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PULMONARY FUNCTIONS IN WORKERS OF FERTILISER AND CHEMICAL INDUSTRY

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Abstract : The present study was undertaken to assess the pulmonary functions in fertiliser and chemical industry workers of Kerala. Pulmonary function test was performed with Vitalograph Compact-II spirometer. Exposure to different chemicals, noxious gases and particulate matter of fertiliser and chemical industry was injurious to the normal pulmonary function. Significant decline in lung volumes, viz. FVC, FEV_1, FEV_1/FVC% ($P<0.01$) and flow rates, viz. $FEF_{25-75\%}$, $FEF_{0.2-1.2}$, $FEF_{25\%}$, $FEF_{50\%}$, $FEF_{75\%}$ ($P<0.01$) among non-smokers and smokers than that of normal controls was observed. The findings of this study also suggest that with increased duration of exposure to industrial pollutants, lung volumes and flow rates were significantly reduced in workers.

Key words : pollution flow rate pulmonary function fertiliser and chemical industry lung volume

INTRODUCTION

Industrial workers are generally at risk due to constant exposure to different types of pollutants emitted from the industries. Inhalation of these substances can cause damage to the membrane structure and mechanical efficiency. This will lead to alteration in the functional properties of the lungs, resulting various respiratory diseases. Duration of exposure, ageing and smoking can together complicate pulmonary functions. Pulmonary function tests give a clear idea about the condition of pulmonary dysfunction.

The different plants in fertiliser and chemical industry include ammonia plant, urea plant, sulphuric acid plant, phosphoric acid plant, NPK plant etc. where various chemical processes involving high pressure and temperature are needed. Various raw materials and chemicals of hazardous nature are needed for the production of chemicals including fertilisers, viz. ammonia, urea, phosphoric acid, sulphuric acid, ammonium sulphate, NPK, gypsum by-products, ammonium chloride, super phosphate, ammonium phosphate etc. The presence of sulphur dust, gaseous effluents, chemicals and other air-borne contaminants in the

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factory is absorbed high in the airway, increasing the airway resistance thereby adversely affecting the pulmonary functions of workers. Hence this study was undertaken with a view to analyse the respiratory status of fertiliser and chemical industry workers of Kerala.

METHODS

A total of 175 male workers from Fertilisers and Chemicals Travancore Ltd. (FACT), Udyogamandal Division, Ernakulam District, Kerala were selected for the study. In each case, age of the subject, smoking habits, type of work, duration of exposure, type of irritants, physical status and health conditions were recorded through questionnaire. Subjects with less exposure period were excluded from the study. The clinical data were collected from workers and respiratory function test were carried out in the health centres of respective factory in the presence of medical officers. Same procedure was carried out for controls also. Sixty-one age group matched non-smoking normal male controls were selected from non-industrial area who were supposed to have never exposed to any industrial pollutants.

Pulmonary functions were measured with a portable PC based spirometer with printer (Vitalograph Compact-II, Buckingham). According to ATS recommendations (1987), spirometry was performed during morning hours in a climate-controlled room in a clean area of the industry. Spirometer was calibrated with twice daily by pumping 5 litre of air through the instrument with 1 litre precision syringe. After calibration of the machine, the procedure of pulmonary tests was carefully explained to each group of subjects who were then allowed to make trial manoeuvres. Before the test, the subjects were given full physical and mental rest. They were made 3-5 efforts while standing and wearing a nose clip. The best of the multiple trials was recorded automatically by the spirometer at BTPS (body temperature, ambient pressure and saturated with water vapour).

Parameters studied

Lung volumes : Forced vital capacity (FVC), Forced expiratory volume at 1 second (FEV_1), ratios between forced expiratory volume in 1 second to forced vital capacity (FEV_1/FVC%).

Flow rates : Forced mid expiratory flow rate ($FEF_{25-75\%}$), Mean forced expiratory flow rate at 0.2 to 1.2 litres ($FEF_{0.2-1.2}$), forced expiratory flow rate at 25%, 50% and 75% of FVC ($FEF_{25\%}$, $FEF_{50\%}$, $FEF_{75\%}$).

Data analysis : Statistical analysis of results were carried out using unpaired 't'-test. (i) To test the significance of lung volumes and flow rates between normal controls vs. smokers, normal controls vs. non-smokers and smokers vs. non-smokers. (ii) To explore the relationship between exposure duration and respiratory parameters, coefficient of correlation was carried out and it was tested for significance.

RESULTS

The mean duration of exposure was 21.06 years. Among 175 workers 39.4%

(n = 69) were non-smokers, 60.6% (n = 106) were smokers.

