

Upper respiratory impairment in restorers of cultural heritage

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Background	There is a paucity of data regarding respiratory health in restorers of cultural heritage or similar occupations, such as visual artists or museum workers, although they are exposed to a complex mixture of various respiratory hazards.
Aims	To evaluate atopy and respiratory health parameters, including bronchial and nasal non-specific reactivity, in restorers and conservators of cultural heritage (restorers).
Methods	Fifty-six restorers and 62 controls provided general data and data on ever experienced rhinitic or asthma-like symptoms, spirometry, non-specific bronchial and nasal responsiveness to histamine, skin prick testing to common inhalational allergens and serum total IgE levels.
Results	Spirometry values were in the range of normal values in 55 of 56 restorers and did not differ significantly from those in control subjects. However, restorers had more than two times higher prevalence of nasal hyper-responsiveness (NHR), with 2.3 times higher risk of NHR compared to controls [95% confidence interval (CI): 1.4–3.6, $P < 0.001$]. The risk of NHR was slightly reduced by increasing age (odds ratio 0.95, 95% CI: 0.91–0.99, $P < 0.05$). NHR was not associated with gender, smoking status, bronchial hyperresponsiveness (BHR), upper or lower respiratory symptoms or atopy status.
Conclusions	Compared with controls, the studied group of workers occupationally exposed to respiratory hazards during restoration/conservation activities had no deterioration of lung function but had an increased non-specific nasal responsiveness that was not correlated with upper and lower respiratory symptoms, BHR or atopy. The relationship of this finding to future clinical outcome should be investigated in a longitudinal study.
Key words	Cross-sectional studies; nasal provocation tests; occupational exposure.

Introduction

Restoration and conservation of cultural heritage involves a variety of occupational hazards. From the toxicological point of view, restorers/conservators (further referred to as 'restorers') are usually exposed to complex mixtures of different types of harmful substances that could be responsible for a wide array of toxic effects, from mildly irritative changes in the upper airways induced by nuisance dust to the carcinogenic effects of certain paints and pigments [1–4]. Nevertheless, there is a paucity of data on respiratory health and atopic status in these workers [3,4]. Restoration/conservation activities regularly implies exposure to a number of substances able to exert irritative and/or allergenic effects on the respira-

tory system, including ammonia, bleach, acids and alkalis, ethyl and acetyl acetate, certain solvent vapours, turpentine, metal fumes, dye and pigment dust, wood-working, stone and clay, organic dusts containing endotoxin and β (1 $\rightarrow$ 3)-glucans, moulds and mites, certain paints and toluene diisocyanate [5,6].

The aim of the present study was to evaluate atopy and respiratory health parameters, including non-specific bronchial and nasal reactivity, in restorers/conservators employed in two Croatian institutions dedicated to restoration, conservation and archiving of cultural heritage. To our knowledge, respiratory health has never been evaluated by upper and lower airway reactivity markers in restorers/conservators of cultural heritage or in similar occupations, such as visual artists or museum workers.

Methods

The study was designed as a cross-sectional study. All subjects were recruited on a voluntary basis after an announcement given by their employers, during the years 2004–05. Restorers were recruited from departments for restoration and conservation of two Croatian institutions dedicated to restoration, conservation and archiving of cultural heritage. Control subjects were administrative workers in an insurance company and librarians. Exclusion criteria were acute upper or lower respiratory tract infections and medical conditions that contraindicate scheduled clinical tests or affect reliability of test results. Figure 1 shows the selection of study subjects.

The study was designed in accordance with the Declaration of Helsinki and was approved by the Ethical Committee of the Institute for Medical Research and Occupational Health, Zagreb. All subjects signed informed consent and they were free to leave the study at any time. Study protocol included medical interview and physical examination, venous blood sampling for total IgE measurement, skin prick testing (SPT) to common inhalational allergens (including testing with

negative and positive control solutions) and non-specific bronchial challenge test on Test Day 1 and non-specific nasal challenge test on Test Day 2 (48 h after Test Day 1).

