile organic compounds (“MVOCs”), which become airborne and account for the unpleasant odors and tastes associated with some mold.<sup>25</sup> Under certain conditions, some species of mold will also produce secondary metabolites known as mycotoxins.<sup>26</sup> Mycotoxins are large molecules that do not become airborne except when attached to a mold spore or particle that is airborne.<sup>27</sup>

Possible health effects associated with mold exposure generally fall into one of three categories: allergic, infectious, or toxic.<sup>28</sup> First, a small but significant percentage of people suffer from mold allergies.<sup>29</sup> Second, certain immunocompromised individuals may suffer opportunistic mold infections, usually in their lungs.<sup>30</sup> Finally, *ingestion* of certain molds and mycotoxins growing on foods can produce a toxic effect.<sup>31</sup> The effect can be quite serious, ranging from headache and nausea to death.<sup>32</sup> Despite media hype, however, there is no scientific proof that exposure to indoor mold through *physical contact* or *inhalation* produces a similar toxic health effect.<sup>33</sup> Some plaintiffs’ experts have argued that certain animal studies establish the toxic effect of low-level chronic inhalation of indoor mold. However, the scientific community has found these studies lacking in scientific merit and the causal connection unproven.<sup>34</sup>

Although mold does cause allergic reactions or infections in some people, “[i]t is not known \* \* \* what quantity of mold is acceptable in indoor environments with respect to health.”<sup>35</sup> “[B]ecause individuals have different sensitivities to molds, setting standards and guidelines for indoor mold exposure levels is difficult and may not be practical.”<sup>36</sup> Indeed, each case of an alleged mold-related health effect is fundamentally individualized.

### **C. Individualized Issues**

Proof of a mold exposure claim will necessarily be dominated by individualized inquiries. Even if the plaintiffs were exposed to mold under laboratory-like conditions, the central questions of causation — whether and to what extent a standardized dose of mold affected each of them — would demand a detailed inquiry into each plaintiff’s medical history, lifestyle, and environment. The individuality of each claim is even greater in real life, where the quantity and type of mold, the duration and method of exposure, and the cause of the mold growth itself are different in each case. The following sections describe various characteristics of mold and indoor mold exposure that serve to make mold-exposure claims “uniquely individualized” and hence to preclude class-wide proof in any single-building/multiple-occupant class action.<sup>37</sup>

#### **1. The Amount And Type Of Mold Present In A Building Unit And The Causes Of That Mold Growth Vary From Unit To Unit**

Although mold is present everywhere, the extent and type of mold growth within a building can vary dramatically between units and even from room to room. The species of mold in a unit will depend on which spores entered, for example, when a door or window was left open or by becoming attached to clothing or pets.<sup>38</sup> The *extent* of mold growth will depend on environmental factors such as humidity, temperature, airflow, and nutrient base; whether the mold has been left alone or disturbed; and whether any chemicals such as cleaning agents have been applied to the mold.<sup>39</sup>

Thus, determining the type and extent of mold growth in a particular unit requires a first-hand investigation.

Furthermore, many of the factors that affect mold growth in building units depend on choices made by the occupants: presence and type of carpeting; type and positioning of furniture; presence, type and cleanliness of pets; temperature; use of air conditioning or alternate ventilation systems; whether and how often doors or windows are left open; cleanliness habits of the occupants; and any alteration of the airflow in the unit (caused by, *e.g.*, closed vents or general clutter).<sup>40</sup> There is no single cause of mold growth in most units, but rather a large set of contributing factors. Thus, establishing the causes of mold growth in a unit requires a detailed individualized investigation of the conditions present in that unit and the behavior of its occupants.

## **2. The Existence, Amount, And Type Of Mycotoxins Produced Are Idiosyncratic To Each Instance Of Mold Growth**

Only a select group of molds produce mycotoxins.<sup>41</sup> Moreover, “[m]ycotoxin production for a given species is highly dependent on growth conditions, such as nutrient availability, temperature, and humidity.”<sup>42</sup> For example, “[a]flatoxin production by *Aspergillus* is dependent on concentrations of O<sub>2</sub>, CO<sub>2</sub>, zinc, and copper, as well as physical location.”<sup>43</sup> Thus, this toxin is not normally produced by mold growing on building materials.<sup>44</sup> “Additionally, even under the *same* conditions of growth, the profile and quantity of mycotoxins produced by toxigenic species can vary widely from one isolate to another.”<sup>45</sup> “[I]t does not necessarily follow from the mere presence of a toxigenic species that mycotoxins are also present.”<sup>46</sup> Because there are no known principles of mycotoxin production (even for individual species), proof of the amount (or even the presence) of mycotoxins in a particular unit requires a first-hand, particularized, investigation into the actual conditions in that unit.

## **3. The Exposure Of Building Occupants To Mold And / Or Mycotoxins — Even When They Are Present — Depends On The Specific Environment And Activities Of The Occupants**

The primary method of exposure to indoor molds and mycotoxins is inhalation.<sup>47</sup> The amount of mold spores and fragments in the air depends on many environmental factors, including the growth substrate, ventilation and general airflow, and physical disturbance.<sup>48</sup> Less obviously, differences in “carpeting type, pets, dust control measures, and humidification” affect airborne spore counts.<sup>49</sup> Thus, even if an apartment has an elevated level of mold growth, it will not necessarily have an elevated level of airborne spores and fragments.<sup>50</sup> Also important to each occupant’s actual exposure level is the amount of time he or she spends in the particular unit. Finally, the timeline of remediation efforts will affect the actual exposure levels of occupants. For all of these reasons, even if two units had identical mold growth, the occupants would likely have different exposure levels, and the actual levels would not be ascertainable except by studying the particular conditions of each case.

