



level in the ambient environment, where they are produced and inhaled in much higher amount by the workers of these industries. Recently there has been concern about the potential health hazard from occupational exposure to synthetic polymers inhaled as micro fibers or 'flocks'. Several cases of particular occupational interstitial lung diseases termed as 'flock worker lung' have been reported among workers exposed to nylon flock in USA and Canada (8-10). Occupational asthma due to heated polypropylene has also been reported (11).

In India, consumption of plastics has increased many fold, from 4000 tones per annum (1990) to 5 million tones per annum (2005). With increase of plastic consumption, its waste is also increasing. Waste plastics are recycled under very unhygienic conditions without knowing the health hazards and safety parameters required during such kind of activities. During recycling process the agglomerates are compressed and melted at a high temperature. This causes liberation of fumes in the environment which are inhaled by the workers (12). To the best of our knowledge the effects of exposure is not been studied in the workers of plastic factory where recycling of plastic material was done. India being a country where plastic is freely recycled this could be an important occupational hazard.

Hence the present study is planned to study the pulmonary functions of workers in the plastic factory where recycling of plastic material was done. These workers were constantly exposed to fumes throughout the day.

## METHODS

Workers of plastic factory between the ages of 18-40 years were assessed for their pulmonary functions. Thirty one male plastic factory workers formed the study group. The pulmonary function parameters were compared with that of 31 males of same socio-economic status and age not exposed to similar environment who served as controls. Subjects having acute or chronic respiratory disorder, systemic illness or on medication which directly or indirectly affects the respiratory system were excluded from the study. Also the subjects working for at least one year were selected for the study. Ethical committee clearance was taken and an informed written consent was taken from the subject after explaining the procedure.

The factory selected for the present study was a recycling plastic factory located in Bawana, Delhi. Initially plastic waste collected from scrap dealers was cleaned and than separated into different category on the basis of the material of plastic. The segregated plastic was than recycled through the machine. The agglomerates were fed into hopper which than passes to feed zone than to compression zone and finally into metering zone or melting zone. Temperature in the machine varies from 175°C to 240°C. The melted material which comes out is cooled down and processed further. During this whole process fumes are produced which are liberated in the surrounding environment.

Anthropometric measurements including age, height and weight were recorded. Height was measured by stadiometer to

nearest 1 cm with subject standing without shoes and weight by weighing scale to the nearest 1 kg. Further a preliminary medical examination was carried out to rule out any medical complication. The pulmonary function test was carried out using Sibelmed Datospir 120 B portable spirometer with inbuilt computer programme, using the standard laboratory methods. The spirometer was calibrated regularly with a three litre syringe. The questionnaire was filled up and the relevant data (name, age, sex, height, weight, occupation, smoker/non-smoker) was entered into the computer.

The test module was now activated and the subject was given proper instruction about the procedure to be performed. All pulmonary function tests were done on the subjects comfortably seated in an upright position. The subject was asked to breath through the mouthpiece for sometime before the test, in order to familiarize him with the equipment. During the test the subject was adequately encouraged to perform at their optimum level and also a nose clip was applied during the entire maneuver. The flow volume curve was recorded. The procedure for performing the maneuver was demonstrated to the subject. After a few practice breathing, three reproducible lung function values (defined as FVC within 10% of the maximal FVC) were produced. The subjects were encouraged to perform these tests to the best of their ability. Graphs which showed hesitancy or cough were not used in the analyses. Of the acceptable recordings for each subject, the one which showed the largest FVC was used to extract the data. All lung volumes, capacities and flow rates were measured.

#### Statistical analysis

The outcome of pulmonary function test was presented as mean $\pm$ SD for each of the parameters. All the statistical analysis was carried out using SPSS 17.0 statistical package. The two groups were compared by unpaired 't' test and P values of less than 0.05 were considered as significant. Odd's ratio was obtained using logistic regression by adjusting for smoking status. The subjects were divided into three groups on the basis of duration of polypropylene exposure. One way ANOVA was done to compare the parameters in them followed by Tukey test at 5% level of significance.

## RESULTS

The anthropometric measurements of the two groups are shown in Table I. The two groups did not differ significantly on these parameters. Most of the expiratory flow rates (PEF, MEF 50%, MEF 75%, FEF 25-75%) as well as the lung volumes (FVC, FEV1, VC, TV, ERV, MVV) were significantly decreased in the plastic factory workers (Table II). All the parameters when reanalyzed after adjusting for smoking status showed similar results (Table II). The subjects were divided into three groups on the basis of duration of

TABLE I: Anthropometric measurements of controls and study group.

