J Occup Health 2003; 45: 324–330

Journal of Occupational Health

Field Study

Respiratory Effects of Chronic Animal Feed Dust Exposure

Sevin BASER[1], Fatma Evyapan FISEKCI[1], Sibel OZKURT[1] and Mehmet ZENCIR[2]

[1]Pulmonology Department, Pamukkale University Medical Faculty and [2]Public Health Department, Pamukkale University Medical Faculty, Turkey

Abstract: Respiratory Effects of Chronic Animal Feed Dust Exposure: Sevin BASER, *et al.* Pulmonology Department, Pamukkale University Medical Faculty, Turkey—Aim—The aim of our study was to assess the prevalence of chronic work related respiratory symptoms and to determine lung function abnormalities in animal feed industry workers. Method—108 workers with a mean age of ± SD: 32 ± 7.11 yr employed in the animal feed industry and 108 unexposed subjects as a control group were enrolled in the study. All subjects filled out a questionnaire on their respiratory symptoms. Pulmonary function tests (PFTs) were conducted. Airborne dust (respirable fraction) was sampled during an 8-h work shift. Dust sampling was performed with a Casella AFC 123 machine. Results—A significantly higher prevalence of work related upper and lower respiratory tract symptoms such as cough (12%), dyspnea (5.6%) and sinusitis (8.3%) were found among the workers than in the control group (p=0.001, p=0.04 and p=0.008 respectively). Irritation symptoms such as pruritis of the eyes (11.1%), skin lesions (7.4%) and nose symptoms (8.3%) were also significantly higher among workers that in the control group (p=0.001, p=0.014 and p=0.005 respectively). The mean PFTs (predicted %) of the workers; forced vital capacity (FVC)% ± SD (85.23 ± 12.06), 1-s forced expiratory volume (FEV_1)% ± SD (88.73 ± 13.09), peak expiratory flow (PEF)% ± SD (70.64 ± 18.76) and forced expiratory flow rate at 25–75% of the FVC (FEF_{25-75})% ± SD (88.42 ± 25.94) were found significantly lower than in the control group ($p<0.0001$, $p<0.0001$, $p<0.0001$, $p<0.0001$ respectively). Our data indicate that exposure to animal feed dust is an important factor in the occurrence of respiratory symptoms and decline in lung functions. *(J Occup Health 2003; 45: 324–330)*

Key words: Animal feed dust, Respiratory symptoms, Pulmonary function tests, Organic dust

Received Feb 3, 2003; Accepted Aug 18, 2003
Correspondence to: S. Baser, Pulmonology Department, Pamukkale University Medical Faculty, Ataturk Cad. Tuna Apt. No:16/1 20100, Denizli, Turkey

There is increasing evidence of the deleterious effects of organic dust on respiratory functions in exposed workers[1–5].

Animal feed dust is a complex organic dust composed mainly of grain (corn, wheat, barley, rye, oats), residues of crushed seeds, waste products from the food industry (corn bran, wheat bran), fats, molasses, vitamins and minerals.

Numerous studies[1, 6–10] have demonstrated that grain dust is a biological active dust capable of inducing respiratory tract irritation and inflammation, and increases airways reactivity with temporary or permanent persistent functional changes.

The different syndromes or diseases caused by organic dust include hypersensitivity pneumonitis[11], organic dust toxic syndrome[12, 13], occupational asthma[3] and bronchitis[4, 14]. Less distinctive syndromes include mucous membrane irritation syndrome due to an exaggerated physiologic response and occupational simple chronic bronchitis. The nonspecific upper airway mucous membrane irritation and simple bronchitis are more common than occupational asthma or organic dust toxic syndrome. Hypersensitivity pneumonitis is rare[15].

Reactions of the respiratory system to organic dust may potentially be caused or aggravated by a number of different mechanisms including nonspecific airway irritation, allergic reactions to antigens in dust, inflammatory reactions to various agents widely distributed in organic dust such as endotoxins[1]. Organic dusts are frequently contaminated by endotoxins and endotoxin exposure can be an important factor in the development of respiratory impairment[9, 16–18]. Also organic dust causes changes in the clearance of particles from the lungs leading to deposition[1].