Smokers and non-smokers showed significant decrease in the values of FVC and FEV_1 ($P<0.01$) from that of normal controls in all age groups. The FEV_1/FVC% was also showed decrease in two age groups, i.e. G_2 and G_3 in case of smokers and non-smokers ($P<0.01$) than normal controls but the decrease was insignificant between G_1 normal controls and smokers. Highly significant decline in flow rates, viz. $FEF_{25-75\%}$, $FEF_{0.2-1.2}$, $FEF_{25\%}$, $FEF_{50\%}$ and $FEF_{75\%}$ ($P<0.01$) was observed in all age groups of smokers and non-smokers than that of normal controls. FVC of smokers had significant decrease in G_1 ($P<0.01$) and FEV_1/FVC% in G_2 ($P<0.01$) from non-smokers. But the ratio was higher in G_1 smokers than non-smokers ($P<0.01$). However smokers and non-smokers of FACT did not exhibit any significant difference in flow rates (Table I).

TABLE I: Pulmonary functions of normal control (C), non-smoker (NSW) and smoker (SW) workers under different age groups. (Mean ± SD and 'p' values).

Parameters (Units)		*Control (C)*	*FACT workers*		*P<*		
			NSW	*SW*	*C Vs. NSW*	*C Vs. SW*	*NSW Vs. SW*
FVC (L)	G_1	3.40±0.39 (23)	2.17±0.74 (29)	2.16±0.63 (29)	0.01	0.01	0.01
	G_2	3.88±0.68 (21)	2.36±0.57 (10)	2.24±0.60 (9)	0.01	0.01	NS
	G_3	2.61±0.38 (17)	2.15±0.60 (30)	2.11±0.56 (68)	0.01	0.01	NS
FEV_1 (L)	G_1	2.90±0.38	1.85±1.02	1.99±0.67	0.01	0.01	NS
	G_2	3.59±0.41	1.53±0.74	1.76±0.48	0.01	0.01	NS
	G_3	2.28±0.33	1.39±0.52	1.37±0.61	0.01	0.01	NS
FEV_1/FVC%	G_1	95.27±9.94	61.10±19.25	73.60±23.41	0.01	NS	0.01
	G_2	93.76±4.76	65.89±26.69	64.14±33.45	0.01	0.01	0.01
	G_3	92.35±9.62	60.87±30.80	62.96±34.75	0.01	0.01	NS
$FEF_{25-75\%}$ (L/s)	G_1	5.85±0.91	2.30±1.00	2.21±0.69	0.01	0.01	NS
	G_2	4.77±0.67	2.19±0.54	1.97±0.51	0.01	0.01	NS
	G_3	4.06±0.56	1.76±0.62	1.63±0.57	0.01	0.01	NS
$FEF_{0.2-1.2\%}$ (L/s)	G_1	7.81±1.28	2.99±1.31	2.54±0.83	0.01	0.01	NS
	G_2	6.87±1.24	2.60±1.17	2.33±0.84	0.01	0.01	NS
	G_3	5.19±1.05	1.93±0.86	1.79±0.87	0.01	0.01	NS
$FEF_{25\%}$ (L/s)	G_1	7.92±1.24	2.57±1.88	2.69±1.08	0.01	0.01	NS
	G_2	6.99±1.09	2.45±1.44	2.06±1.29	0.01	0.01	NS
	G_3	5.89±1.15	1.84±1.12	1.75±1.22	0.01	0.01	NS
$FEF_{50\%}$ (L/s)	G_1	5.78±1.04	1.93±1.46	2.42±1.16	0.01	0.01	NS
	G_2	5.34±0.70	2.13±1.06	1.78±1.02	0.01	0.01	NS
	G_3	4.38±0.70	1.58±0.91	1.48±0.92	0.01	0.01	NS
$FEF_{75\%}$ (L/s)	G_1	3.80±1.04	1.21±0.81	1.53±0.68	0.01	0.01	NS
	G_2	3.39±0.38	1.33±0.60	1.06±1.59	0.01	0.01	NS
	G_3	2.94±0.39	0.94±0.48	0.92±0.53	0.01	0.01	NS

G_1<35 years; G_2 35-45 years; G_3 > 45 years; parenthesis indicate number of subjects; NS – not significant

A significant negative correlation between exposure duration and pulmonary functions was observed. With increase in the duration of exposure, lung volume, i.e. FEV_1 ($P<0.01$) and flow rates, viz. $FEF_{25-75\%}$ ($P<0.01$), $FEF_{0.2-1.2}$ ($P<0.01$); $FEF_{25\%}$ ($P<0.05$), $FEF_{50\%}$ ($P<0.05$) and $FEF_{75\%}$ ($P<0.01$) were found to be significantly decreased (Table II).

TABLE II: Correlation analysis of pulmonary functions with exposure duration of FACT workers.

