

An incidence survey of fibromyalgia in mold exposed patients

Jessica A Zagory, Michael R Gray, MD MPH
Progressive Healthcare Group, Benson, AZ
jzagory1@swarthmore.edu

Abstract

An incidence survey of fibromyalgia (FM) was done on mold exposed patients at Progressive Healthcare Group, Benson, AZ. Through a medical records audit, percentages of FM diagnosis were calculated for patients who had been exposed to toxigenic structural molds, those who had been exposed to chemicals, and those in the regular internal medicine practice population. Of those with mold exposure, 21% were diagnosed with FM in comparison with 10.6% in the chemical exposure group and 3.8% in the regular practice. The finding that FM cases are almost twice as common in the mold as in the chemical patients suggests that factors other than simply small airways blockage, as seen in the clinical pulmonary function test results, contribute to muscle hypoxia and FM. Future investigations might include collection of environmental hygiene reports from every patient as well as investigation into the incidence of chronic fatigue syndrome and its relationship to FM.

Summary

Common symptom complaints reported in a pre-appointment questionnaire suggest that those exposed to toxigenic structural molds often experience fatigue and pain. Although FM etiology and pathology still remain unclear, according to the American College of Rheumatology it is defined by 1) history of widespread pain, steady or intermittent for at least three months and present on both the right and left sides of the body, above and below the waist, and midbody, i.e. neck, midchest, or midback, and 2) have pain on pressing at least 11 of the 18 trigger points and none in the four distracter points tested [1]. These patients report lower threshold of pain, found more prominently in sites with underlying nerves rather than bony and pure muscle areas [2]. Henriksson proposed that the most likely cause of pain is the combination of muscle tension and muscle hypoxia, which may lead to a state of energy deficiency in the resting painful muscle, and within the painful muscle he found ragged red fiber, a symptom also seen in mitochondrial disorders [3, 4].

Initial impressions suggested high FM incidence in patients exposed to mold. Some of these molds, such as *Stachybotrys chartarum*, thrive in buildings because they consume cellulose [5]. Ranging in size from 7 to 0.3-0.03 micron, the airborne spores are small enough that they are inhaled and are capable of penetrating the alveoli, stimulating an immune response as well as releasing mycotoxins that are absorbed into the bloodstream and circulate throughout the body [6].

Many of the mycotoxicosis patients had inflammation and elevated levels of immune activity. When inflamed, the body produces fibrin, but in the absence of a rupture to repair, fibrin is laid down in capillaries, where it thickens the walls and impairs oxygen transfusion. Without sufficient oxygen, the tissues metabolize anaerobically, leading to tissue fatigue. Moreover, some metabolites, notably aflatoxin B₁ (AFB₁) produced by the *Aspergillus* mold family, are DNA adductors [explain what a DNA adductor is/does] that target mitochondrial DNA. Insufficient production of mitochondrial proteins such as ATP synthase and the lack of ATP in the central nervous system can lead to functional impairments. By both methods, mycotoxicosis impairs mitochondrial function and can cause destruction of the mitochondria.

A records audit of patient progress notes found that 21% of mold patients, 11% of chemical patients, and 4% of regular patients had been diagnosed with FM, showing a significant association between FM and mold exposure (χ^2 , $P << 0.001$). The large difference between the chemical exposure and mold groups is also of interest since it suggests causes of FM beyond tissue hypoxia rooted in the lungs; although many of the patients in both groups see as much as a 50% reduction in small airways, there must be another factor from the mold that contributes to doubling the cases of FM.

References

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