The inflammatory effects of organic aerosols also have been explained by nonspecific or specific release of mediators in the airway or by the presence of histamine or other mediators in these organic dusts which may directly constrict airway smooth muscle and activate cells

and chemotoxins[1].

Some investigators[1] have suggested that bronchial hyperresponsiveness may be due to increased permeability of the airway mucosa to irritants secondary to epithelial damage, resulting in a direct effect on airway smooth muscle contraction. Repeated damage to the airway epithelium may be an important step in the pathogenesis of respiratory impairment.

Respiratory effects of organic dust have been reported from several studies. The main exposures that attracted scientific attention were cotton and grain dust and their constituents[4, 16, 19, 20].

There is limited evidence of respiratory effects due to animal feed dust exposure. In Turkey there are approximately 420 factories and 15,000 workers employed in this industry. Taking this into consideration, we aimed to investigate the effect of exposure to animal feed dust on respiratory symptoms and functions. The factory in which we did the study has the highest production capacity (1,200 tones/day) in the Middle East and Balkans.

Methods

Study subjects

Our study group was composed of animal food processing workers employed in a factory in Denizli, located in western Turkey. All subjects were volunteers who gave informed written consent to their participation in the study. 97 males and 11 females (mean age ± SD: 32 ± 7.11 yr, range:19–55 yr) employed in the animal feed industry who are exposed to animal food dust were studied. Control subjects (n=108) were employed at a hospital and also are of similar age, sex, smoking habit, social and economic status and performing comparable (manual) work but without organic dust exposure.

Respiratory symptoms and questionnaire

All of the subjects completed a modified American Thoracic Society questionnaire which included items regarding following points:

–Episodes of wheezing or chest tightness.
–Symptoms of dyspnea, cough, phlegm.
–Symptoms of sneezing, rhinitis, throat itching.
–Eye symptoms or dermatologic symptoms such as pruritis, erythema.
–Time of onset of symptoms.
–Duration of symptoms.
–Relationship of symptoms to work, i.e. whether they were worse at work or at home or whether they only arose exclusively at work
–If symptoms were relatively persistent or whether there was an improvement while away from work during rest periods or on holiday.
–Whether treatment had been received for the symptoms.
–Health history including allergic status and smoking habit.

The questionnaire was administered in a person to person interview.

Smoking History

A detailed smoking history was obtained from each worker and control subject. All of the subjects were grouped as smokers, non-smokers and ex-smokers. We also grouped the workers into three groups according to their number of package-years: the first group (0–4 packet-yr), second group (5–9 packet-yr) and third group (10–↑ packet-yr).

Pulmonary Function Tests (PFTs)

PFTs were performed with a portable spirometer (MIR Spirobank). 1-s forced expiratory volume (FEV_1), forced vital capacity (FVC), peak expiratory flow (PEF) and forced expiratory flow rate at 25–75% of the FVC ($FEF_{25–75}$) were determined. The predicted normal values used were those of Morris and colleagues[21].

The maneuvers were performed by using the standard protocol of the American Thoracic Society[22]. The spirometer was calibrated prior to each daily use.

The measurements were taken after completing 6 hours of normal work.

Environment dust measurement

Total and respirable dust samples were collected over each 8-h work shift by means of personal samplers worn in the breathing zone of each study participant during his or her work shift in all the work places of the workers examined except offices. Dust sampling was performed by means of a Casella AFC 123 machine. Dust concentrations were determined by gravimetric analysis. Cumulative exposure was calculated for every worker by multiplying the working years by the exposed dust concentration.