## **4. There Is No Common Effect Of Mold Exposure: Some People Have No Reaction To Even Elevated Levels While Others Are Hypersensitive To Small Doses**

“Exposure to mold does not always result in a health problem.”<sup>51</sup> Studies indicate that approximately 10% of the population have allergic antibodies to common molds, and that 5% are actually sensitized to mold and thus will suffer allergic symptoms from exposure.<sup>52</sup> Thus, 90-95% of people will not have an allergic response to mold in their apartments. Furthermore, the sensitivity levels of the 5-10% of people who are allergic to mold vary dramatically.<sup>53</sup> “What one person can tolerate with little or no effect, may produce symptoms in another similarly exposed individual. In fact, the reaction to both the amount and types of mold varies from one person to the next.”<sup>54</sup> Because each person reacts (or doesn’t react) to mold differently, “[s]tandards for judging what is an acceptable, tolerable, or normal quantity of mold have not been established.”<sup>55</sup>

For those who do suffer from mold allergies, the symptoms caused by exposure “are most commonly experienced as allergic asthma or allergic rhinitis (‘hay fever’).”<sup>56</sup> A person with mold-induced “hay fever” may have only a mild runny nose or may experience itching/watering eyes, general congestion, atopic dermatitis, or asthma symptoms.<sup>57</sup> Although some individuals’ symptoms get worse if they are exposed to a greater quantity of mold, for others, mold-allergy symptoms are not directly correlated with the amount of exposure.<sup>58</sup> And “[allergic] reaction is highly specific[;]” an individual may experience allergies from one species, but have no reaction to even a closely related species.<sup>59</sup> The severity of an individual’s allergic reaction to a given species and quantity of mold (and whether there will be one at all) cannot be known without an individualized analysis of his or her allergic sensitivities.

**5. *Symptoms Associated With Mold Allergies Are Non-specific And May Be Caused By Many Environmental And Health Factors.***

The allergic symptoms commonly caused by mold “are very nonspecific and may be related to exposure to other sources (such as dust mites, animal dander, pollen or other allergens) or to infectious agents such as viruses that cause common colds or flu.”<sup>60</sup> In general, moreover, people who exhibit allergic responses to mold are also allergic to other environmental allergens.<sup>61</sup> The actual cause of an individual’s allergic symptoms, therefore, cannot be attributed to mold in a particular building unit (or to mold at all) without a medical analysis of his or her allergic sensitivities, other health conditions, and other exposures.

**6. *Allergic Reactions To Mold May Be Exacerbated By A Person’s Behavior, Work, Or Other Factors***

Sensitivity to mold can be heightened, and symptoms of mold allergies can be exacerbated, by many factors, including other allergies, exposure to certain chemicals or biological agents, and various health-related lifestyle choices. For example, “marijuana smoking may lead to development of fungal hypersensitivity reactions,” and smoking in general will make allergic symptoms worse (especially respiratory symptoms).<sup>62</sup> A person’s work may also cause or exacerbate allergic symptoms.<sup>63</sup> Finally, other general health conditions, including “mental stress,” are correlated with higher levels of allergic symptoms.<sup>64</sup> Even if a person is experiencing an allergic reaction to mold in a particular building unit, an individualized analysis may reveal that other factors are contributing to the symptoms or that the person is responsible for exacerbating his or her condition.

**7. *Individuals Who Suffer From Asthma May Experience Varying Responses To Mold In The Air, But Asthmatic Symptoms Can Be Caused Or Exacerbated By Many Factors***

“Molds can trigger asthma attacks in persons who are allergic (sensitized) to molds. The irritants produced by molds may also worsen asthma in non-allergic (non-sensitized) people.”<sup>65</sup> However, molds do not cause asthma to develop: they only exacerbate symptoms in people who already suffer from asthma.<sup>66</sup> Therefore, most individuals will not have an asthmatic response to inhaled mold because they are not asthmatic.<sup>67</sup> Among asthmatic individuals, “[t]he types and severity of symptoms depend, in part, on the types of mold present, the extent of an individual’s exposure, the ages of the individuals, and their existing sensitivities or allergies.”<sup>68</sup>

Much like general allergic symptoms, asthma may be caused or exacerbated by many environmental factors. Animal dander, dust mites, pollens, tobacco smoke, air pollution, perfumes, and changes in humidity can all be “triggers” for asthma.<sup>69</sup> Other individuals may suffer asthma symptoms in response to aspirin, sulfites, or beta-blockers.<sup>70</sup> And “the most common cause of asthma symptoms” is “viral respiratory infection.”<sup>71</sup> The causes of an individual’s asthma symptoms (and any exacerbating effects) cannot be determined without an individual medical evaluation.

## **8. Individual Testing Is Necessary To Identify The Source Of A Mold Infection**

Certain species of mold (*e.g.*, *Aspergillus fumigatus*) may infect the lungs or other opportunistic sites within individuals who have weakened immune systems.<sup>72</sup> Because a mold infection involves the growth of a particular species of mold in or on the human body, potential sources of the infection can be either confirmed or ruled out through individual testing.<sup>73</sup> Although it may be impossible to determine the actual source of the infection, it is possible to rule out certain potential sources, such as a particular building unit, if the relevant species is not found there. Thus, individual testing is required to identify the cause of a mold infection.

## **9. Because There Is No Proven Link Between Indoor Mold Exposure And Health Effects Other Than Exacerbation Of Existing Allergies And Asthma, To The Extent Any Individual Seeks To Recover For Any Other Health Effects, There Is No Conceivable Way In Which Causation Could Be Established On A Class-wide Basis**

Despite widespread media hype over “toxic mold,” scientific study has not established any connection between exposure to indoor mold and any effect other than allergies, asthma, or infections.<sup>74</sup> Therefore, defense counsel can argue that a toxic effect from inhalation of indoor mold cannot be proved — even in an individual suit — given the current state of the science. More to the point here, any attempt to prove such harm cannot possibly be accomplished on a class-wide basis. Among many potential subjects of individualized proof, there would have to be evidence that the individual was susceptible to the mycotoxin in question, proof that the mycotoxin was actually present in the apartment and that the individual was exposed to a dose (whether low-level chronic or a single large dose) exceeding his or her tolerance level, proof that this particular dose of inhaled mycotoxins can cause certain symptoms, proof that the individual suffered those symptoms, and proof that the individual’s symptoms do not have another scientifically recognized cause.