Medical history, including smoking habit, years of working exposure at the present or similar workplace, exposure to dust and chemicals at the workplace and data on ever experienced respiratory symptoms compatible with diagnoses of rhinitis and asthma, were recorded using a simple questionnaire. Upper airway symptoms were compatible with the recent Allergic Rhinitis and its Impact on Asthma Workshop guidelines' definition of allergic rhinitis and included sneezing, rhinorrhoea and nasal obstruction (not related to the common cold) [7]. Lower airway—asthma-like symptoms included episodic dry cough (not related to the common cold), wheezing, chest tightness and dyspnoea were compatible with the Global Initiative for Asthma guidelines for diagnosing asthma [8]. The variables 'positive rhinitic symptoms' and 'positive asthma-like symptoms' were defined as the presence of at least two upper or lower airway symptoms, respectively. Smoking habit was analysed as a variable with three categories: never-smokers, former smokers (all subjects stopped smoking >1 year ago) and current smokers.

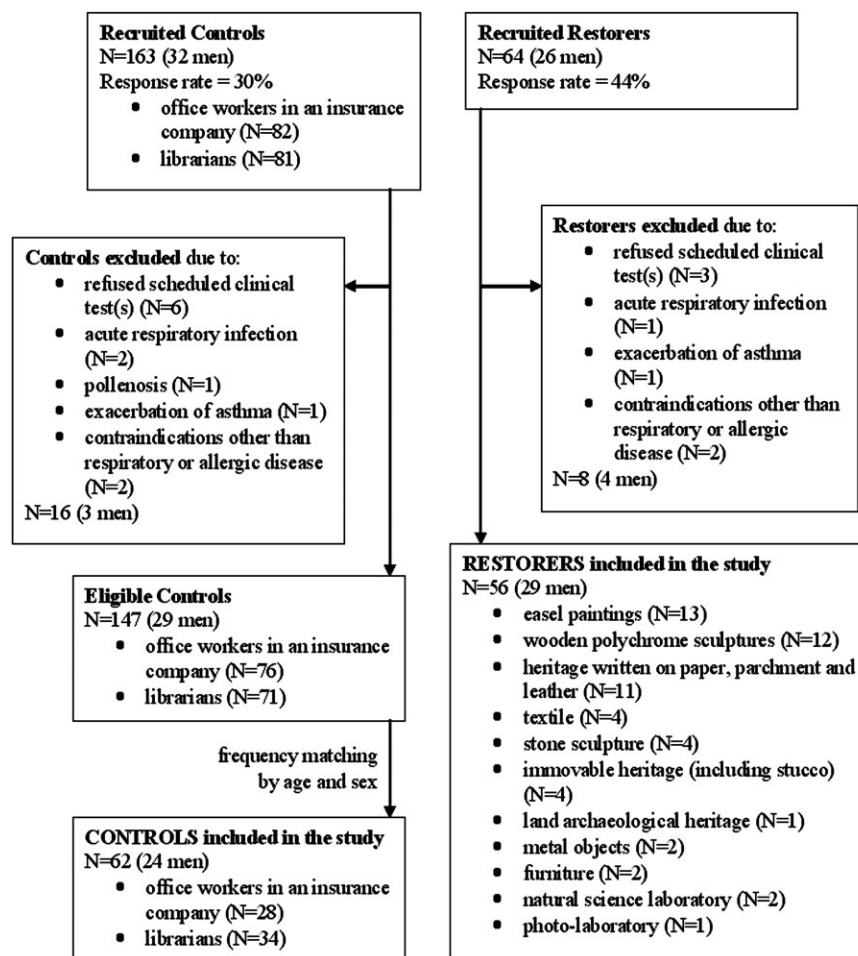


Figure 1. Selection of study subjects.