Statistics

Statistical analysis was performed with the SPSS statistical package program. The χ^2 test was used to compare the presence of respiratory symptoms in all groups and to investigate the effect of smoking on respiratory symptoms. Student's t-test was used to compare the PFTs and to investigate the effect of smoking on them. Pearson Correlation Analysis was used to determine the relationship between groups. Logistic regression analysis (stepwise-backward) was used to analyse the effect of animal feed exposure on respiratory symptoms. Multiple regression analysis (backward) was used to analyse the effects of animal feed exposure on pulmonary function tests. In multiple factor analysis methods, exposure, smoking, sex and age were included as risk factors. The Mantel-Haenszel method was used

Table 1. The demographic characteristics of workers and control group

	Workers (n=108)	Control group (n=108)	P value
Mean age (± SD)	32 ± 7.11	30 ± 6.06	NS
Mean age of males (± SD)	32.99 ± 7.10	31.01 ± 6.20	NS
Mean age of females (± SD)	26.09 ± 3.36	28.23 ± 4.36	NS
Number of females	11 (11.1%)	13 (12%)	NS
Number of males	97 (89.9%)	95 (88%)	NS
Mean working years (± SD)	5.6 ± 5.5	6.2 ± 5.3	NS
Smoker	56 (51.9%)	50 (46.3%)	NS
Ex-smoker	11 (10.2%)	12 (11.1%)	NS
Non-smoker	41 (38.0%)	46 (42.6%)	NS
Mean package-years (± SD)	3.50 ± 8.25	3.31 ± 8.97	NS

NS (not significant): p>0.05

Table 2. Work related respiratory symptoms and irritation symptoms

Symptom	Workers		Control group		P value
	n=108	%	n=108	%	
Cough	13	12%	0	0%	P=0.001
Phlegm	3	2.8%	0	0%	NS
Wheeze	5	4.6%	0	0%	NS
Sinusitis	9	8.3%	0	0%	p=0.008
Dyspnea	6	5.6%	0	0%	p=0.04
Piruritis of eyes	12	11.1%	0	0%	P=0.001
Skin lesions	8	7.4%	0	0%	p=0.014
Nose symptoms	9	8.3%	0	0%	p=0.005

to adjust the smoking condition while comparing the prevalence of respiratory symptoms in exposed workers and in the control group. If the p value <0.05, the difference was considered statistically significant.

Results

Ninetyseven males and 11 females totalling 108 workers with a mean age ± SD: 32 ± 7.11 yr (range: 19–55 yr) employed in the animal feed industry were enrolled in the study. The workers had been employed for a mean of 5.6 ± 5.5 yr (range: 1–23 yr) in the animal food factory. The demographic characteristics and smoking history of the workers and control group are shown in Table 1.

Eighty-six workers (79.6%) had worked in the industry for less than 10 yr and 22 workers (20.4%) had worked for more than 10 yr.

Work related respiratory symptoms and irritation symptoms

When exposed workers and the control group were adjusted for smoking condition, prevalence of respiratory symptoms related to work, cough in 13 workers (12%), dyspnea in 6 workers (5.6%) and sinusitis in 9 workers (8.3%), were found significantly higher in workers than in the control group (p=0.001, p=0.04 and p=0.008 respectively) (Table 2).

When exposed workers and the control group were adjusted for smoking condition, prevalence of irritation symptoms related to work, pruritis of the eyes in 12 workers (%11.1), skin lesions in 8 workers (%7.4) and nose symptoms in 9 workers (8.3%) were found significantly higher among workers than in the control group (p=0.001, p=0.014 and p=0.005 respectively) (Table 2).

Respiratory symptoms were analyzed by multiple logistic regression analysis including sex, age, smoking and exposure. There was no relationship between respiratory symptoms and variables.

There was no significant difference between the workers who had worked more than 10 yr and those who had worked less than 10 yr in the work related respiratory symptoms.

Cessation of most of the respiratory symptoms during holidays was stated by most of the workers. These data are summarized in Table 3.

Table 3. Respiratory symptoms at work and cessation during holidays

Symptoms	Workers (n=108)				P
	Work related symptoms		Cessation during holidays		
Cough	13	12%	10	9.2%	P<0.0001
Wheeze	5	4.6%	3	2.7%	P<0.0001
Sinusitis	9	8.3%	6	5.6%	P<0.0001
Dyspnea	6	5.6%	5	4.6%	P<0.0001
Piruritis of eyes	12	11.1%	9	8.3%	P<0.0001
Skin lesions	8	7.4%	4	3.7%	P<0.0001
Nose symptoms	9	8.3%	9	8.3%	P<0.0001

