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Nasal symptoms among workers exposed to soft paper dust

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Abstract ***Objectives:*** To clarify whether occupational exposure to paper-dust is associated with an increased risk of non-infectious rhinitis. ***Methods:*** Thirty-seven workers exposed to paper-dust in a soft-paper mill were compared with 36 unexposed controls. The study was performed under normal working conditions during the non-pollen season. Medical and occupational history was taken down in a comprehensive questionnaire and nasal symptoms were scored on a visual analogue scale (VAS). Pulmonary and nasal function was assessed by spirometry, acoustic rhinometry and peak nasal inspiratory flow. Nasal lavage was analysed for interleukin-8 (IL-8) and nasal transit time was monitored with the saccharine test. Concentrations of inhalable dust for each exposed subject during the day of the clinical study were measured with personal sampling devices. ***Results:*** There was an increased prevalence of nasal blockage and crust formation among the exposed workers. However, there was no difference with regard to acoustic rhinometry, nasal transit time or nasal peak inspiratory flow. In the whole population, IL-8 in nasal lavage was higher among men than among women, 193 ng/l vs 132 ng/l, $P = 0.006$. There was also a positive trend ($P = 0.01$) with increasing nasal IL-8 going from non-smokers (122 ng/l), ex-smokers (126 ng/l) to current smokers (235 ng/l). ***Conclusions:*** We have found that occupational exposure to paper-dust is associated with symptoms of nasal blockage and nasal crusting. We find no objective signs of nasal inflammation, even among the subgroup with the highest current exposure.

Key words Paper-dust · Nasal blockage · Acoustic rhinometry · Nasal peak inspiratory flow · IL-8

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Introduction

Previous studies have indicated that the prevalence of symptoms from both the upper and lower respiratory tract is increased among workers in paper mills, especially soft-paper mills (Ericsson et al. 1988, Thorén et al. 1989a, Zuskin et al. 1998). Regarding the upper airways, it has been suggested that the underlying mechanism is an inflammatory reaction in the nasal mucosa. However, to our knowledge there are no clinical studies that verify this (Zuskin et al. 1998). In the present study, we have investigated nasal function in a group of paper-dust exposed workers from the converting process at a soft-paper mill, and compared them with unexposed controls from the same mill.

The aim of the study was to determine whether nasal symptoms among paper-dust exposed workers are associated with objective signs of nasal inflammation.

Subjects and methods

Subjects

The study population consisted of a random sample of 42 workers with an exposure time to paper-dust exceeding 8 years. The workers were selected from workers in two converting halls in a large Swedish soft-paper mill. As referents, all unexposed office workers ($n = 41$) were selected. From the original sample, five exposed workers and five controls were excluded. The reasons were pregnancy ($n = 1$), absence because of a sick child ($n = 2$), otitis ($n = 1$) and unwillingness to participate ($n = 6$). Hence, the study was performed on 37 exposed workers and 36 controls. The subjects are described in Table 1.

Table 1 Study population with regard to age, gender, smoking habits and atopy (standard error)

	Exposed $n = 37$	Controls $n = 36$
Gender		
Males	12	19
Females	25	17
Age (years)	49.3 (1.2)	49.8 (1.2)
Non-smokers	12 (32%)	17 (47%)
Ex-smokers	7 (19%)	14 (39%)
Smokers	18 (49%)	5 (14%)
Atopy	5 (14%)	6 (17%)
Employment time (years)	23.2 (1.0)	21.7 (1.7)

Methods

The study was conducted during 3 weeks of ordinary work-shifts during the non-pollen season. All subjects received a questionnaire by mail, with questions about physician's diagnosed asthma, wheezing and dyspnoea, smoking habits, duration of employment and type of work. The questionnaire has been used previously (Torén et al. 1996). In addition, questions about nasal congestion, sneezing, nasal itching, nasal secretion and nasal crusts were used. The nasal items were worded "Since the age of 18, have you suffered from sneezing etc. for more than three consecutive days?" In addition, the change in nasal symptoms during the workday was monitored by the question "How does the nasal congestion/sneezing/secretion etc. change during a workday?" Each questionnaire was checked for missing answers before being accepted. Presence of atopy was defined as an affirmative answer to the question "As a child, did you suffer from any kind of allergy such as eczema, asthma or hayfever?"