For current smokers, smoking consumption was expressed as smoking index (number of cigarettes smoked/day multiplied by number of smoking years). Overall exposure pattern was typical for restoration/conservation occupation: a complex mixture of different types of dust and chemicals. Almost all restorers ($\geq 90\%$) reported exposure to mainly organic or mixed organic and inorganic dusts. Seven subjects, engaged in restoration of stone sculptures, metal objects and land archaeological heritage, reported to be primarily exposed to inorganic dust. Dust originated mainly from material being restored, namely paper, textile, leather, paintings, wood, stone, clay and in rare cases metal, and comprised of inorganic and organic constituents, including microorganisms and biologically active substances such as endotoxins and β (1 $\rightarrow$ 3)-glucans. Table 1 shows the most common chemicals used in restoration/conservation of easel paintings, wooden polychrome sculptures, heritage written on paper, parchment and leather, textile and stone sculptures (i.e. departments from which more than two employees were recruited in the present study). Chemical exposure varied according to restoration/conservation activity, but all subjects were exposed to a mixture of organic solvents,

including 95% ethanol, acetone, benzene, white spirit, toluene and xylene. Many of them also had in common exposure to turpentine, ammonia, perchloroethylene, fungicides, vinyl polymers and epoxy resins.

Spirometry, non-specific bronchial and nasal challenge test, SPT and serum total IgE antibodies analysis have been described elsewhere [9–11]. Forced vital capacity and forced expiratory volume in 1 s (FEV₁) were determined by a standard method using the spirometer Pneumoscreen II (Jaeger, Würzburg, Germany) [12] and were expressed and analysed as a percentage of referent values (CECA II). Non-specific bronchial reactivity was assessed by histamine challenge testing [13]. Bronchial hyperresponsiveness (BHR) was established if after the inhalation of ≤ 8 mg/ml of histamine (as diphosphate saline solution) FEV₁ dropped $\geq 20\%$ of the value measured after the inhalation of the control solution (saline). Non-specific nasal reactivity was assessed by means of active anterior rhinomanometry after histamine challenge testing [14]. Standard titrations of histamine diphosphate saline solutions were sprayed from spray bottles (Lomudal Nasal, Fisons Ltd, West Malling, UK) into both nostrils every 3 min. The starting concentration of

Table 1. Examples of chemicals used in art restoration/conservation processes

Object of art	Chemicals
Easel painting	Cleaning agents: 95% ethanol, acetone and other ketones, benzene, white spirit, varnish solvents, enzymes, resin soaps Adhesives: glues, synthetic resins including polyvinyl acetate and ethylene-vinyl acetate copolymer Paints: pigments (some of them containing toxic metals), ammonia, powdered lime, turpentine Varnishes: polymeric (hydrogenated hydrocarbon resins, acrylic polymers), natural resins (terpene resin, triterpenoid resin), ketone resins (cyclohexanone polycondensation resin), varnish solvents (xylene, toluene, isopropanol, ethanol, acetone, white spirit, turpentine)
Wooden polychrome sculpture	Cleaning and deinfestation: detergents, 95% ethanol, acetone, toluene, methylene chloride, turpentine, ammonia, fungicides Repairing: resin glues, dry casein glues, glue solvents (hexane, mineral spirits, naphtha, 1,1,1-trichloroethane) Polychrome finishes: pigments and dyes (earth pigments such as umbers, ochres, siennas, chalk, Indian red and terra verde; ash black, natural tar, metal complex pigments, synthetic organo metallic and azo dyes), waxes (beeswax, mineral or synthetic waxes), linseed oil, walnut oil, tung oil, synthetic resins (e.g. alkyds) or natural resins (e.g. shellac, amber, dammar), varnish solvents (turpentine, white spirit, ethanol, methanol)
Heritage written on paper, parchment and leather	Deinfestation and disinfection: ethanol, thymol Cleaning: detergents, varnishes with methanol, ethanol or acetone, toluene, dimethyl formamide, acetic acid, diethylene glycol, polyethylene glycol, perchloroethylene Fixing of inks: acetone, 95% ethanol, xylene, toluene, perchloroethylene Bleaching: citric acid, chloramine-T Deacidifying agents: calcium hydroxide, magnesium oxide Hygroscopic stabilization: lanolines, waxes, paraffins, glycerines, castor oil, cedar oil Adhesives: vinyl polymers, semi-synthetic cellulose derivatives, glues
Textile	Cleaning and disinfection: detergents, organic solvents (e.g. ethanol, methanol, perchloroethylene), fungicides (e.g. <i>p</i> -dichloro-benzene, thymol, eugenol, <i>p</i> -chloro- <i>m</i> -cresol, chlorophenols) Repairing: polymers and resins (methyl hydroxy ethyl cellulose, ethyl acrylate, methyl methacrylate, vinyl acetate, butylacrylate, ketone resin)
Stone sculpture	Deacidification: calcium or barium hydroxide, methoxy magnesium methyl carbonate Cleaning: organic solvents (e.g. turpentine), ammonia, acids and alkalies, urea Repairing and consolidation: epoxy resins and curing agents for epoxy compounds, silicate materials, benzene, turpentine, ethanol, acetone Contamination prevention: vinyl polymers, waxes

