Neural autoantibodies and neurophysiologic abnormalities in patients exposed to molds in water-damaged buildings.

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Adverse health effects of fungal bioaerosols on occupants of water-damaged homes and other buildings have been reported. Recently, it has been suggested that mold exposure causes neurological injury. The authors investigated neurological antibodies and neurophysiological abnormalities in patients exposed to molds at home who developed symptoms of peripheral neuropathy (i.e., numbness, tingling, tremors, and muscle weakness in the extremities). Serum samples were collected and analyzed with the enzyme-linked immunosorbent assay (ELISA) technique for antibodies to myelin basic protein, myelin-associated glycoprotein, ganglioside GM1, sulfatide, myelin oligodendrocyte glycoprotein, alpha-B-crystallin, chondroitin sulfate, tubulin, and neurofilament. Antibodies to molds and mycotoxins were also determined with ELISA, as reported previously. Neurophysiologic evaluations for latency, amplitude, and velocity were performed on 4 motor nerves (median, ulnar, peroneal, and tibial), and for latency and amplitude on 3 sensory nerves (median, ulnar, and sural). Patients with documented, measured exposure to molds had elevated titers of antibodies (immunoglobulin [Ig]A, IgM, and IgG) to neural-specific antigens. Nerve conduction studies revealed 4 patient groupings: (1) mixed sensory-motor polyneuropathy (n = 55, abnormal), (2) motor neuropathy (n = 17, abnormal), (3) sensory neuropathy (n = 27, abnormal), and (4) those with symptoms but no neurophysiological abnormalities (n = 20, normal controls). All groups showed significantly increased autoantibody titers for all isotypes (IgA, IgM, and IgG) of antibodies to neural antigens when compared with 500 healthy controls. Groups 1 through 3 also exhibited abnormal neurophysiologic findings. The authors concluded that exposure to molds in water-damaged buildings increased the risk for development of neural autoantibodies, peripheral neuropathy, and neurophysiologic abnormalities in exposed individuals.

In summary, 119 individuals exposed to mold colonies in water-damaged buildings were found to have autoantibodies against 9 different neural antigens. Neurophysiological recordings of latencies, amplitudes and velocities on 4 motor nerves and 3 sensory nerves revealed peripheral neuropathies in 99 (83%). Three abnormal neuropathies were found: mixed sensory-motor polyneuropathy, motor neuropathy, and sensory neuropathy. We recommend that mold exposed individuals with symptoms of neuropathy be evaluated for antibodies against neural antigens and for neurophysiological abnormalities. Additional work is needed to correlate and clarify the extent of the peripheral nerve pathology and demyelination, as well as the role of neural autoantibodies in this process.

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