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Dampness and moulds in workplace buildings: Associations with incidence and remission of sick building syndrome (SBS) and biomarkers of inflammation in a 10 year follow-up study

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ABSTRACT

There are few longitudinal studies on health effects of dampness and moulds in workplace buildings. We studied associations between dampness and indoor moulds in workplace buildings and selected biomarkers as well as incidence and remission of sick building syndrome (SBS). The study was based on a ten-year prospective study (1992–2002) in a random sample of adults (N = 429) from the Uppsala part of the European Community Respiratory Health Survey (ECRHS). The 10-year incidence (onset) of general, mucosal, dermal symptoms and any symptom improved when away from the workplace (work-related symptoms) was 7.2%, 11.6%, 6.4% and 9.4% respectively. The 10-year remission of general, mucosal, dermal symptoms and work-related symptoms was 71.4%, 57.1%, 70.4% and 72.2% respectively. Signs of dampness in the floor construction in any workplace building during follow up (cumulative exposure) was associated with incidence of mucosal symptoms (OR = 2.43). Cumulative exposure to moldy odor was associated with incidence of work-related symptoms (OR = 2.69). Cumulative exposure to dampness or moulds was associated with decreased remission of work-related symptoms (OR = 0.20 for water leakage, OR = 0.17 for floor dampness, and OR = 0.17 for visible indoor mould growth). Working in a building repaired because of dampness (repaired building) or mould was associated with decreased remission of work-related symptoms (OR = 0.32). Any dampness or moulds at baseline in the workplace building was associated with increased bronchial responsiveness (BR) and higher levels of Eosinphilic Cationic Protein (ECP) in serum and Eosinophilic counts in blood at baseline. Cumulative exposure to dampness and moulds, and work in a repaired building, was associated with increased BR at follow-up. In general, dampness and moulds in the workplace building is associated with increased incidence and decreased remission of SBS, as well as increased bronchial responsiveness and eosinophilic inflammation.

1. Introduction

Non-specific building related symptoms, often called sick building syndrome (SBS) has been an important issue the last decades and has been defined by a working group of WHO (WHO, 1983). The term SBS has been used to describe symptoms (including headaches, fatigue, and irritation in the upper respiratory tract, nose, throat, eyes, hands and/or facial skin) that can be influenced by the indoor environment (Redlich et al., 1997; WHO, 1983). Various factors reported to influence SBS, such as personality trait, work stress, female gender, air contaminants, VOCs, molds and low ventilation rates (Burge, 2004; Hansen et al., 2008; Norback et al., 1990; Runeson et al., 2004; Takigawa et al., 2010, 2009; Teeuw et al., 1994; Zhang et al., 2011).

Dampness in buildings, such as water damage, wet spots, visible mold or mold odor, is a common problem in many countries (Ruotsalainen et al., 1995; WHO, 2009). It has been shown that building dampness and mold have an increased risk of respiratory symptoms, asthma-related symptoms and SBS (Fisk et al., 2007; WHO, 2009), and one study has found an association between microbial VOC (MVOC) and SBS (Araki et al., 2010). A review study concluded that dampness in buildings is a risk factor for respiratory health effects among atopic and non-atopic subjects both in domestic and public environments (Bornehag et al., 2004), but there are few studies on associations between dampness or molds in workplace buildings and SBS (Chao et al., 2003; Ruotsalainen et al., 1995; Wan and Li, 1999).

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The sick building syndrome includes symptoms from the ocular and nasal symptoms, and biomarkers have been used to study environmental effects on the ocular and nasal mucosa (Norback and Wieslander, 2002). Some previous studies have shown that building dampness could influence biomarkers in the ocular and nasal mucosa. In one office building study, the concentrations of eosinophil cationic protein (ECP), MPO and albumin were higher among workers in the damp building than among controls (Walinder et al., 2001). Another study found that, dampness in the floor construction was associated with an increase of lysozyme in nasal lavage fluid (NAL) in two geriatric hospitals (Wieslander et al., 1999). Finally, teachers in Finland working in a moldy school building had higher concentrations of tumor necrosis factor-alpha, interleukin-6 (IL-6) and nitrate in NAL when compared with unexposed controls (Hirvonen et al., 1999).

It is unclear to what extent SBS is related to biomarkers of IgE mediated allergy and upper or lower airway inflammation. Two studies have shown that clinically verified atopy is related to SBS (Bjornsson et al., 1998; Malkin et al., 1998), while others have failed to find such associations (Norbäck, 2009; Runeson et al., 2006) One prevalence study have reported an association between serum myeloperoxidase (MPO), an indicator of neutrophilic inflammation and SBS (Metso et al., 1993).

Within the Uppsala part of the European Community Respiratory Health Survey (ECRHS), a large longitudinal multi-centre study in young adults, biomarkers of allergy and lower airway inflammation have been measured. We have previously published data on incidence and remission of SBS in relation to the indoor environment in the dwelling (Sahlberg et al., 2012), but we have found no previous cohort study on effects of dampness and molds in workplace buildings on biomarkers or incidence and remission of SBS.

