

Evaporative coolers are not associated with dust mite or mold sensitization in a large pediatric cohort



The primary species of house dust mites (HDMs), *Dermatophagoides farinae* and *Dermatophagoides pteronyssinus*, are associated with asthma, atopic dermatitis, and allergic rhinitis.¹ Mold exposure also is implicated in allergic disease, most prominently with asthma.² HDM survival is dependent on a relative humidity greater than 55% to 75%,³ and most species of fungi grow at a relative humidity greater than 60%.⁴ Evaporative (or swamp) coolers (ECs) are used as a low-cost air-conditioning alternative in arid and semiarid climates. ECs use a pump to circulate water from a reservoir onto a cooling pad. A fan draws air from outside through the wet pad, cooling and moistening the indoor air. ECs have been shown to raise the relative humidity of the home by approximately 10%.¹ This increase becomes most important in arid or semiarid climates, such as Colorado, Wyoming, Nevada, Arizona, and Utah, where the humidity does not typically support HDM survival or mold growth. Small studies conducted in Colorado, Utah, and Australia have suggested that EC use increases household HDM allergen content,^{5–7} and 1 small cohort in Nevada found an increase in HDM and mold sensitization in 190 mostly pediatric patients.⁸ In this study we evaluated whether EC use in Colorado leads to HDM and/or mold sensitization in a very large, atopic, pediatric cohort.

We performed a retrospective review of all Colorado residents no older than 21 years seen at National Jewish Health from 2008 to 2017 who had atopy (≥ 1 positive environmental skin prick test result) and had HDM and/or mold skin prick testing performed. These data were downloaded from the National Jewish Health Research Database. Patients were analyzed for whether they had an EC, HDM sensitization, mold sensitization, and atopic comorbidities. Common molds and those endemic to the area were tested, including *Aspergillus* species (spp), *Penicillium* spp, *Chaetomium globosum*, *Curvularia spicifera*, *Fusarium solani*, *Alternaria tenuis*, *Cladosporium herbarum*, *Helminthosporium sativum*, and *Ustilago* spp (corn and wheat smut). Sensitization was based on a positive skin prick test reaction, defined as a mean wheal size at least 3 mm larger than the saline control. HDM and mold cohorts were very similar, with a mean age of approximately 9 years, a slight male predominance, and a significant number affected by atopic comorbidities (Table 1). The rate of allergic rhinitis was slightly lower among patients with an EC in the 2 cohorts, and this difference was statistically significant (Table 1). Of the 8,503 patients with HDM testing results, 992 had an EC, with 284 (28.6%) showing HDM sensitization, and

7,511 did not have an EC, with 2,128 (28.3%) showing HDM sensitization (relative risk 1.01, 95% confidence interval 0.91–1.12, $P = .8452$). Of the 9,286 patients with mold testing results, 1,092 had an EC, with 487 (44.6%) showing any mold sensitization, and 8,194 did not have an EC, with 3,550 (43.3%) showing any mold sensitization (relative risk 1.03, 95% confidence interval 0.96–1.10, $P = .4255$, respectively). These results held true when stratifying for sex, age, and atopic comorbidity. In addition, mold sensitization was not associated with EC use when stratifying for individual mold species or mold characteristics (indoor, temperate, and associated with water damage).

We show that although the use of ECs has been shown to raise the relative humidity in arid environments, they did not increase HDM or mold sensitization in a large cohort of children with atopy in Colorado. The major strength of our study is the large pediatric cohort, which is several-fold larger than in previous studies. In addition, we examined relevant atopic comorbidities and found no association with HDM and mold sensitization. Highly susceptible individuals with atopic dermatitis who have a compromised skin barrier and a propensity toward cutaneous sensitization⁹ also did not show an increase in sensitization. No association was found when evaluating patients according to various age groups, including infants, school-age children, adolescents, and children younger or older than 6 years.

We also present the first detailed assessment of EC use as it pertains to mold sensitization and found no association with ECs for any mold species. Furthermore, we found no association when molds were differentiated based on environmental association, including indoor molds (*Aspergillus* spp, *Penicillium* spp, *A tenuis*), temperate molds (*C herbarum*, *H sativum*), water damage-associated molds (*C globosum*, *C spicifera*), and outdoor molds (*Ustilago* spp). A recent article by Lemons et al¹⁰ found that homes with ECs had different fungal species compared with homes with air conditioners in Nevada. Although we did not evaluate all mold species from this study, the most representative molds that were found to be different, *Alternaria* spp in air-conditioned homes and *Aspergillus* spp in EC homes, were analyzed in our study, and no significant difference was noted. In addition, despite the difference in fungal species, Lemons et al¹⁰ did not find EC homes to have increased indoor humidity in their small sample of 19 homes. This is commonly cited as the reason why ECs increase mold exposure.^{2,4,10,11}