Table 4. Respiratory symptoms of positive smoking history and non-smoker workers

Symptoms	Smoker and ex-smoker		Non smoker		P
	Workers (n=67)	%	Workers (n=41)	%	
Cough	9	13.4	4	9.8	NS
Phlegm	3	4.5	0	0	NS
Wheeze	4	6	1	2.4	NS
Dyspnea	4	6	2	4.9	NS
Sinusitis	6	9	3	7.3	NS

NS (not significant): p>0.05

Table 5. Pulmonary Function Tests (PFT) of workers and control group

PFT Predicted %	Workers (n=108) mean ± SD	Control group (n=108) mean ± SD	P value
FEV_1	88.73 ± 13.09	98.76 ± 12.76	p<0.0001
FVC	85.23 ± 12.06	92.11 ± 11.02	p<0.0001
PEF	70.64 ± 18.76	92.34 ± 18.43	p<0.0001
FEF_{25}	73.29 ± 20.00	94.31 ± 21.24	p<0.0001
FEF_{50}	86.16 ± 26.46	98.72 ± 25.89	p=0.001
$FEF_{25–75}$	88.42 ± 25.94	105.14 ± 27.77	p<0.0001

Chronic respiratory symptoms related to work were not significantly different in the organic dust exposed workers who had a smoking history (smokers and ex-smokers) and non-smokers (Table 4).

When 67 positive smoking history workers were categorized into three groups according to their number of package-years; in the first group (0–4 packet-yr) there were10 workers, in the second group (5–9 packet-yr) there were 16 workers and in the third group (10–↑ packet-yr) there were 41 workers. There was no significant difference in symptoms among these 3 groups.

Pulmonary Function Tests

The mean PFTs (predicted %) of the workers; FVC% ± SD (85.23 ± 12.06), FEV_1% ± SD (88.73 ± 13.09), PEF% ± SD (70.64 ± 18.76), FEF_{25}% ± SD (73.29 ± 20.00), FEF_{50}% ± SD (86.16 ± 26.46) and $FEF_{25–75}$% ± SD (88.42 ± 25.94) were found to be significantly lower than those of the control group (p<0.0001, p<0.0001, p<0.0001, p<0.0001, p=0.001, p<0.0001 respectively) (Table 5).

Multiple regression analysis methods showed that exposure was the cause of a decline in PFT values.

There was no correlation between the number of working years and the mean PFT (predicted %) for FVC, FEV_1, PEF, FEF_{25}, and $FEF_{25–75}$ (r=–0.033, r=–0.032, r=0.048, r=0.013 and r=–0.092 respectively).

Multiple regression analysis showed that the predicted

Table 6. PFTs values of workers and control group who had a positive smoking history

PFT	Workers (n=67)	Control group (n=62)	P
Predicted %	mean ± SD	mean ± SD	
FEV_1	89.59 ± 12.78	98.32 ± 14.66	p<0.0001
FVC	86.22 ± 12.13	91.76 ± 12.11	p=0.006
PEF	70.50 ± 18.05	92.47 ± 18.50	p<0.0001
FEF_{25}	74.07 ± 18.65	93.47 ± 20.58	p<0.0001
FEF_{50}	86.28 ± 23.76	97.44 ± 27.57	p=0.008
$FEF_{25–75}$	88.83 ± 24.13	103.27 ± 29.23	p=0.001

Table 7. PFTs values of non-smoker workers and non-smoker control group

PFT	Workers (n=41)	Control group (n=46)	P
Predicted %	mean ± SD	mean ± SD	
FEV_1	87.27 ± 13.63	99.34 ± 9.73	p<0.0001
FVC	83.57 ± 11.89	92.56 ± 9.46	p<0.0001
PEF	70.85 ± 20.12	92.14 ± 18.52	p<0.0001
FEF_{25}	71.97 ± 22.28	95.55 ± 22.26	p<0.0001
FEF_{50}	85.95 ± 30.76	100.44 ± 23.62	p=0.016
$FEF_{25–75}$	87.72 ± 29.02	107.66 ± 25.74	p=0.001