histamine was 0.5 mg/ml, and the maximum dose used was 8 mg/ml. Nasal responsiveness was measured by means of total nasal airway resistance (NAR) after each inhaled dose on spirometer Pneumoscreen II (Jaeger) using the attached facemask and open interruption method. Nasal hyper-responsiveness (NHR) was established if after spraying of ≤ 4 mg/ml of histamine, total NAR increased $\geq 75\%$ of the value measured after spraying of the control solution (saline), and further testing was stopped. SPT was performed using a standard method with a panel of common commercial inhalational allergens: grass pollen mixture, birch, hazel, weed (*Ambrosia elatior*, *Artemisia vulgaris*) pollens, mites *Dermatophagoides pteronyssinus*, *Dermatophagoides farinae* and *Lepidoglyphus destructor*, cat, dog and moulds *Cladosporium herbarum* and *Alternaria alternata* (Allergopharma, Reinbeck, Germany) [15]. The results of SPT were considered positive (positive SPT) when the mean wheal diameter was larger than the negative control (buffer solution) for >3 mm to at least one tested allergen. Serum total IgE antibodies were measured from venous blood samples by the ELISA method (IASON, Graz, Austria) [16]. IgE levels were expressed in kIU/l. Values ≥ 150 kIU/l were considered elevated.

Asthmatic subjects were defined as subjects with a history of asthma-like symptoms (as defined above) plus BHR. According to the recent revision of nomenclature for allergy [17], atopics were defined as subjects with history of rhinitic or asthma-like symptoms (as defined above) plus elevated IgE and/or positive SPT.

Statistical analysis was performed using software Stata/SE 10.0 for Windows (StatCorp LP, TX, USA). All results were considered statistically significant at a P value < 0.05 . Univariate statistics (Student's t -test, Wilcoxon rank-sum test, Pearson χ^2 test or Fisher's exact test) was applied to analyse differences in general, respiratory and atopic parameters between two groups. All tests were two tailed.

The only respiratory health or atopic parameter that differed between groups, namely NHR, was tested as dependent variable in a multiple logistic regression model to evaluate its relationship to other studied parameters. Predictors in the model were age, gender, smoking status, asthma-like and rhinitic respiratory symptoms, BHR, atopy and exposure as restorer (coded as follows: restorers as 1 and controls as 0). FEV₁ was omitted from both models due to collinearity with other predictors. Risk ratio and unadjusted odds ratio (OR) for NHR were also calculated.

Results

After the exclusion of non-eligible subjects, 56 restorers (22 men) and 147 control subjects (29 men) were included in the study. The controls differed significantly from the restorers group by having a greater proportion

of women (80%) and higher mean age. Since these variables could affect predictors and outcomes of interest in our study, a proportionate stratified random sample of 24 male and 38 female controls was frequency matched to restorers by gender and age. The final control group comprised 28 workers employed in an insurance company and 34 librarians (who were not involved in restoration/conservation activities). The majority of restorers were engaged in restoration and conservation of easel paintings ($n = 13$ subjects), wooden polychrome sculptures ($n = 12$), heritage written on paper, parchment and leather ($n = 11$), textile ($n = 4$) and stone sculpture ($n = 4$). The rest ($n = 12$) were recruited from departments for restoration and conservation of immovable heritage (including stucco), land archaeological heritage, metal objects and furniture and from chemical and photo-laboratory.