The dust sampling and the clinical investigation were performed at random during one 8-h work-shift. All subjects were investigated only once in the same day and in the following order: spirometry, ENT-investigation, acoustic rhinometry, nasal peak inspiratory flow, nasal lavage and nasal transit time.

Weight and height were measured for each subject. Spirometry was performed with a dry spirometer, Vitalograph, according to the American Thoracic Society (ATS 1979). All spirometries were performed by the same trained nurse. All subjects were investigated by an ENT specialist (J.H.). Visible inflammation was regarded as being present if red mucous membranes were seen. Acoustic rhinometry was performed with a Rhin 2000, SR Electronics (Rhinometrics, Copenhagen) with anatomical nosepieces (Rhinometrics) and Vaseline contact-gel. The probe was hand-held and the subject was sitting in an upright position holding the breath during the measurement (Hilberg et al. 1989). The minimum cross-sectional area at 0–3 cm from the nostril (MCA1) as well as the volume in the same interval (Vol 1) were determined separately for the left and right nostril, and the results are presented as the mean of the two sides. Acoustic rhinometry was performed once on each side. Nasal peak inspiratory flow (NPIF) was determined with a commercially available NPIF meter, (Youlten, Airmed, Sussex, UK). The best of three consecutive measurements was recorded (Youlten et al. 1980, Hellgren et al. 1997). Mucociliary clearance was assessed as nasal transit time, using the saccharine test (Andersen et al. 1974). Nasal lavage was carried out according to the Baltimore method (Nacleiro et al. 1983). Nasal lavage was performed once. The lavage fluid was measured and then centrifuged. The supernatant was instantly frozen to −20 °C and then stored at −70 °C. It was later analysed for the concentration (ng/l) of interleukin-8 (IL-8) (DuoSeT, Genzyme). Exhaled and nasal nitric oxide (NO) was measured and the data have been presented elsewhere (Olin et al. 1998).

Exposure

Soft paper is used in the production of toilet–paper, paper towels and napkins. The raw material for soft paper is mainly waste paper. The paper is produced in huge paper-machines and is later processed (converted) to the final products by smaller converting machines in other parts of the mill. Short wood-fibres from the recycling process and a rough surface contribute to a high degree of dust emission from the paper during production. Nowadays, the highest levels of dust can be found in the converting departments. The dust exposure in this particular mill has been characterised before (Sahle et al. 1990), Thorén et al. 1989a. In summary, stationary and personal samplings of total dust have been performed since 1971. Since the 1970s the workers in the converting departments have been exposed to total dust levels below 5 mg/m^3.

For this study, personal sampling of inhalable dust was performed with IOM samplers (Mark and Vincent 1986) on each exposed subject. Sampling was done during a whole 8 h work-shift. The clinical investigation was performed during the same work-shift as was the dust sampling. Each filter was weighed before and after the measurement and the increase in weight corresponded to the amount of dust sampled.

Statistical analyses

Odds ratios for the nasal symptoms have been calculated using the Mantel-Haenszel test with stratification for smoking habits (never smoked/ever smoked). The 95% confidence interval (95% CI) has been calculated. Owing to small numbers, we used Fisher's exact test when analysing improvement to symptoms.

For the comparison between the exposed and the unexposed group with regard to continuous variables such as NPIF, acoustic rhinometry, the saccharine test, IL-8 in nasal lavage and pulmonary function, we used Student's t-test and calculated the P values. For the trend test for IL-8 and smoking, the Kruskal-Wallis test was used. For the correlations, we used Spearman's non-parametric test.

Results

Personal inhalable dust exposure ranged from 0.5 mg/m^3 to 10 mg/m^3, mean value 3.9 mg/m^3, median level 3.6 mg/m^3. There was no difference between men and women. Each exposed worker was assigned an estimate of current paper-dust exposure based on the measurements performed.