Table 8. The mean PFTs (pred%) of workers who had a smoking history according to the number of packet-yr group

PFT	0–4 pac-yr (n=10)	5–9 pac-yr (n=16)	10–↑ pac-yr (n=41)	P
Predicted %	mean ± SD	mean ± SD	mean ± SD	
FEV_1	90.90 ± 9.87	85.81 ± 13.11	90.75 ± 13.25	NS
FVC	87.50 ± 13.27	82.31 ± 15.05	87.43 ± 10.50	NS
PEF	75.20 ± 21.47	70.06 ± 19.11	69.53 ± 17.03	NS
FEF_{25}	78.90 ± 18.70	73.06 ± 20.92	73.29 ± 18.00	NS
FEF_{50}	84.30 ± 10.70	86.43 ± 25.16	86.70 ± 25.83	NS
$FEF_{25–75}$	88.10 ± 12.63	87.62 ± 19.62	89.48 ± 27.91	NS

NS (not significant): p>0.05

% FEV_1, FVC, PEF and FEF_{25} were significantly lower in male workers independent of age and smoking condition.

Dust measurements

8-h personal inspirable dust samples were taken from the workers and gravimetric dust concentrations were determined. Respirable dust concentrations varied from 0.39154 mg/m^3 to 2.80053 mg/m^3. These results were below the standards. We didn't find a relationship between dust concentrations, exposure dose (time of working in feed dust exposure × concentration of the feed dust) and respiratory symptoms, PFTs.

Effect of smoking on PFTs

The mean PFT predicted % values of the 67 workers who had a smoking history (smokers and ex-smokers) were found to be significantly lower than those of the 62 control subjects who had a smoking history (smokers and ex-smokers) (Table 6).

The mean PFT predicted % values of the 41 non-smoker workers were found significantly lower than the mean values of 46 non-smoker control subjects (Table 7).

When 67 positive smoking history workers were categorized into three groups according to their number of package-years; in the first group (0–4 packet-yr) there were10 workers, in the second group (5–9 packet-yr) there were 16 workers and in the third group (10–↑ packet-yr)

there were 41 workers. There was no significant difference in PFT values among these 3 groups (Table 8).

The decline in lung functions was due to organic dust exposure independent of the smoking effect.

Discussion

Our results suggest that exposure to animal food dust is responsible for the development of chronic respiratory symptoms and lung function abnormalities. In our study we found a significantly higher prevalence of respiratory symptoms related to work such as cough in 13 workers (12%), dyspnea in 6 workers (5.6%) and sinusitis in 9 workers (8.3%). Zuskin *et al.*[1] studied a group of 71 male workers employed in animal feed processing and found chronic respiratory symptoms such as cough in 40 workers (56%), dyspnea in 8 workers (11.3%), sinusitis in 15 workers (21.1%) and phlegm in 36 workers (50.7%). In our study the workers had been employed a mean of 5.6 yr and their mean age was 32 yr. In the Zuskin *et al.*[1] study the mean age of workers was 40 and they had been employed for 15 yr. When compared with the Zuskin *et al.*[1] study, the low prevalence of work related respiratory symptoms in our study might be due to the difference in the total exposure time. Zuskin[1] concluded that this high prevalence of cough might be due to damage to the airway mucosa.

Most of our workers had these symptoms exclusively during exposure to the animal food dust or their existing symptoms were exacerbated at work. This fact supports a cause-effect relationship between respiratory symptoms and dust exposure.

Smid *et al.*[17] explored the relationships between exposure to organic dust and respiratory symptoms and chronic lung function changes in 315 animal feed workers. They found that the prevalence of respiratory symptoms ranged from 4% (chest tightness) to 16% (wheezing).

In our study, 86 workers (79.6%) had been working for less than 10 years and 22 workers (20.4%) had been working for more than 10 years. We found no significant difference between these two groups in the work related respiratory symptoms. Smid *et al.*[17] found that the prevalence of most chronic respiratory symptoms tended to decrease with increasing years of exposure. Similarly to Smid *et al.*[17], we called this finding a "healthy worker effect".