<i>Parameter</i>	<i>Cases (n=31)</i>	<i>Control (n=31)</i>
Age(year)	29 $\pm$ 6.28	30 $\pm$ 7.56
Height(cm)	154 $\pm$ 10.23	152 $\pm$ 8.91
Weight(kg)	58 $\pm$ 3.22	55 $\pm$ 6.65
BMI (kg/m <sup>2</sup> )	22.24 $\pm$ 3.21	23.15 $\pm$ 2.34

Data presented are mean $\pm$ SD.

TABLE II: Comparison of lung function parameters between cases and control.

Lung variables ↓	Control (n=31) Mean±SD	Cases (n=31) Mean±SD	Mean difference	P value un-adjusted <sup>o</sup>	P value adjusted <sup>@</sup>	95% CI for difference		OR
						Lower	Upper	
FVC(l)	3.52±0.63	2.50±0.57	1.01516	<0.001*	<0.001*	0.7086	1.3216	0.061
FEV1(l)	3.17±0.43	2.19±0.67	0.9748	<0.001*	<0.001*	0.6859	1.2638	0.044
FEV1/FVC(%)	92.66±7.53	91.93±8.13	0.7317	0.721	0.72	-3.354	4.818	0.99
PEF(l)	6.74±1.327	4.19±2.04	2.55129	<0.001*	<0.001*	1.6720	3.4305	0.412
MEF 25%(l/s)	2.41±0.86	2.02±0.81	0.39613	0.069	0.295	-0.031	0.8234	0.698
MEF 50%(l/s)	4.17±0.81	3.10±.44	1.0729	<0.001*	0.012*	0.4746	1.6711	0.507
MEF 75%(l/s)	6.04±1.341	4.06±2.15	1.98065	<0.001*	0.004*	1.064	2.8972	0.581
FEF 25-75%(l/s)	3.84±0.86	3.20±1.12	0.63487	0.018*	0.035*	0.1132	1.1564	0.560
FIVC(l)	1.66±1.19	2.15±0.68	-0.48658	0.085	0.095	-1.043	0.0704	1.784
PIF(L/s)	1.98±1.57	2.63±1.04	-0.64863	0.095	0.136	-1.413	0.116	1.468
VC(l)	3.87±0.65	3.03±0.69	0.8429	<0.001*	0.001*	0.4987	1.1870	0.184
TV(l)	0.77±0.25	0.52±0.18	0.24774	<0.001*	0.002*	0.1368	0.3586	0.008
ERV(l)	1.47±0.48	0.94±0.65	0.53548	<0.001*	0.005*	0.2413	0.8296	0.206
IRV(l)	1.58±0.6158	1.47±0.57	0.10258	0.501	0.298	-0.200	0.4056	0.603
IC(l)	2.35±0.45	2.09±0.65	0.25871	0.076	0.083	-0.027	0.5449	0.4
MVV(l)	118.01±27.03	79.74±30.98	38.26	<0.001*	0.001*	23.19	53.33	0.957

<sup>o</sup>unpaired t test;  
<sup>@</sup>adjusted for smoking status by Logistic regression;  
\*significant P<0.05  
OR - Odd's ratio; Data presented are mean±SD.

TABLE III: Comparison of lung function parameters on the basis of duration of exposure.

Lung variables	Controls (unexposed) (n=31) {A}	Cases (Exposure < 5 yr) (n=18) {B}	Cases (Exposure > 5 yr) (n=13) {C}
FVC(l)	3.52±0.63†	2.5267±0.56786	2.4754±0.6062
FEV1(l)	3.17±0.43†	2.284±0.7507	2.072±0.552
FEV1/FVC(%)	92.66±7.53	94.555±5.8861	88.212±9.6073
PEF(l/s)	6.74±1.32†	4.4594±2.2689	3.8192±1.70787
MEF 25%(l/s)	2.41±0.86	2.1922±0.84755	1.7815±0.73746
MEF 50%(l/s)	4.17±0.18	3.4494±1.56885	2.6246±1.12868
MEF 75%(l/s)	6.04±1.34†	4.5078±2.35894	3.4554±1.7514
FEF 25-75%(l/s)	3.84±0.86†	3.5931±1.13572	2.6983±0.93417*
FIVC(l)	1.66±1.19	2.0914±0.78	2.2291±0.56587
PIF	1.98±1.57	2.8314±1.05214	2.38±1.01607
VC(l)	3.87±0.65†	2.9183±0.57588	3.1923±0.8341
TV(l)	0.77±0.25†	0.5511±0.19599	0.4877±0.15632
ERV(l)	1.47±0.48†	0.9094±0.53558	0.9831±0.8215
IRV(l)	1.58±0.61	1.4111±0.48987	1.5708±0.68927
IC(l)	2.35±0.45	2.0178±0.66151	2.2085±0.64797
MVV(l)	118.01±27.03†	83.0256±31.0758	75.1992±31.5264