Among the exposed workers, there was an increased prevalence of nasal blockage (odds ratio (OR) 1.5, 95% CI 1.1–2.0) and nasal crusting (OR 2.2, 95% CI 1.5–3.3) when controlled for smoking habits (Table 2). Recalculation controlling for gender did not change the OR. Among individuals reporting nasal blockage, the symptom impaired after a full workday in 9 of 11 in the exposed group but remained unchanged in all controls ($P < 0.05$). Nasal itching and crusting also increased in the majority of exposed individuals suffering from nasal obstruction but remained unchanged among the controls. Sneezing and sense of smell remained unchanged in both groups after a full workday. In the clinical examination of the nose, reddening of the nasal mucosa, as a sign of nasal inflammation, was found in 46% (17/37) of the exposed subjects and in 31% (11/36) of the controls, $P = 0.18$.

The measurements of pulmonary function, acoustic rhinometry, NPIF, nasal transit time and IL-8 in nasal lavage are presented in Table 3. Analyses showed no positive correlation between nasal airflow, internal nasal

Table 2 Odds ratios for nasal symptoms controlled for smoking habits, according to Mantel-Haenszel, among workers exposed to paper dust ($n = 37$) compared with unexposed controls ($n = 36$)

Symptom	Exposed		Controls		Odds ratio (95% CI)
	Yes	No	Yes	No	
Nasal obstruction	16	21	5	30	1.5 (1.1–2.0)
Nasal discharge	6	31	7	28	1.0 (0.8–1.2)
Nasal itching	9	28	6	29	1.0 (0.9–1.4)
Sneezing attacks	11	26	7	28	1.1 (0.9–1.5)
Impaired sense of smell	8	29	2	33	1.2 (0.99–1.5)
Nasal crusting	23	14	6	30	2.2 (1.5–3.3)
Use of nasal decongestants	6	31	2	33	1.1 (0.95–1.3)
History of wheezing	13	24	3	31	1.4 (1.1–1.8)

geometry (MCA1, Vol 1), nasal transit time, IL-8, exposure time or the assigned current exposure to paper-dust.

In an extended analysis the exposed workers were divided into two groups with regard to current dust levels: at median level or below and above the median level. There were no significant differences between these groups with regard to nasal airflow, internal nasal geometry (MCA1, Vol 1), nasal transit time and IL-8.

In the whole population, IL-8 in nasal lavage was significantly higher among men than among women, 193 ng/l vs 132 ng/l, $P = 0.006$. The gender-difference was also seen among those who had never smoked and those who had ever smoked, but formal significance was obtained only among those who had never-smoked (158 ng/l vs 96 ng/l, $P = 0.001$). There was also a significant positive trend ($P = 0.01$) with increasing nasal IL-8 going from non-smokers (122 ng/l), ex-smokers (126 ng/l) to current smokers (235 ng/l).

Discussion

In this study we have verified our previous observation that workers exposed to paper-dust have an increased prevalence of nasal blockage and nasal crusts. We have also found that the symptoms increase during the workday. However, we find little support for inflammation in the nasal mucosa of the exposed subjects.

In a study with this design there are some methodological aspects that have to be considered. We have studied persons with long employment in the paper mill in order to have subjects with a long exposure to paper-dust. An obvious problem with a long period of employment is selection bias, which would result in a "resistant" population and an underestimation of the effect. Despite this, the study showed that the exposed workers had an increased prevalence of symptoms related to nasal obstruction, e.g. blockage and crusts. The prevalence of impaired sense of smell was almost significantly increased among the exposed subjects.

The prevalence of a history of wheezing was increased among the exposed subjects. Previous studies among soft-paper workers have demonstrated a decreased lung function (Heederik et al. 1987; Ericsson et al. 1988) and an increased mortality because of obstructive lung disease (Thorén et al. 1989b). The observed increased prevalence of wheezing, despite possible selection bias, indicates an increased risk of asthma. However, the study was not designed to study this.