Table 1
Demographics

	Dust mite cohort				Mold cohort			
	Swamp cooler	No swamp cooler	P value	Total	Swamp cooler	No swamp cooler	P value	Total
Age (y), mean $\pm$ SD	8.9 $\pm$ 4.7	8.8 $\pm$ 4.6	.9794	8.8 $\pm$ 4.6	8.9 $\pm$ 4.6	8.9 $\pm$ 4.6	.6484	8.9 $\pm$ 4.6
Male sex, n (%)	576 (58)	4355 (58)	.9604	4,931 (58)	633 (58)	4,765 (58)	.9072	5,398 (58)
Atopic comorbidities, n (%)								
Allergic rhinitis	560 (56)	4,526 (60)	.0215	5,086 (60)	628 (58)	5,000 (61)	.0257	5,628 (61)
Asthma	632 (63)	4,543 (60)	.0505	5,175 (61)	707 (65)	5,060 (62)	.0556	5,767 (62)
Atopic dermatitis	135 (14)	1,030 (14)	.9284	1,165 (14)	149 (14)	1,225 (15)	.2538	1,374 (15)
Total, n	992	7,511		8,503	1,092	8,194		9,286

Our results are in agreement with a more recent study by Johnston et al¹ in Utah that found that although relative humidity was increased in homes with ECs, levels were still below the critical equilibrium humidity required for HDM survival. They argued that continuous humidity monitoring in their study was more accurate than single measurements and likely accounted for their opposing findings compared with previous studies. Limitations of our study include its retrospective nature. Also, other important factors that can affect HDM and mold sensitization, including water damage and housing characteristics, such as age of home and mattress, amount and type of furniture, type of flooring, and occupant density, were not evaluated.^{1,7,11} In conclusion, ECs increase the relative humidity in the home, but this does not increase HDM or mold sensitization in pediatric patients with atopy residing in Colorado. These results indicate that providers and patients in arid or semiarid climates who choose to use an EC do not need to be concerned about developing HDM or mold sensitization or this contributing to their atopic disease.

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A case of neuropathic pain in monoclonal mast cell activation syndrome



Neuropathic pain is an uncommon symptom associated with mast cell disorders but can significantly impact quality of life. Mast cells are known to be gatekeepers of nociception and play an important role in the development of neuropathic pain in the central and peripheral nervous system.¹ Although musculoskeletal pain has been associated with mast cell disorders,² neuropathic pain associated with mast cell disorders and its management has not been well described in the literature. We present a patient with neuropathic pain associated with monoclonal mast cell activation syndrome (MMAS) and review the agents used in his treatment.

The patient was a 49-year-old male with a past medical history significant for allergic rhinitis on allergen immunotherapy, who presented with a history of spontaneous flushing episodes for 4 years, 2 associated with loss of consciousness. He underwent extensive evaluations ruling out cardiac, neurologic, or endocrine etiologies of these episodes. After an episode of anaphylaxis after an allergen immunotherapy injection (serum tryptase 28.6 ng/mL at 1 hour after symptom onset), he had persistently elevated baseline serum tryptase (17.6 ng/mL) 1 week later. Repeat serum tryptase 1 month later remained elevated at 14.4 ng/mL, which prompted further evaluation for an underlying mast cell disorder.² Physical exam was without evidence of concerning skin lesions such as urticaria pigmentosa. Bone marrow biopsy revealed no mast cell aggregates or abnormal morphology but was positive for the KIT D816V mutation, leading to the diagnosis of MMAS, as no other criteria for systemic mastocytosis were met.³ No other studies evaluating mast cell mediators in the blood, urine, or cerebrospinal fluid were performed.

The patient's flushing episodes worsened, increasing in frequency to 10 times daily. Concurrent with the increased frequency of flushing, he began complaining of pain in his wrists and forearms, progressing to his lower extremities over 1 year. Pain was described as severe burning, tingling, and numbness, becoming so debilitating he became wheelchair-bound. Prior to the onset of pain, he did not receive any vaccines nor were there any changes to his medications or daily activities. Treatment regimens for flushing included H₁ and H₂ antihistamines (fexofenadine 180 mg twice daily, ranitidine 150 mg twice daily), a leukotriene antagonist (montelukast 10 mg daily), cromolyn sodium 200 mg orally 4 times daily, and aspirin 81 mg daily for refractory flushing. Flushing improved on this regimen; a short trial of omalizumab had no effect.

Despite control of the flushing, his pain worsened and became increasingly difficult to treat, prompting a neurology referral. On evaluation he showed no evidence of monoclonal gammopathy, chronic infections, autoimmune disease, metabolic, toxic, or genetic etiologies of pain. Skin biopsy revealed normal numbers of epidermal nerve fibers; however, autonomic function testing revealed abnormal heart rate response to deep breathing, abnormal blood pressure response to Valsalva maneuver, and abnormal quantitative sudomotor axon reflex testing in the proximal and distal leg. Based on his clinical presentation and test results, the patient was diagnosed with painful small-fiber neuropathy and pandysautonomia.

Several medication trials for the treatment of his pain over the course of 6 months (Table 1), including nonsteroidal anti-inflammatory drugs, tricyclic antidepressants, a selective norepinephrine reuptake inhibitor, gamma-aminobutyric acid analogs, opioids, and topical pain medications, were ineffective in improving his pain. He further underwent a trial of high-dose intravenous gammaglobulin⁴ for 3 months without improvement in pain, and repeat autonomic testing showed worsening pandysautonomia.

Disclosures: Authors have nothing to disclose.

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