†Significant difference between A & B, A & C, no difference between B & C.  
\*Significant difference between B and C; Data presented are mean±SD.

exposure as unexposed(A), less than 5 yrs of exposure (B) and more than 5 yrs of exposure (C). Significant difference between A & B,

A & C, and no difference between B & C was observed for most of the parameters (Table III). Only FEF 25-75% was significantly

different between B & C. The comparison of smokers and non smokers is shown in Table IV. There is no significant difference between the two groups.

TABLE IV: Comparison among cases between smokers and non-smokers.

<i>Lung variables</i>	<i>Non smoker (n=16)</i>	<i>Smoker (n=15)</i>
FVC(l)	2.74±0.54	2.25±0.508
FEV1(l)	2.43±0.684	1.94±0.58
FEV1/FVC(%)	91.06±7.36	87.61±16.58
PEF(l/s)	4.83±2.18	3.50±1.703
MEF 25%(l/s)	2.19±0.84	1.83±0.76
MEF 50%(l/s)	3.43±1.34	2.75±1.502
MEF 75%(l/s)	4.82±2.22	3.25±1.82
FEF 25-75%(l/s)	3.29±1.25	2.61±1.32
FIVC(l)	2.38±0.804	1.97±0.53
PIF	2.89±1.23	2.43±0.85
VC(l)	3.16±0.78	2.89±0.58
TV(l)	0.490±0.161	0.56±0.197
ERV(l)	1.056±0.59	0.81±0.724
IRV(l)	1.478±0.64	1.47±0.576
IC(l)	2.11±0.714	2.08±0.602
MVV(l)	82.85±25.87	76.42±36.29

Data presented are mean±SD.

## DISCUSSION

Results of this study are indicating that the workers of plastic factory are prone to respiratory dysfunction. Most of the flow rates, lung volumes and capacities were decreased in workers as compared to controls. Taking smoking and duration of exposure into consideration, it was observed that even non smokers as well as workers working for less than five years had deranged lung functions. The ventilating and air conditioning system were inadequate and the ambient air was polluted with high but undocumented levels of fumes of organic compounds. Lung volumes and capacities were so much reduced that they suggested high level of difficulty and inability in

expansion of thoracic cavity. FEF 25-75% is suggestive of early airflow limitation particularly in smaller airways.

The results of this study are in harmony with previous studies (10–14). Several cases of overt occupational diseases including those related to chronic exposure to plastic pollutants like “flock worker’s lung” have been reported in recent years (8, 9). Also in a similar study done by Eschonbacher WL et al. on nylon flock associated interstitial lung disorder, a high frequency of respiratory and systemic symptoms were reported in nylon flock workers (9) with the symptom prevalence being related to the number of days worked per week. In addition polypropylene flocking workers also reported improvement when away from work at higher rate than control workers. Consistent with these observations, the present study found a significantly higher rate of restrictive impairment with some obstructive impairment also. The diffusion capacity could not be determined in the present study, though we presume that the diffusion must be impaired in these workers as observed by others (10). These subjects worked at least 10 hours per day and 6 days per week, without any respiratory protective measures.

In an experimental study assessing pulmonary toxicity of inhaled polypropylene fibers in rats, Hesterberg et al. found that the severity of pulmonary injury appeared to increase with the dose and duration of exposure (15). However in the present study it was found that exposure upto 1-10 years has not much significant difference, except for FEF 25-75%. The reason of discordance between lung function derangement and duration of exposure may be that maximum

damage to lungs has been done during early period of exposure. More over young workers exposed to less duration might have been assigned heavy load of work as compared to senior ones. Similarly Wong & Musselman (1993) reported significantly increased mortality due to lung cancers among slag wool workers employed for less than 5 years but not among those employed for more than 5 years (16). Similar results (no association between duration of exposure and lung functions) have been observed by others (17, 18).

Taking smoking into consideration, it was observed that even non smokers had deranged lung functions. Smoking is an important independent risk factor deteriorating the lung functions but in these workers it seems that influence of contaminated air is much more. All our subjects were smoking for at least one year. Out of these only 4 subjects were heavy smokers (more than 20 cigarettes/40 bidis per day). Rest all were light smokers hence they were not analyzed separately and grouped together as smokers.