Except for age and gender ratio, frequency matched controls (62 subjects) did not differ from the rest of the eligible control subjects ($n = 85$) in observed general and health parameters. This suggests that the matching procedure did not produce obvious selection bias. Since the control group comprised subjects from two types of working environment (insurance company offices and library), possible differences in analysed variables were tested between these two subgroups prior to main data analysis. Apart from age [administrative workers from the insurance company were on average 6 years older than librarians (41.8 ± 1.74 versus 35.9 ± 1.48 ; $t = 2.58$, $P < 0.05$)], there was no significant differences in demographic factors between both subgroups. They were, therefore, analysed as one group in further data evaluation.

Table 2 shows data about general characteristics and Table 3 data regarding respiratory and atopic parameters

Table 2. General characteristics of restorers and control subjects

	Controls	Restorers
<i>n</i> (number of men)	62 (24)	56 (22)
Age in years, mean $\pm$ SD	38.6 $\pm$ 9.3	37.6 $\pm$ 10.0
Working exposure in years, median (interquartile range, 25–75%)	6.5 (4–10)	6.0 (4–12.5)
Smoking status, <i>n</i> (%)		
Never-smoker	32 (52)	24 (43)
Former smoker	9 (15)	16 (29)
Current smoker	21 (34)	16 (29)
Smoking index for current smokers ^a (interquartile range, 25–75%)	140 (90–360)	180 (70–400)

Differences between controls and restorers were analysed by Student's t -test (age), Wilcoxon rank-sum test (working exposure and smoking index) and Pearson χ^2 test (smoking status). There were no statistically significant differences between groups.

^aNumber of cigarettes smoked/day multiplied by number of smoking years.

in restorers and control subjects. Age, years of working exposure and smoking parameters did not significantly differ between restorers and controls, and there were no differences in the prevalence of rhinitic and asthma-like symptoms.

Spirometry values were similar in controls and restorers, as well as the prevalence of below-normal values of FEV₁ ($\leq 80\%$ predicted). BHR was evenly distributed between study groups. The risk ratio for NHR in restorers compared with controls was 2.3 (95% confidence interval (CI): 1.4–3.6), and the unadjusted OR was 4.4 (1.9–10.4). The prevalence of NHR was also analysed in subjects without rhinitic or asthma-like symptoms (25 controls and 26 restorers). NHR was again significantly more prevalent in restorers compared with controls (16 versus 6 subjects with NHR, respectively; $\chi^2 = 7.32$, $P < 0.05$), with the risk ratio in restorers compared to controls of 2.6 (1.2–5.5) and unadjusted OR of 5.1 (1.3–20.6). Age, smoking habit, working exposure, spirometry values, BHR and atopy markers did not differ between asymptomatic controls and restorers.

The prevalence of positive SPT and elevated serum total IgE, as well as the number of atopic subjects, did not

significantly differ between groups, as well as serum total IgE levels.

Similar results for comparisons controls versus restorers were obtained for men and women analysed separately (data not shown).

Multiple logistic regression analysis with NHR as dependent variable confirmed the observation obtained by univariate statistics and unadjusted OR, showing that the odds of NHR in restorers were 4.6 times the odds in controls (95% CI: 1.97–10.9), while controlling for age, gender, smoking habit, respiratory symptoms and atopy status. NHR was not associated with gender, smoking status, respiratory symptoms or atopy and had a weak negative relationship with age (OR 0.95, 95% CI: 0.91–0.99) (Table 4).

Discussion

Our study found that there was no difference in the prevalence of respiratory symptoms, atopy markers, spirometry values and bronchial reactivity between employees involved in conservation and restoration activities and controls. However, this observation could be the result of selection bias, i.e. greater participation in the study among persons with known respiratory health problems. Compared with data obtained from population-based studies in Croatia [10], our study revealed almost twice the expected prevalence of nasal symptoms and more than four times the expected prevalence of lower respiratory symptoms in both controls and restorers. Atopy was also

