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**Title:** Effect of source of inhalation antigen on manifestation and prognosis of EAA patients

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**Body:** Exposure to moulds in domestic environment leading to EAA is often reported. Inhalation exposure to moulds may lead to development of profibrotic Th2 cells. Aim of the study: To examine influence of inhalation antigen on EAA manifestation and prognosis. Fiftyfour patients of the mean age  $55,9 \pm 17,5$  were included to the retrospective study. They underwent complex diagnostic program including detailed history assesment, physical examination, serum specific IgG tests, lung functions tests (spirometry, diffusing capacity of the lung for CO), HRCT of the chest, bronchoscopy with BAL and TBB. Patients were divided into five groups according to their antigen exposure history – unknown source of exposure, professional exposure in chemical industry workers, exposure to moulds, exposure to bird antigens and exposure to mammal's fur and epithelium. Patients with mould exposure history had significantly higher FVC ( $p < 0,05$ ), FEV1 ( $p < 0,01$ ) and Dlco ( $p < 0,01$ ) at the time of diagnosis than other groups. Significant improvement of FVC and Dlco was detected in the patients with history of exposure to bird antigens. We found no difference in BALF differential cell counts among patient groups. BALF PMN cell count at the time of diagnosis negatively correlated with FVC and FEV1 a year after diagnosis in the whole group ( $p < 0,05$ ). EAA caused by moulds does not have a worse manifestation and prognosis that EAA caused by other inhalation antigens. Better outcome of patients exposed to bird antigens is probably influenced by easy removal of the antigen's source. The type of inhalation antigen very likely does not influence the manifestation of EAA.