In field studies a self-administered questionnaire may have low sensitivity when compared with the usual oral interviews; to determine the correct results we administered the questionnaire as a person to person interview.

Smid *et al.*[17] showed the relationship of respiratory symptoms to smoking, and showed that the number of pack-years was related to symptoms. In our study, chronic work related respiratory symptoms were not significantly different in workers who had a smoking history (smokers and ex-smokers) from those in non-smokers. When workers and the control group were adjusted for smoking condition we found a significantly higher prevalence of respiratory symptoms among workers. Also in our study there was no significant difference between the symptoms of workers when they were grouped according to their number of package-years. We think that organic dust is the cause of chronic respiratory symptoms independent of the smoking effect.

Pulmonary function testing is an essential means for diagnosing airways disease. Changes in lung function have been reported in farmers[23], textile workers[4], cocoa workers[24], fur workers[25] and spice factory workers[26].

In our study, however, the chronic effect of animal feed dust was investigated so we applied the PFTs only once to the workers. When compared with the control group of similar age, sex, smoking habits, social and economic status, we found out that the mean PFT (predicted %) values of the workers were significantly lower than in the control group. Post *et al.*[2] followed up 140 animal feed processing workers for five years and showed that the annual decline in FEV_1 and maximal mid-expiratory flow were significantly related to occupational exposure. Similar to these findings Smid *et al.*[17] showed lung function decline especially in FEV_1 with the increase in production years. In our study there was no correlation between the number of working years and the mean PFTs. The short exposure time (mean; 5.6 yr) in our study group and the greater number of working years (mean; 13 yr) in the Smid *et al.*[17] study might be the reason for this. Following up the workers and determining the annual decline in PFTs will be appropriate.

Zuskin *et al.*[27] studied 71 animal feed workers and found significantly lowered measured values for FVC, FEV_1 and FEF_{50} in both smokers and non-smokers. They suggested that smoking appears to aggravate these changes. Smid *et al.*[17] showed the number of packet-yrs was related to lung functions. In our study there was no significant difference between the PFT values for workers when they were grouped according to their number of package-years (Table 8). The distinct findings might be due to the mean packet-yrs which is 3.5 in our study and 11.1 in the Smid *et al.*[17] study.

Our results showed that PFTs of male workers were affected negatively by organic dust irrespective of age and smoking condition. This result might be due to the lower percentage of female workers in the study group rather than male susceptibility to organic dust.

In our study we found that the mean PFT predicted % values of the workers who had a positive smoking history was significantly lower than in the control group with the same smoking history. We also determined that the mean PFTs predicted % values of the 41 non-smoker workers was significantly lower than the 46 non-smoker control subjects. With these results we conclude that

organic dust exposure might be the cause of a decline in lung functions, independent of the smoking effect.

Conclusion

Our data indicate that exposure to animal feed dust is an important factor in the development of chronic respiratory symptoms and decline in lung functions. Exposure to animal feed in an occupational setting can effect the respiratory health of workers. This effect occurs independent of the smoking status.