Table 3. Respiratory and atopic parameters in controls and restorers

	Controls	Restorers	<i>P</i>
<i>n</i>	62	56	
Rhinitic symptoms, <i>n</i> (%)	23 (37)	17 (30)	NS
Asthma-like symptoms, <i>n</i> (%)	8 (13)	4 (7)	NS
Spirometry			
FVC, mean $\pm$ SD	107.8 $\pm$ 11.6	108.78 $\pm$ 12.3	NS
FEV ₁ , mean $\pm$ SD	104.9 $\pm$ 11.7	107.1 $\pm$ 13.7	NS
FEV ₁ below normal ($\leq 80\%$), <i>n</i> (%)	3 (5)	1 (2)	NS
BHR, <i>n</i> (%)	13 (21)	13 (23)	NS
NHR, <i>n</i> (%)	17 (27)	35 (63)	<0.001
Serum total IgE in kIU/l, median (interquartile range, 25–75%)	19.4 (3.3–57.0)	32.7 (16.7–80.2)	NS
Elevated IgE (≥ 150 kIU/l), <i>n</i> (%)	10 (16)	8 (14)	NS
Positive SPT, <i>n</i> (%)	26 (42)	21 (38)	NS
Asthmatic subjects, <i>n</i> (%)	5 (8)	2 (4)	NS
Atopic subjects, <i>n</i> (%)	18 (29)	11 (20)	NS

P, significance level for difference between controls and restorers analysed by Pearson χ^2 test (rhinitic symptoms, BHR, NHR, elevated IgE, positive SPT and number of atopic subjects); Fisher's exact test (asthma-like symptoms, below normal FEV₁ and number of asthmatic subjects); Student's *t*-test (FVC and FEV₁) and Wilcoxon rank-sum test (serum total IgE). There were no subjects with FVC below normal ($\leq 80\%$ predicted). FVC, forced vital capacity expressed as percentage of referent values (CECA II); FEV₁, forced expiratory volume in 1 s expressed as percentage of referent values (CECA II); BHR, non-specific bronchial hyper-responsiveness; NHR, non-specific nasal hyper-responsiveness.

Table 4. Logistic regression analysis for non-specific NHR in controls and restorers

	Non-specific NHR	
	OR (95% CI)	<i>P</i>
Gender	1.34 (0.56–3.22)	NS
Age	0.95 (0.91–0.99)	<0.05
Smoking status		
Never-smoker ^a	1 (ref)	–
Former smoker	1.29 (0.40–4.19)	NS
Current smoker	0.73 (0.28–1.88)	NS
Positive airway symptoms		
Rhinitic	2.59 (0.74–9.14)	NS
Asthma-like	2.49 (0.51–12.1)	NS
BHR	1.94 (0.67–5.64)	NS
Atopy	0.26 (0.06–1.22)	NS
Exposure as restorer	4.63 (1.97–10.9)	<0.001
<i>P</i> -value for the model	<0.01	
Pseudo <i>R</i> ²	0.16	

Study included 62 control subjects (24 men) and 56 restorers (22 men). BHR, non-specific bronchial hyper-responsiveness.

^aNever-smoker is set as the reference category. FEV₁ was omitted from the model due to collinearity with other predictors.

prevalent in both restorers and controls (20–29%, respectively) compared to the 12% prevalence observed in general population [10]. This suggests selection bias in the control group. Higher than expected prevalence of respiratory symptoms and atopy in restorers could be the result of their impaired respiratory health but could also be a consequence of selection bias. Selection bias and rather low response rates limit the interpretation of respiratory symptoms and atopy prevalence in this study. To circumvent selection bias for objective respiratory health parameters, only asymptomatic controls and restorers were compared, and the regression model was adjusted for the presence of respiratory symptoms.