Rhinitis is defined as the presence of one or more symptoms of inflammation in the nasal mucosa, such as blockage, itching, sneezing or nasal secretion (International Rhinitis Management Working Group 1994). We hypothesised that inflammation in the nose would lead to swelling of the nasal mucosa, with reduced internal geometry, increased nasal airway resistance and decreased airflow, reduced mucociliary clearance and an influx of pro-inflammatory cytokines such as IL-8. In the present study, we found no difference in intranasal geometry, monitored by acoustic rhinometry, between the exposed workers and the unexposed controls as would be expected with an inflammatory swelling large enough to cause symptoms of nasal obstruction. It has been demonstrated that nasal peak expiratory flow (NPEF) decreases during a working week among wood-dust exposed vocational teachers in Sweden (Åhman and Söderman 1996). In the present cross-sectional study, the exposed paper-workers did not exhibit any reduction in NPIF compared with the controls, however.

Mucociliary clearance can be impaired by irritants. In our material, there was no impairment of mucociliary clearance among the exposed workers compared with the controls. Paper dust can be assumed to be less reactive, than for instance, lime dust (Torén et al. 1996). It has been reported that water-insoluble starch particles, which more resemble paper dust, can evoke a sensation of dryness in the nose without affecting nasal geometry or mucociliary clearance, when given intranasally (Holmberg et al. 1994).

IL-8 is a pro-inflammatory cytokine with neutrophil-activating capacity that is elevated in nasal secretions in both acute and chronic allergic inflammation (Bachert et al. 1995). In the exposed population, there was no increased level of IL-8 in nasal lavage compared with

Table 3 Results of objective measurements with regard to exposure to paper dust (standard error) (*MCA1* minimum cross-sectional area at 0–3 cm from the nostril, *Vol 1* volume in the same interval, *NPIF* nasal peak inspiratory flow, *FVC* forced vital capacity, FEV_1 one-second forced expiratory volume, *IL-8* interleukin-8)

	Exposed	Controls	Significance level[a]
Acoustic rhinometry			
MCA1 (cm^2)	0.54 (0.03)	0.60 (0.02)	$P = 0.11$
Vol 1 (cm^3)	2.0 (0.05)	2.1 (0.06)	$P = 0.26$
NPIF (l/min)	157 (10.7)	158 (8.7)	$P = 0.99$
Nasal transit time (s)	609 (44.4)	687 (64.0)	$P = 0.32$
FVC (% of expected)	104 (2.2)	98 (1.9)	$P = 0.06$
FEV 1 (% of expected)	103 (2.8)	102 (2.4)	$P = 0.73$
Nasal IL-8 (ng/l)	152 (16.3)	163 (28.2)	$P = 0.73$

[a] Student's *t*-test

controls. We did, however, find a positive correlation between the degree of smoking and IL-8 (non-smoker, ex-smoker, smoker). Cigarette smoke is a well-known and potent airway-irritant that has been demonstrated to cause a dose-dependent rise in IL-8 in the lower airways in bronchoalveolar lavage (BAL) (Kuschner et al. 1996).

We find it unlikely that the increased prevalence of nasal symptoms among the exposed workers is caused by an inflammatory reaction in the nasal mucosa. In the unblinded clinical investigation, there was a tendency for more reddening of the nasal mucosa to be observed among the exposed subjects. However, the objective measurements did not indicate presence of inflammation in the mucous membranes.

It is possible that dust deposition on the mucosa with subsequent mechanical obstruction and crust formation contributed to the increased symptoms in the exposed group. The reason that we cannot see this in our measurements could be that the subject would have restored normal conditions by blowing his or her nose before coming to us. Repeated nose-blowing could also increase nasal symptoms and awareness of the nose, and could explain the subjective deterioration in nasal symptoms over the day.

In conclusion, we have found that occupational exposure to paper dust is associated with symptoms of nasal blockage and nasal crusting. We find no objective signs of nasal inflammation, even among the subgroup with the highest current exposure.

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