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In sum, these and other individual factual issues would inevitably swamp any common ones in a single-building/multiple-occupant mold-exposure case, making class status improper under the predominance and superiority requirements of Rule 23(b)(3) of the Federal Rules of Civil Procedure, or its state law counterparts.<sup>75</sup> Defense counsel will obviously strengthen their opposition to class certification if they can develop concrete examples of these scientific issues in their particular cases (*e.g.*, evidence that different building units have different quantities and/or types of mold growth and different plaintiffs have different allergic sensitivities and medical histories).

## **D. Due Process**

Even setting aside the specific requirements of the class action device, every lawsuit — including class actions — must conform with the constitutional requirements of due process under either the Fifth or the Fourteenth Amendments.<sup>76</sup> The Supreme Court has made clear that Rule 23(b)(3)’s manageability test is satisfied only if trial of the case as a class action can be accomplished “without sacrificing procedural fairness” and without “abridg[ing], enlarg[ing] or modify[ing] any substantive right.”<sup>77</sup> “What this means, as a practical matter, is that, in an effort to achieve manageability, courts may not relieve plaintiffs of the burden of proving the individualized elements of their claims \* \* \* and may not deprive defendants of the right to put on individualized evidence, to raise individualized defenses, and to receive a verdict on the individualized facts of each class member’s claims.”<sup>78</sup> A single-building/multiple-occupant mold exposure class action would inevitably violate these due process restrictions.

If a single occupant living in a defendant’s building were to sue for injuries allegedly caused by mold in her unit, she would have to prove by competent admissible evidence, among other things,

that: (i) there was mold growth in her unit, (ii) the mold growth was caused by the defendant's negligence, (iii) she was exposed to a certain type and amount of mold, (iv) she was biologically susceptible to that mold exposure, (v) she actually suffered from certain relevant symptoms, and (vi) her symptoms were caused by the mold in her unit rather than other environmental or health-related factors. She would also have to prove that she suffered damages, and in what amount.

For its part, the defendant would have a right to present all of the evidence available under the applicable law.<sup>79</sup> Specifically, it would have the right to present rebuttal evidence on each element of the plaintiff's case,<sup>80</sup> including evidence that: (a) the mold growth was caused or made worse by the plaintiff's behavior, (b) the specific type and/or quantity of mold in her unit has not been scientifically proven to cause the symptoms she alleges, (c) she is not biologically susceptible to the mold in her unit (*e.g.*, she is not allergic to mold or to that species of mold), (d) although there is mold in her unit, for various reasons she did not receive a significant dose (*e.g.*, she was rarely present in the unit), (e) any mold in her unit did not produce mycotoxins (if she is attempting to establish a toxic effect), (f) her symptoms were caused by another source (*e.g.*, exposure to another allergen or a pre-existing health condition), and/or (g) her symptoms were exacerbated by another environmental condition or her own behavior (*e.g.*, smoking). Furthermore, depending on the applicable law, the defendant would have a right to raise affirmative defenses such as the statute of limitations, contributory or comparative negligence, intervening and superseding cause, waiver, and assumption of risk.<sup>81</sup>

Even if this plaintiff's claims were subsumed in a class action, her right to collect damages for mold exposure could not be determined without individualized proof on these issues.<sup>82</sup> Indeed, if the claims of hundreds of occupants were lumped together in a single action, each plaintiff's burden to make these separate showings, the defendant's right to investigate and challenge each plaintiff's evidence and to offer individualized defenses, and the trial court's duty to make individualized case-by-case findings on these issues would be undiminished.<sup>83</sup> Thus, if a mold-exposure case is allowed to proceed as a class action, the trial will either be dominated by individual issues for each of hundreds (or thousands) of class members, or it will violate the due process clause by relieving plaintiffs of their obligation to prove each element of their case and depriving the defendant of its right to present evidence in its defense. Because neither alternative is permissible, such classes should not be certified.<sup>84</sup>

### **E. Other Toxic Exposure Cases**

Many of the arguments against certification of a single-building/multiple-occupant mold exposure class can be found, by analogy, in other purported toxic exposure class actions. Courts facing a wide array of such cases have almost uniformly denied certification, often citing predominance and superiority problems analogous to those described in Part C.<sup>85</sup> Those cases contain language that is potentially useful in a mold exposure case on at least the following issues:

- Causation of the contamination<sup>86</sup>
- Extent of the contamination<sup>87</sup>
- Varying types of contamination<sup>88</sup>
- Exposure to the contaminant<sup>89</sup>
- Susceptibility to the contaminant<sup>90</sup>
- Causation of the injury including non-specific nature of symptoms<sup>91</sup>
- Causation of specific symptoms<sup>92</sup>

- Alternative causation of the injury or exacerbation by other causes<sup>93</sup>
- Remediation of contamination<sup>94</sup>
- Affirmative defenses<sup>95</sup>
- General requirement for individualized proof<sup>96</sup>

This is not an exhaustive catalog of relevant cases, but only a resource for language that may be useful in the context of a mold exposure case. Before drawing analogies to these opinions in a mold exposure case, however, counsel must weigh the usefulness of such precedent against the risk of associating exposure to mold — a naturally occurring and often harmless organism — with exposure to significantly more menacing human-made substances (*e.g.*, PCBs or asbestos).

## **F. Conclusion**

Because the potential nationwide exposure for single-building/multiple-occupant mold class actions is so huge, it is critical that counsel for building owners and other defendants be prepared with well-reasoned and scientifically informed arguments at the class certification stage. Although there is superficial appeal to a single-building/multiple occupant class action, trial courts that are effectively educated about the highly individualized nature of mold related claims should realize that individual issues must predominate and that class treatment is not a superior method of adjudicating these lawsuits. In short, this is an issue on which consistently good lawyering at the class certification stage can avoid potentially huge exposures both for defendants in the suit at hand and for countless future defendants facing similar allegations.