The mechanism by which these disorders were caused and the action of these particular dust occurs in yet unknown but

probable mechanism may be adherence of these micro fibrils with the surface of alveoli (decreasing the diffusing area) and walls of the air pathways resulting in slight narrowing. A significant infiltration of neutrophils, macrophages, mast cells during pathogenesis of interstitial pulmonary disorder and the increased release of IL-8 and TNF- $\alpha$  has been observed (10). The increased release of IL8 and TNF- $\alpha$  are indicative of ongoing pro-inflammatory process in these workers.

The present study stresses on the fact that short duration of exposure as low as one year is sufficient to cause the damage but progress of disease is slow. Moreover the changes in pulmonary functions are also observed in non smokers confirming the fact that the changes are due to the pollutants. The study can be extended to evaluate the status of respiratory membrane, by determining lung diffusion capacities in these workers.

#### Conclusion

Exposure to air pollutants in the work environment is associated with pulmonary function impairment. Appropriate medical surveillance and exposure control should be provided in such factories.

#### REFERENCES

1. Nakai S, Maeda K, Crest JST. Respiratory health associated with exposure to automobile exhaust III. Results of a cross sectional study in 1987, and repeated pulmonary function tests from 1987 to 1990. *Arch Environ Health* 1999; 54: 26-32.
2. Chabra SK, Chabra P, Rajpal S, Gupta RK. Ambient air pollution and chronic respiratory morbidity in Delhi. *Arch Environ Health* 2001; 56: 58-63.
3. Ware JH, Spengler JD, Neas LM, Samet JM, Wagner GR, Coultas D et al. Respiratory and irritant health effects of ambient volatile organic compounds. The Kanawa county health study. *Am J Epidemiol* 1993; 137: 1287-1301.
4. Singhal M, Khaliq F, Singhal S, Tandon OP. Pulmonary functions in petrol pump workers: a preliminary study. *Indian j Physiol Pharmacol* 2007; 51: 244-248.

5. Kilburn KH. Effects of diesel exhaust on neurobehavioral and pulmonary functions. *Arch Environ Health* 2000; 55: 11–17.
6. Bergdahl IA, Toren K, Eriksson K, Hedhind U, Nilsson T, Flodin R, Jarvholm B. Increased mortality in COPD among construction workers exposed to inorganic dust. *Eur Respir J* 2004; 23: 402–406.
7. Eckardt. Occupational and environmental health hazards in the plastic industry. *Environ. Health Perspect.* 1976; 17: 103–106.
8. Kern DG, Kuhn C 3<sup>rd</sup>, Ely EW, et al. Flock worker's lung: broadening the spectrum of clinicopathology, narrowing the spectrum of the suspected etiologies. *Chest* 2000; 117: 251–259.
9. Eschenbacher WL, Kriess K, Loughheed MD, Pransky GS, Day B, Castellan RM. Nylon flock associated interstitial lung disease. *Am J Respir Crit Care Med* 1999; 159: 2003–2008.
10. Atis S, Tutluoglu B, Levent E, Ozturk C, Tunaci A, Sahin Ket al. The respiratory effects of occupational polypropylene flock exposure. *Eur Respir J* 2005 Jan; 25(1): 110–117.
11. Malo JL, Cartier A, Pineault L, Dugas M, Desjardins A. Occupational asthma due to heated polypropylene. *Eur Respir J* 1994; 7: 415–417.
12. Central control Pollution Board. Plastic waste management issues & options. Delhi: Ministry of Environment & Forests; 2007; 4–9.
13. Soutar CA, Copland LH, Thornley PE, Hurley JF, Ottery J, Adams WG, Bennett B. Epidemiological study of respiratory disease in workers exposed to polyvinylchloride dust. *Thorax* 1980; 35: 644–652.
14. Burkhardt J, Piacetelli C, Schwegler-Berry D, Jones W. Environmental study of nylon flocking process *J Toxicol Environ Health A* 1999; 57: 1–23.
15. Hesterberg TW, McConell EE, Miller WC, Hamilton R, Burn WB. Pulmonary toxicity of inhaled polypropylene fibres in rats. *Fundam Appl Toxicol* 1992; 19: 358.
16. Wong O, Musselman RP. Carcinogenicity of insulation wools: further comments and some new data. *Regul Toxicol Pharmacol* 1993; 18(2): 202–205.
17. Gubéran E, Usel M. Unusual mortality pattern among short term workers in the perfumery industry in Geneva. *Br J Ind Med* 1987; 44(9): 595–601.
18. McDonald AD, Fry JS, Woolley AJ, McDonald JC. Dust exposure and mortality in an American chrysotile asbestos friction products plant. *Br J Ind Med* 1984; 41(2): 151–157.