References

1) E Zuskin, EN Schachter, B Kanceljak, TJ Witek and E Fein: Organic dust disease of airways. Int Arch Occup Environ Health 65, 135–140 (1993)
2) W Post, D Heederik and R Houba: Decline in lung function related to exposure and selection processes among workers in the grain processing and animal feed industry. Occup Environ Med 55, 349–355 (1998)
3) JR Balmes: Occupational respiratory diseases. Occup Environ Med 27, 1009–1038 (2000)
4) R Niven, AM Fletcher, CAC Pickering, D Fishwick, CJ Warburton, JC Simpson, H Francis and LA Oldham: Chronic bronchitis in textile workers. Thorax 52, 22–27 (1997)
5) D Fishwick, A Fletcher, C Pickering, R Niven and B Faragher: Respiratory symptoms and dust exposure in Lancashire cotton and man-made fiber mill operatives. Am J Respir Crit Care Med 150, 441–447 (1994)
6) PJ Jagielo, PS Thorne, JL Watt, KL Frees, TJ Quinn and DA Schwartz: Grain dust and endotoxin inhalation challenges produce similar inflammatory responses in normal subjects. Chest 110, 263–270 (1996)
7) DA Schwartz: Grain dust, endotoxin, and airflow obstruction. Chest 109, 57–63 (1996)
8) JF Trapp, JL Watt, KL Frees, TJ Quinn, MW Nonnenman and DA Schwartz: The effect of glucocorticoids on grain dust induced airway disease. Chest 113, 505–513 (1998)
9) S Becker, WA Clapp, J Quay, KL Frees, HS Koren and DA Schwartz: Compartmentalization of the inflammatory response to inhaled grain dust. Am J Respir Crit Care Med 160, 1309–1318 (1999)
10) PJA Borm, RP Schins, TJ Derhaag, IJ Kant and TH Jorna: Cross-Shift changes in blood inflammatory markers occur in the absence of airway obstruction in workers exposed to grain dust. Chest 109, 1078–1085 (1996)
11) JN Fink: Hypersensitivity Pneumonitis. Clin Chest Med 13, 303–309 (1992)
12) SF Wintermeyer, WG Kuschner, H Wong, A D'Alessandro and PD Blanc: Pulmonary responses after wood chip mulch exposure. J Occup Environ Med 39, 308–314 (1997)
13) P Malmberg, A Rask-Andersen and L Rosenhall: Exposure to microorganisms associated with allergic alveolitis and febrile reaction to mold dust in farmers. Chest 103, 1202–1209 (1993)
14) EO Terho, K Husman and I Vohlonen: Prevalence and Incidence of Chronic Bronchitis and Farmer's Lung with Respect to Age, Sex, Atopy, and Smoking. Eur J Respir Dis Suppl 152, 19–28 (1987)
15) GA Pico: Hazardous exposure and lung disease among farm workers. Clin Chest Med 13, 311–328 (1992)
16) RM Castellan, SA Olenchock, KB Kinsley and JL Hankinson: Inhaled endotoxin and decreased spirometric values ; An exposure-response relation for cotton dust. N Engl J Med 317, 605–610 (1987)
17) T Smid, D Heederik, H Remko and HQ Philip: Dust and Endotoxin-related Respiratory Effects in the Animal Feed Industry. Am Rev Respir Dis 146, 1474–1479 (1992)
18) R Rylander, B Bake, J Fischer and IM Helander: Pulmonary function and symptoms after inhalation of endotoxin. Am Rev Respir Dis 140, 981–986 (1989)
19) R McL Niven and CA Pickering: Byssinosis: a review. Thorax 51, 632–637 (1996)
20) R Altin, S Ozkurt, F Fisekci, AH Cimrin, M Zencir and C Sevinc: Prevalence of byssinosis and respiratory symptoms among cotton mill workers. Respiration 69, 52–56 (2002)
21) JF Morris, A Koski and LC Johnson : Spirometric standards for healthy non smoking adults. Am Rev Respir Dis 103, 57–67 (1971)
22) Official Statement of the American Thoracic Society: Standardization of Spirometry. 1994 Update. Am J Respir Crit Care Med 152, 1107–1136 (1995)
23) M Rautalahti, EO Terho, I Vohlonen, J Nuutinen, K Husman and O Korhonen: Effect of indoor feeding season for cattle on lung function of dairy farmers. Eur J Respir Dis Suppl 152, 188–196 (1987)
24) E Zuskin, B Kanceljak, EN Schachter, J Godnic-Cvar, J Mustajbegovic and A Budak: Respiratory function and immunological status in cocoa and flour processing workers. Am J Ind Med 33, 24–32 (1998)
25) E Zuskin, Z Skuric, B Kanceljak, D Pokrajac, EN Schachter and TJ Wittek: Respiratory symptoms and lung function in furriers. Am J Ind Med 14, 189–196 (1988)
26) E Zuskin, Z Skuric, B Kanceljak, D Pokrajac, EN Schachter and TJ Witek: Respiratory findings in spice factory workers. Arch Environ Health 43, 335–339 (1988)
27) E Zuskin and EN Schachter: Respiratory Function in Workers Employed in Animal Food Processing. Am J Ind Med 17, 70–75 (1990)