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## **ENDNOTES**

1. See John Parker Sweeney & Sheri A. Mullikin, *The 'Mold Monster': Myth or Menace?*, 1 MEALEY'S LITIG. RPT.: MOLD, 1, 1 & nn.4-5 (2001) (citing 8 cases prior to 1996).
2. Some individuals have theorized that the construction of "airtight" buildings that began during the energy crisis of the 1970's resulted in higher levels of indoor mold. See, *e.g.*, *Mold: A Growing Problem: Hearing Before the Subcomms. on Oversight and Investigations and Housing and Community Opportunity, House Comm. on Financial Services*, 107th Cong. (July 18, 2002) (statement of Gerald M. Howard, EVP/CEO Nat'l Ass'n of Home Builders) ("Howard Statement"), available at 2002 WL 20319155. There is no documented proof for such an assertion, and indeed, this explanation does not account for the fact that mold litigation did not become significant until the late 1990's. See, *e.g.*, *id.*
3. See, *e.g.*, Dennis Hevesi, *The Turmoil over Mold in Buildings*, N.Y. Times, Mar. 23, 2003, § 11, at 1 ("about 10,000 mold-related lawsuits have been filed nationwide in the last three years").
4. *The Truth About Mold*, 28 Harv. Health Letter 3 (Harv. Med. Sch.), Jan. 2003, at 2; see also United States Centers for Disease Control and Prevention, *Update: Pulmonary Hemorrhage/Hemosiderosis Among Infants - Cleveland, Ohio, 1993-1996*, 49 Morbidity & Mortality Wkly. Rep. 9, 180 (2000) (discussing the retraction of the original report).
5. See, *e.g.*, 48 Hours: *This Mold House* (CBS television broadcast, Mar. 2, 2000); Michelle Conlin, *Is Your Office Killing You? Sick Buildings Are Seething with Molds, Monoxide — and Worse*, Business Week, June 5, 2000, at 114.
6. See, *e.g.*, Sweeney, *supra*, at 1-2.

7. *Id.* at 1.
8. See Dean Calbreath, *Increasingly Expensive Mold Infestation Claims Wreaking Havoc*, Copley News Service, Feb. 18, 2003.
9. *Id.*
10. *Id.*
11. See, e.g., Donna Kimura, *Owners hit with soaring insurance fees* (2002), at [http://www.housingfinance.com/aft/aft\\_articles/2002/02MarchAprilInsuranceFees/index.html](http://www.housingfinance.com/aft/aft_articles/2002/02MarchAprilInsuranceFees/index.html) (last visited June 9, 2003) (analogizing crisis in mold coverage to crisis in terrorism coverage); Lloyd's of London, *Increasing Difficulties in Breaking the Mould*, Lloyd's List International, April 10, 2003, at 6.
12. *Davis v. Henry Phipps Plaza South*, No. 116331/98 (N.Y. Sup. Ct. Aug. 8, 2001) (Order denying class certification at 11) (alteration and first two omissions in original) (internal quotation marks omitted).
13. *Id.* at 12 (internal quotation marks omitted).
14. See *Wheeler v. AvalonBay Communities*, No. B 153535, 2002 Cal. App. Unpub. LEXIS 10906 (Cal. App. Nov. 22, 2002).
15. *Id.* at \*3.
16. *Id.* at \*8-9 (internal quotation marks omitted). The appellate court also upheld the trial court's conclusion that "no amendment will cure the problems [with the complaint] and Plaintiffs can never realistically prove community of interest in law and facts concerning liability and causation, either as to the personal injury cause of actions or the remaining cause of actions [sic]." *Id.* at \*15 (internal quotation marks omitted).
17. See, e.g., Centers for Disease Control and Prevention, *Questions and Answers on Stachybotrys Chartarum and Other Molds ("CDC Q&A")*, Q.3, at <http://www.cdc.gov/nceh/airpollution/mold/stachy.htm> (last reviewed Nov. 30, 2002).
18. *Id.*
19. See, e.g., Stephen J. Henning & Daniel A. Berman, *Mold Contamination: Liability and Coverage Issues: Essential Information You Need to Know for Successfully Handling and Resolving Any Claim Involving Toxic Mold*, 8 Hastings W.-NW. J. Envtl. L. & Pol'y 73, 80 (2001).
20. See, e.g., Envtl. & Occupational Disease Epidemiology, N.Y. City Dept. of Health & Mental Hygiene, *Facts About Mold*, at <http://www.ci.nyc.ny.us/html/doh/html/epi/epimold.html> (Feb. 2001).
21. *State of the Science on Molds and Human Health: Hearing Before the Subcomms. on Oversight and Investigations and Housing and Community Opportunity, House Comm. on Financial Services*, 107th Cong. (July 18, 2002) (statement of Stephen C. Redd, M.D., Chief, Air Pollution and Respiratory Health Branch National Center for Environmental Health, Centers for Disease Control and Prevention) ("Redd Statement"), available at 2002 WL 1587891 (F.D.C.H.).
22. Robert K. Bush and John W. Yunginger, *Standardization of Fungal Allergens*, 5 Clin. Rev. Allergy 3 (1987).
23. CDC Q&A, *supra*, at Q.2.
24. Am. Coll. of Occupational & Envtl. Med., *Evidence Based Statements: Adverse Human Health Effects Associated with Molds in the Indoor Environment* 1 (Oct. 27, 2002) ("ACOEM"), available at <http://www.acoem.org/guidelines/pdf/Mold-10-27-02.pdf>.
25. *Id.* at 4.