In this study, our first aim was to study the prevalence and changes of SBS symptoms and environment exposures both in baseline and follow-up. Moreover, we studied the incidence and remission between SBS symptoms and building dampness. A further aim was to study the associations between reports on dampness and molds and bronchial responsiveness (BR) as well as selected biomarkers in serum or blood. The biomarkers included eosinophilic cationic protein (ECP) and eosinophil counts (EOS), both markers of eosinophilic inflammation. Moreover we analyzed associations for high resolution C reactive protein (HCRP), serum immunoglobulin E (IgE) and interleukin- 6 (IL-6).

2. Materials and methods

2.1. Study design and study population

The European Community Respiratory Health Survey (ECRHS) is a multi-centre study performed in 48 centers in 23 countries throughout the Europe. This study is based on data from the centre in Uppsala, Sweden. A short questionnaire with questions on personal factors, asthma and asthma-related symptoms was mailed to a population sample of 3600 individuals (20–44 years of age) from the municipality of Uppsala, randomly chosen from the Uppsala county council population register. Then, a random sample of 800 subjects, and 261 additional symptomatic subjects not included in the random sample, were selected from the population sample of 3600 subjects for the clinical test. Of these, 562 answered the self-administered questionnaire on SBS symptoms and building characteristics in 1992. Symptomatic subjects were those who had reported attacks of asthma during past 12 months, nocturnal breathlessness during the past 12 months, or current use of asthma medication. A follow-up study ECRHS II was carried out in 2002 in Uppsala using the same study protocol (Janson et al., 2001). The population included in this study is restricted to subjects who participated both at ECRHS and ECRHS II, and who answered the additional questionnaire on SBS symptoms and building characteristics of the workplace building in both the initial study and follow-up. The occupations reported at baseline were classified in accordance with the Nordic Classification of Occupations (NYK82) which is based on the International Standard Classification of Occupations (ISCO) with a few modifications (Norback and Edling, 1991).

2.2. Information on questionnaire

The subjects answered an additional questionnaire to collect data about symptoms compatible with SBS prior to the medical investigation. Symptoms reported during the preceding three months included five dermal symptoms (facial itch; facial rash; hand itch; hand rash; eczema), seven mucosal symptoms (eye irritation; swollen eyelids; nasal catarrh and obstruction; dryness in the throat; sore throat; irritating cough) and four general symptoms (headache; nausea; sensation of getting a cold; tiredness). Each question had 3 alternative answers: no-never, yes, sometimes, yes often (every week). In the statistical calculations, weekly symptoms were coded 1 and no-never and yes sometimes were coded 0. In addition, a question about whether any of the SBS symptoms improved when they stayed away from workplace was included. The same questionnaire was used at follow-up.

The additional questionnaire also had a number of questions on the working environment, not available in the ECRHS questionnaire. It requested information on the current work environment, including building age, environmental tobacco smoke (ETS), and four questions on dampness or indoor molds in the current workplace building (water leakage, visible mold, mold odor and signs of floor dampness). The same questions were repeated in the follow-up study, and in addition there were question on ever exposed to dampness or molds at any workplace during follow up period of 10 years (cumulative exposure). The exact wordings of the question on cumulative exposure are: Has any of the following been identified in you workplace building during the past 10 years? Water leakage or water damage indoors on walls, floors or ceilings; Bubbles or yellow discoloration on plastic floor covering, or black discoloration on parquet floor; Visible mold growth indoors on walls, floor or ceilings; Smell of mold in one or more rooms (not the basement). Then there was one additional question on repairing of the workplace building: Has your workplace building been repaired during the past 10 years because of building dampness or water damage?

2.3. Clinical investigation

We measured forced vital capacity (FVC) and forced expiratory volume in 1 s (FEV1) using the Spiro Medics computerized dry-rolling seal spirometer system 2130 (Sensor Medics, Anaheim, CA, USA). Bronchial responsiveness (BR) was performed using standardized techniques (Chinn et al., 1997). BR to methacholine was presented as "slope" using the following formula: 100/(regression coefficient + 10), where the regression coefficient was calculated by regressing percentage falls in forced expiratory volume in 1 s on log10 (methacholine dose). A lower slope value indicates a higher degree of bronchial responsiveness.

Blood and serum samples were collected and stored at −20 C for measurement of ECP and EOS at baseline, CRP and IL-6 at follow-up and total levels of IgE at both occasion (Dept. of Clinical Biochemistry, Landspitali-University Hospital, Iceland). The same procedure for the clinical investigations was used both in 1992 and 2002.

Specific IgE in serum was measured at a central laboratory. Specific serum immunoglobulin E (IgE) levels against cat, timothy grass and the mold, *Cladosporium herbarium*, and the house dust mite, *Dermatophagoides pteronyssinus*, were determined by using Pharmacia CAP system (Pharmacia diagnostics, Uppsala, Sweden) in both ECRHS and ECRHS II. A level above 0.35 kU/L was considered a positive reaction. Skin prick tests were only performed in ECRHS I. A