Only one studied health parameter, NHR, differed between restorers and controls. Compared with controls, restorers had more than two times higher prevalence of NHR with a risk ratio of >2 even when only asymptomatic subjects were compared. NHR was not related to gender, smoking status, spirometry values, BHR and atopy nor with rhinitic symptoms indicating occupational exposure as the most likely causative factor. Occupational exposures to various respiratory hazards, even without exposure to high concentration of these substances, have been associated with rhinitic symptoms, nasal airflow obstruction and/or nasal inflammation. The significance of so far insufficiently defined conditions of occupational rhinitis and work-exacerbated rhinitis was recently emphasized [5]. Nasal reactivity has been insufficiently investigated in workers exposed to respiratory hazards, but increased non-specific nasal reactivity to histamine was established in workers exposed to chemical respiratory irritants [18], in teachers exposed to dampness in school [19] and in healthy volunteers exposed briefly to swine dust [20]. Specific nasal reactivity with occupational allergens was suggested as a valuable method for confirmation of the link between rhinitic symptoms and potential occupational causative agents [21]. On the other hand, an adequately specific and standardized non-specific nasal provocation test does not exist at present, due to the clinical and pathophysiological heterogeneity of rhinitic patients [22]. The limitation of the nasal provocation test with histamine mostly applies to the fact that a significant effect is only observed in rhinitic subjects where the obstructive component predominates ('blockers'), while notable glandular secretion is not induced [22]. The mechanisms responsible for increased nasal reactivity in subjects occupationally exposed to respiratory hazards other than known specific allergens are not clear. Nasal hyper-reactivity related to organic dust exposure probably occurs due to the neutrophilic inflammation caused by endotoxins and other proinflammatory substances mixed in organic dust [20]. Up-regulation of histamine receptors in nasal mucosa was suggested after exposure to diesel exhaust particles [23]. Lower threshold was found for irritative nasal symptoms after provocation with odour in laboratory technicians exposed to organic

solvents [24], and it was suggested that irritant-induced nasal congestion is predominantly related to up-regulated nasal neuroreflexes, including autonomic nervous system and trigeminal reaction [25,26]. Our results indicate that changes in nasal reactivity of the restorers occupationally exposed to various respiratory irritants and allergens could be found in subjects without expression of respiratory symptoms. Increased prevalence of NHR was also found in asymptomatic subjects living in sick buildings [27]. The clinical significance of NHR in asymptomatic subjects is uncertain. As pointed out in the recent European Academy of Allergy and Clinical Immunology position paper on occupational rhinitis [5], asymptomatic subjects with BHR had an increased risk for development of asthma compared with subjects with normal bronchial responsiveness [28,29]. Whether this pattern also applies to NHR is not yet clarified and should be investigated in a follow-up study design both in atopics and non-atopics, preferably accompanied by biomarkers of inflammation in nasal mucosa.

Among the limitations of the study, we would like to point out that we were not able to evaluate occupational risks related to a particular type of restoration/conservation activity, primarily due to rather small number of subjects in each restoration department (from 1 to 13 subjects). Nevertheless, all studied restorers had exposure to respiratory irritants in common, including both dusts and number of chemicals, and the majority of them were also exposed to respiratory allergens, related mostly to organic dust. Additionally, exact risk assessment for this occupation is difficult to obtain due to varying levels of exposure depending on the restored object and the degree of damage, a number of non-standardized restoration techniques and working environment that includes on-site activities without proper ventilation and personal respiratory protection [30]. Another issue concerns selected control subjects that comprised office workers and librarians. In both occupations, a certain level of exposure to dust can be expected. However, the librarians included in the study worked in a modern office with appropriate hygienic and climatic working conditions and were not exposed to contaminated archival material nor engaged in restoration/conservation activities.

To conclude, in spite of occupational exposure to various respiratory irritants and allergens in restorers, spirometry did not indicate clinically relevant impairments of respiratory health in the restorer group. However, significantly increased non-specific NHR to histamine was found in these workers in comparison with control subjects. Increased reactivity of nasal mucosa was not related to rhinitic symptoms, indicating subclinical changes in nasal mucosa of restorers, probably related to their occupational exposure to respiratory hazards. The relevance of this finding for the future clinical course of occupational or work-exacerbated rhinitis should be elucidated through further studies, as well as the underlying mechanisms.

Key points

- More than two times higher prevalence of nasal hyper-responsiveness to histamine was found in restorers and conservators of cultural heritage compared to control subjects.
- Non-specific nasal hyper-responsiveness was not related to rhinitic or asthma-like symptoms, non-specific bronchial hyper-responsiveness, atopy status, gender or smoking status.
- No deterioration of lung function was found in the studied group of restorers.

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Conflicts of interest

None declared.

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