26. *Id.*
27. *Id.*
28. *See, e.g., ACOEM, supra, at 1.*
29. *See, e.g., id. at 2.*
30. *See, e.g., id. at 3.*
31. *See, e.g., id. at 4.*
32. *See, e.g., Coreen A. Robbins et al., Health Effects of Mycotoxins in Indoor Air: A Critical Review, 15 Applied Occupational & Env'tl. Hygiene 773, 775 (2000).*
33. *See, e.g., ACOEM, supra, at 4-5; Robbins, supra, at 774; Redd Statement, supra* ("Linkages between indoor airborne exposures to molds and other health effects [beside infections and allergies] have not yet been scientifically substantiated"); Yehudith Assouline-Dayana et al., *Studies of Sick Building Syndrome. IV. Mycotoxicosis*, 39 J. of Asthma 191 (2002) ("there is no evidence in humans that mold exposure leads to nonmucosal pathology"); Texas Medical Association's Council on Scientific Affairs, *Black Mold and Human Illness*, CSA Report 1-I-02, at 4 (Sept. 2002) ("the proposition that molds in indoor environments may lead to adverse health effects through mechanisms other than infection and allergic/immunologic reactions is an untested impression"), available at [http://www.texmed.org/has/CSA Black Mold.doc](http://www.texmed.org/has/CSA%20Black%20Mold.doc); John Payne et al., *Latest Developments in Mold Exposure Litigation*, 17-Fall Nat. Resources & Env't. 132 (2002) (describing studies conducted by the CDC, California Department of Health Services, California Research Bureau, Washington State Department of Health, and two individual scientists finding no toxic effect).

A rare and significantly more serious condition, hypersensitivity pneumonitis, sometimes results from agricultural or industrial exposure to extremely high concentrations of mold and other biological particles in the air. *See ACOEM at 2.* Hypersensitivity pneumonitis is generally not a risk in residential or commercial settings. Furthermore, although plaintiffs in some cases have claimed to be suffering from a generalized mold hypersensitivity state, "[t]he existence of this disorder is not supported by reliable scientific data." *ACOEM, at 3.*

34. *See, e.g., Yehudith Assouline-Dayana, supra* (criticizing studies for various methodological errors and disanalogies to human pathophysiology); *Robbins, supra* (same); *Fischer, at 79-80* ("Reports on health effects due to ingestion of mycotoxins are of concern only in livestock breeding and food microbiology. \* \* \* The recent literature on health effects of mycotoxins in indoor air does not provide compelling evidence that [inhalation] exposure at levels expected in most mould-contaminated indoor environments is likely to result in measurable health effects.").
35. *Redd Statement, supra.*
36. *Id.; see also CDC Q&A, supra, at Q.15* ("Standards for judging what is an acceptable, tolerable, or normal quantity of mold have not been established.").
37. *See Liggett Group, Inc. v. Engle*, No. 3D00-3400, 2003 Fla. App. LEXIS 7500, at \*18, n.9, 16 (Fla. 3d DCA May 21, 2003) (decertifying class of smokers because, *inter alia*, "specific medical causation is inherently individualized" and "each class member had unique and different experiences that will require the litigation of substantially separate issues").
38. *See, e.g., CDC Q&A, supra, at Q.3.*
39. *See, e.g., Harv. Health Letter, supra, at 1.*
40. *See, e.g., id.*
41. *Robbins, supra, at 774.*

42. *Id.*; see also ACOEM, *supra*, at 4 (“The amount (if any) and type of mycotoxin produced is dependent on a complex and poorly understood interaction of factors that probably include nutrition, growth substrate, moisture, temperature, maturity of the fungal colony, and competition from other microorganisms.”).
43. D.M. Kuhn & M.A. Ghannoum, *Indoor Mold, Toxicogenic Fungi, and Stachybotrys Chartarum: Infectious Disease Perspective*, 16 *Clinical Microbiology Reviews* 144, 150 (2003).
44. See, e.g., Guido Fischer & Wolfgang Dott, *Relevance of Airborne Fungi and Their Secondary Metabolites for Environmental, Occupational and Indoor Hygiene*, 179 *Arch Microbiol* 75, 80 (2003).
45. *Id.* at 4 (emphasis added).
46. *Id.*; see also Harv. Health Letter, *supra*, at 2; EPA, Indoor Environments Division, *Mold Remediation in Schools and Commercial Buildings* (“EPA, Remediation”) (2001), at 42, available at <http://www.epa.gov/iaq/molds/graphics/moldremediation.pdf> (last updated June 25, 2001), Kuhn, *supra*, at 147.
47. See, e.g., California Department of Health Services, *Stachybotrys Chartarum: A Mold That May Be Found in Water-Damaged Homes* (“CDHS, Stachybotrys”), at <http://www.dhs.ca.gov/ehib/ehib2/topics/stachygp00.doc> (Nov. 2000).
48. See, e.g., Rafa\_ L. Górný, *Fungal Fragments as Indoor Air Biocontaminants*, 68 *Applied & Env’tl. Microbiology* 3522 (2002).
49. Kuhn, *supra*, at 146 (citations omitted).
50. See, e.g., Harriet M. Ammann, Washington State Department of Health, *Is Indoor Mold Contamination a Threat to Health?*, at <http://www.doh.wa.gov/ehp/oehas/mold.html> (last visited May 21, 2003); ACOEM, *supra*, at 6.
51. Redd Statement, *supra*; see also CDC Q&A, *supra*, at Q.10.
52. ACOEM, *supra*, at 2.
53. *Id.*
54. Minnesota Department of Health, *Indoor Mold: Hazard Identification and Control*, at <http://www.health.state.mn.us/divs/eh/indoorair/mold/hazardid.html> (last updated April 14, 2003); see also Redd Statement, *supra*.
55. CDC Q&A, *supra*, at Q.15; see also Redd Statement, *supra*.
56. ACOEM, *supra*, at 1.
57. See, e.g., *id.* at 2.
58. See, e.g., Bush, *supra*, at 5-6; Fischer, *supra*, at 77.
59. Ammann, *supra*; see also Bush, *supra*, at 14-15.
60. CDHS, *Stachybotrys*, *supra*.
61. ACOEM, *supra*, at 2.
62. Bush, *supra*, at 6.
63. See, e.g., Fischer, *supra*, at 77 (“professions in the fields of agriculture, forestry \* \* \* food production, \* \* \* [and] [w]aste collectors are at increased risk”).
64. See, e.g., Assouline-Dayana, *supra*, at 196.

65. EPA, *Remediation*, at 40.
66. See, e.g., Redd Statement, *supra*.
67. See, e.g., 1 American Academy of Allergy, Asthma, and Immunology, *The Allergy Report* ("AAAAI, Allergy") at 2 (2001), available at <http://www.theallergyreport.org/reportindex.html> (approximately 15 million Americans are asthmatic).
68. EPA, *Remediation, supra*, at 40.
69. See, e.g., 2 AAAAI, *Allergy, supra*, at 51-53.
70. See, e.g., *id.*
71. *Id.* at 53.
72. See, e.g., Fischer, *supra*, at 76; EPA, *Remediation, supra*, at 41.
73. See ACOEM, *supra*, at 3.
74. See, e.g., authorities cited at note 33, *supra*.
75. Although this article focuses on personal injury claims, many of the arguments suggested above are applicable to other mold-related claims. For example, property damage claims require individualized proof of the amount and causes of mold in a building unit (Part C.1) in addition to individualized proof that articular instances of property damage are actually the result of mold (or that the mold developed while the property was located in the unit). Similarly, warranty of habitability claims or nuisance claims require individualized proof of the amount, type and causes of mold (Part C.1), and the individual's sensitivity to that mold (Part C.4) because the mold in a particular unit may be hazardous to one person and innocuous to another. This article also does not discuss class actions seeking injunctive relief under F.R.C.P. 23(b)(2).
76. See generally, Evan M. Tager, *The Constitutional Limitations on Class Actions*, MEALEY'S LITIG. RPT.: CLASS ACTIONS, January 2001, at 34.
77. *Amchem Prods., Inc. v. Windsor*, 521 U.S. 591, 613, 615 (1997) (quoting the Rules Enabling Act, 28 U.S.C. § 2072(b)).
78. Tager, *supra*, at 35.
79. See, e.g., *Lindsey v. Normet*, 405 U.S. 56, 66 (1972) ("[d]ue process requires that there be an opportunity to present every available defense") (internal quotation marks and citation omitted).
80. See, e.g., *W. Elec. Co. v. Stern*, 544 F.2d 1196, 1199 (3d Cir. 1976) ("defendants must be allowed to present any relevant rebuttal evidence they choose, including evidence that there was no [violation of the law with respect to] one or more members of the class").
81. See, e.g., *Engle*, 2003 Fla. App. LEXIS, at 21 ("affirmative defenses and damages must be litigated individually").
82. See, e.g., *id.* at 31 ("Specific medical causation and legal causation, along with other elements of liability, must be established on an individualized basis.").
83. See, e.g., *Broussard v. Meineke Discount Muffler Shops, Inc.*, 155 F.3d 331, 345 (4th Cir. 1998) ("[A] class action is an exception to the usual rule that litigation is conducted by and on behalf of the individual named parties only. It is axiomatic that the procedural device of [a class action] cannot be allowed to expand the substance of the claims of class members. Thus courts [must] avoid the real risk \*\*\* of a composite case being much stronger than any plaintiff's individual action would be.") (internal quotation marks and citations omitted); see generally, Tager, *supra*, at 36 n.17 (citing cases making similar points).

84. In jurisdictions imposing such a requirement, it is also important to remind the court that it must formulate a manageable trial plan for the class in its certification order. *See, e.g., Castano v. American Tobacco Co.*, 84 F.3d 734, 740 (5th Cir. 1996) (trial court's failure to explain how it intends to manage a class action is reversible error).
85. *See, e.g., Amchem Prods., Inc. v. Windsor*, 521 U.S. 591 (1997) (upholding decertification of class of individuals exposed to asbestos in defendants' products); *Boughton v. Cotter Corp.*, 65 F.3d 823 (10th Cir. 1995) (upholding denial of certification of class of individuals exposed to emissions from uranium mill); *In re Fibreboard Corp.*, 893 F.2d 706 (5th Cir. 1990) (vacating order for class action trial of individuals exposed to asbestos in defendants' products); *Jacobs v. Osmose*, 213 F.R.D. 607 (S.D. Fla. Feb. 25, 2003) (denying certification of putative class of individuals whose property was contaminated by CCA, a wood treatment, that leached from treated wood); *Neehan v. Carnival Corp.*, 199 F.R.D. 372 (S.D. Fla. 2001) (denying certification of putative class of cruise ship passengers exposed to waste fumes); *Church v. General Electric Co.*, 138 F. Supp. 2d 169 (D. Mass. 2001) (denying certification of putative class of individuals whose land was contaminated with PCBs that leaked from defendant's plant); *Jones v. Allercare, Inc.*, 203 F.R.D. 290 (N.D. Ohio 2001) (denying certification of putative class of individuals who suffered allergic reaction to dust mite powder and spray); *Reilly v. Gould, Inc.*, 965 F. Supp. 588 (M.D. Pa. 1997) (dismissing class action allegations of individuals whose homes were contaminated by lead from a battery plant); *Hurd v. Monsanto Co.*, 164 F.R.D. 234 (S.D. Ind. 1995) (denying certification of putative class of workers exposed to PCBs during work at electrical plant); *Thomas v. Fag Bearings Corp.*, 846 F. Supp. 1400 (W.D. Mo. 1994) (denying certification of putative class of individuals whose groundwater was contaminated by TCE released from bearings plant); *Puerto Rico v. M/V Emily S*, 158 F.R.D. 9 (D.P.R. 1994) (denying certification of putative class of individuals exposed to fumes from fuel oil spill); *Jackson v. The Glidden Co.*, 2001 Ohio Misc. LEXIS 2 (Ohio Ct. Com. Pl. March 30, 2001) (denying certification of putative class of individuals exposed to lead-based paint in their residences); *Millett v. Atlantic Richfield Co.*, 2000 Me. Super. LEXIS 39 (Me. Super. Ct. March 2, 2000) (denying certification of putative class of individuals whose well water was polluted by spillage of MTBE, a gasoline additive); *Muttart v. American Mortgage & Guaranty Co.*, 1998 Del. Super. LEXIS 30 (Del. Super. Ct. Feb. 9, 1998) (striking class action allegations of individuals exposed to contaminated air in a building).
86. *Jacobs*, 213 F.R.D. at 614 ("a great number of factors can contribute to whether (and to what extent) the CCA in treated wood can escape[;] \*\*\* as more frequently touched structures age, the risk of dislodge increases as exposed surfaces are worn \*\*\* the climate can also have a substantial impact on dislodging or leaching[;] \*\*\* identifying the source and origins of contaminants in soil will mandate a separate inquiry on each parcel of land at issue"); *Reilly*, 965 F. Supp. at 603 ("[a]lthough it is well established that the [battery plant] may have caused the release of lead into the environment, it has also been pointed out by defendant's counsel that there are other prevalent sources of lead emission"); *Thomas*, 846 F. Supp. at 1404 ("Because the results [of sample testing] vary markedly from well-to-well, expert testimony on the actual source of contamination for each well may be required."); *Millett*, 2000 Me. Super. LEXIS at \*49 ("The actual cause of the MTBE contamination of each plaintiff's well will have to be addressed on an individual basis. While there may be class members whose well contamination was specifically caused by the defendants' alleged failure to provide adequate warnings, there are likely to be many other class members whose wells were contaminated by other causes. For example, \*\*\* by the actions of their neighbors or some other third party.") (footnote omitted); *id.* at \*52-53 ("[D]efendants' affirmative defense of comparative negligence \*\*\* raises issues which must be determined on an individual basis. \*\*\* [I]n order to pursue this defense, defendants will need to ask each class member whether they self-contaminated their property.").
87. *Church*, 138 F. Supp. 2d at 182 ("[t]o judge whether there has been a harmful enough invasion by PCBs for liability to attach \*\*\* an expert must necessarily measure the extent of the contamination of the individual properties"); *Reilly*, 965 F. Supp. at 598-99 ("[a] random sampling \*\*\* reveals that other members \*\*\* have varying levels of lead in their soil").
88. *M/V Emily S*, 158 F.R.D. at 13 ("Each [vapor emitted by the fuel oil] has different characteristics, \*\*\* produces different effects on human health \*\*\* changed differently as they reacted to environmental factors, and thus one cannot speak of the vapor itself as of uniform character.").
89. *Amchem*, 521 U.S. at 609 ("class members in this case were exposed to different asbestos-containing products, in different ways, over different periods, and for different amounts of time"); *Boughton*,

- 65 F.3d at 828 (“individual measurements can be quite relevant evidence in determining the extent of an individual’s level of exposure and \*\*\* such measurement may vary from individual to individual”); *In re Fibreboard Corp.*, 893 F.2d at 710 (“[t]he dates of exposure [and] [t]he types of products to which class members were exposed varies among class members”); *Reilly*, 965 F. Supp. at 603-04 (“exposure varies from person to person \*\*\* [and] the effect that lead has on a child \*\*\* is dependent on the amount of lead ingested and the duration of ingestion”); *Hurd*, 164 F.R.D. at 239-40 (“each plaintiff was exposed to different levels of PCBs for different amounts of time in different areas of the plant \*\*\* some class members have been exposed to PCBs only for a few months and at low levels, while others, for decades and at high levels”); *M/V Emily S*, 158 F.R.D. at 14 (“[there are] variances in exposure leading to the need of assessing the individualized circumstances of each personal injury claimed”); *id.* (“environmental factors \*\*\* varied significantly over time, which lead to a variance in duration, intensity, and implications of exposure”); *Jackson*, 2001 Ohio Misc. LEXIS at \*18 (“The plaintiffs were all exposed to lead-based paint for different amounts of time, and to paint that was manufactured and applied over different periods.”); *Muttart*, 1998 Del. Super. LEXIS at \*13-14 (“Class members allegedly shared the common experience of exposure to contaminants in the \*\*\* building. The extent of their commonality ceases to exist at that point, however. The nature of the allegations by the Plaintiffs inevitably will contain individual issues of causation, as they relate to varying exposure to the building contaminants, and issues of pre-existing medical conditions.”).
90. *Jones*, 203 F.R.D. at 301 (“plaintiffs do not allege that the Powder and Spray automatically cause adverse reactions in all those exposed to the products \*\*\* the relevant question in this case will not be whether the products have the capacity to cause harm, but whether the products caused harm and to whom”); *Reilly*, 965 F. Supp. at 604 (“children have a higher lead absorption rate than adults”); *Hurd*, 164 F.R.D. at 239 (“Each putative class member’s susceptibility to injury from PCBs will vary.”); *M/V Emily S*, 158 F.R.D. at 14 (“each person’s susceptibility to injury from the vapors would vary with such factors as age, weight, sex, and preexisting health conditions”).
91. *Amchem*, 521 U.S. at 609 (“some [class members] suffered no physical injury, others suffered disabling or deadly diseases”); *In re Fibreboard Corp.*, 893 F.2d at 710 (“[t]he severity and type of physical or mental injuries varies among class members [and] [n]ot all of the Plaintiffs have been injured by the acts, omissions, conduct or fault of all of the Defendants”); *Jones*, 203 F.R.D. at 301 (“proof of causation will depend on individual factors such as the nature of each plaintiff’s exposure and personal susceptibility”); *Reilly*, 965 F. Supp. at 597 (“True, all plaintiffs are said to have been exposed to lead emission from the site, but whether and to what extent the emissions are said to have affected each class member is not common to all involved.”); *Hurd*, 164 F.R.D. at 239-40 (“[N]o single proximate cause inquiry applies equally to each putative class member; no one set of operative facts establishes liability. \*\*\* Some class members may never experience injury symptoms or will experience long latency periods because of the specific circumstances surrounding their exposure and medical histories, while others may experience almost immediate and perceptible harm. \*\*\* [C]ausation \*\*\* will necessarily be different for every person in the proposed class, based on each person’s length of exposure to [PCBs], notice, pre-existing medical conditions and other factors”) (alteration in original) (quoting *M/V Emily S*, 158 F.R.D. at 15); *M/V Emily S*, 158 F.R.D. at 13 (“the factors that would go into establishing the fact of injury and the causal link between that injury and the spill would be numerous, would change upon complex interactions among variables, and would vary greatly over time \*\*\* and from person to person”); *id.* at 14 (“many of the ailments the plaintiffs wish to attribute to the spill, such as dizziness and headaches, are common and may be caused by factors other than exposure to oil vapors”); *Jackson*, 2001 Ohio Misc. LEXIS at \*18 (“There is no common injury typical of lead exposure, nor are any of the alleged injuries caused by lead exposure exclusive to such exposure.”) *Muttart*, 1998 Del. Super. LEXIS \*13-14 (“Class members allegedly shared the common experience of exposure to contaminants in the \*\*\* building. The extent of their commonality ceases to exist at that point, however. The nature of the allegations by the Plaintiffs inevitably will contain individual issues of causation, as they relate to varying exposure to the building contaminants, and issues of pre-existing medical conditions.”).
92. *In re Fibreboard Corp.*, 893 F.2d at 712 (“The plaintiffs’ lifestyles differed in material respects. To create the requisite commonality for trial, the discrete components of the class members’ claims and the asbestos manufacturers’ defenses must be submerged.”); *Jones*, 203 F.R.D. at 302 (“[b]y proving that the Powder and Spray could cause [the symptoms suffered by the named plaintiffs] plaintiffs have done nothing to further the claims of the proposed class members who experienced [other symptoms]”).

93. *Jones*, 203 F.R.D. at 307 (“even if plaintiffs were able to prove that the Powder and Spray were defective \*\*\* each individual plaintiff would have to prove he or she had an adverse reaction which was caused by the products. Defense experts or fact witnesses could be expected to testify that the adverse reaction was caused by something other than defendants’ products.”).
94. *Reilly*, 965 F. Supp. at 604 (“since the presence of lead in some residences has been reduced due to the remediation process, this fact will play a role in determining the amount and length of exposure”); *Millett*, 2000 Me. Super. LEXIS at \*56 (“determining which remediation method or methods are appropriate for a particular site will depend on a multitude of factors specific to each site”).
95. *Hurd*, 164 F.R.D. at 240 (“the claims of some putative class members may be barred by the statute of limitations. \*\*\* Individual hearings would thus be necessary to discover when each class member first learned that she had been injured by exposure to PCBs.”); *Jackson*, 2001 Ohio Misc. LEXIS at \*21 (“there are defenses to liability which would require evidence specific to each class member \*\*\* [including] intervening/superseding actions of landlords in not abating the lead paint problems”); *Millett*, 2000 Me. Super. LEXIS at \*52 (“[D]efendants’ affirmative defense of comparative negligence \*\*\* raises issues which must be determined on an individual basis. \*\*\* [I]n order to pursue this defense, defendants will need to ask each class member whether they self-contaminated their property.”); *Muttart*, 1998 Del. Super. LEXIS at \*14 (“individual Plaintiffs may be subject to statutory defenses such as statute of limitations or assumption of the risk”).
96. *Boughton*, 65 F.3d at 826 (“individual issues includ[e] whether purchasers were aware contamination existed, the extent and nature of injuries, the degree and length of exposure, the prevalence of contamination[;] \*\*\* there was not a single course of conduct alleged to have caused the injuries, identical with respect to each plaintiff; \*\*\* their injuries derived from more than one source”); *In re Fibreboard Corp.*, 893 F.2d at 712 (“The plaintiffs suffer from different diseases, some of which are more likely to have been caused by asbestos than others. The plaintiffs were exposed to asbestos in various manners and to varying degrees. The plaintiffs’ lifestyles differed in material respects. To create the requisite commonality for trial, the discrete components of the class members’ claims and the asbestos manufacturers’ defenses must be submerged.”); *Jacobs*, 213 F.R.D. at 614 (“unlike single-incident tragedies, more nebulous classes based on injuries that are spread out over a long period of time do not lend themselves well to class certification, in that no single set of operative facts establishes liability and no single proximate cause applies to each potential class member’s injuries”) (citation omitted); *Neenan*, 199 F.R.D. at 376 (“the individual issues of causation and injury in fact will consume the majority of the Court’s time in this matter \*\*\* whether the passenger returned to a cabin with fully operational toilets and water faucets or to a cabin with faulty toilets and water faucets; whether toilets were reinstated in the passenger’s cabin; whether the water in the passenger’s cabin was brown, cloudy, or clear; whether the passenger was compelled to drink bottled water”); *Hurd*, 164 F.R.D. at 240 (“even if a jury could make a class-wide finding of fact regarding the general health risks posed by PCBs, resolution of liability issues would still require an individual inquiry into the circumstances involving each class member’s exposure and susceptibility”); *Thomas*, 846 F. Supp. at 1404 (“[there would be] hundreds or thousands of individual mini-trials on complex causation and damages issues, while the only benefit of a class would be that the ruling of several common, but not particularly daunting issues, would be made applicable to the entire class”); *M/V Emily S.*, 158 F.R.D. at 15 (“the issues that would take the most judicial time to resolve, and which are central to the plaintiffs’ ability to recover, will be individual issues of injury in fact and causation \*\*\* [e]ven if the plaintiffs succeeded in establishing the fault or negligence of one or more defendants, the personal injury claimants would still have the bulk of their cases to prove”). ■





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