Parameters (units)	*Correlation coefficient (n = 175)*
FVC (L)	–0.1175
FEV_1 (L)	–0.2635**
FEV_1/FVC%	0.0754
$FEF_{25-75\%}$ (L/s)	–0.3130**
$FEF_{0.2-1.2}$ (L/s)	–0.3657**
$FEF_{25\%}$ (L/s)	–0.2060*
$FEF_{50\%}$ (L/s)	–0.1913*
$FEF_{75\%}$ (L/s)	–0.2130**

*Significant for $P<0.05$; ** Significant for $P<0.01$

DISCUSSION

This study demonstrated that there is significant decrease in lung volumes and flow rates in non-smoker and smoker workers of FACT in comparison to normal controls. The difference between smokers and non-smokers was insignificant, though the smokers deviate differently from non-smokers. Significant reduction in lung volume and flow rates of smoker and non-smoker workers establish the fact that chronic industrial exposure along with or without smoking is associated with small and large airway obstruction and restrictive condition of lung. Workers employed in FACT are always exposed to a large variety of gases, viz. CO_2, CO, SO_2, chlorine, formaldehyde, ammonia, various oxides of nitrogen, H_2S, H_2SO_4 and particular matter of varying size. Nitrous fumes cause sudden bronchospasm and death from respiratory failure at a concentration of 100-500 ppm. Irritation to upper respiratory tract along with severe alveolar oedema occurs as a result of these fumes (2). These noxious gases, along with dust particles get deposited in the surfactant layer of alveoli, decrease the elastic recoil forces of the lung. Once the noxious gases enter the blood, they bind with haemoglobin or other binding proteins. They are transported to the various parts of the body and exert their effort on respiratory centres. These chemical substances act directly or indirectly through chemoreceptors of the sino-aortic mechanism resulting in respiratory distress leading to reduction in lung volumes and flow rates. It will be worth to note that in low concentrations these toxic substances can stimulate or induce respiration through respiratory centres, but in higher concentration they suppress respiration (3).

The inhaled pollutants bring about the narrowing of the airways followed by alteration in the mechanical properties of the alveolar walls leading to measurable loss of elastic recoil of lung and finally to the overt development of irreversible air flow limitation (4). Airway pollutants such as cigarette smoke, inhaled dust and noxious gases cause bronchoconstriction reflexly through stimulation of receptors in the trachea and large bronchi (5). The elastic

properties of the lung depend on the physical characteristics of the lung tissue and the surface tension of the film lining the alveolar wall. With the deposition of pollutants on the alveolar wall and surrounding bronchioles, elastic properties of the lungs are affected resulting in the reduction of lung volumes. The decrease in flow rates in the present work may be due to the deposition of dust particles which might increase the inflammatory secretion of mucus which in turn results in increase in the airway resistance. This increase in the airway resistance and mucus secretions obstructs the breathing process. Results of the present study support the earlier observation made by Astrand and Rodalh (6) that the constriction of bronchi results in decreased flow rates. The increase in resistance in upper and lower airways results in reduction of PEF, $FEF_{25-75\%}$, $FEF_{75-85\%}$ and $FEF_{0.2-1.2}$ (7). Pollutants such as ammonia, nitrogen dioxide, sulphur dioxide and dust in fertiliser industry have been reported to reduce the lung volumes such as FVC, FEV_1 and FEV_1/FVC% and expiratory flow rates of the workers (8).

Miller et al. (9) reported that reduction in FVC is not necessarily indicative of restriction caused by deposition of dusts but as a result of trapping of air. A reduction in FEV_1 and flow rates is proportional to the reduction in FVC. Therefore, a reduction in FEV_1/FVC% results in obstruction of air passage. Present findings support the earlier observations. Polyvinyl chloride (PVC) workers exposed to HCl, CO, CO_2 and benzene show acute and chronic obstructive ventilatory changes (10). The present work is in agreement with observation of Kolarzyk et al (11) that exposure to SO_2 and NO_x gases cause reduction in FEV_1 and $FEF_{25-75\%}$.

Joshi (8) was of opinion that reduction in FVC is largely due to the effect of smoking rather than exposure to industrial environment. Kreiss et al. (12) observed that smoking had a modifying effect on the pattern of respiratory function impairment. In the present study no significant difference was observed between smokers and non-smokers in pulmonary functions except FVC in G_1. Similar observations were also reported by Korn et al (13). It is clear from this that industrial dust exposure may be associated with change in pulmonary functions and smoking accentuates these changes.

Cigarette smoking and noxious gases together potentate each other diseases (14). Apart from the pollutants present in chemical factories, smoking produce restrictive and obstructive disturbances in ventilation (15) while Gupta *et al.* (16) failed to find any link between respiratory impairment and duration of exposure with smoking among industrial workers. In the Surgeon General's Report of 1985 from US Public Health Service on Cancer and Chronic Lung Disease in Work Place (17), it was mentioned that interaction between smoking and the exposure is complex and needs further development of techniques to study these interactions. The present study also highlights the above plea. The evidence of interaction between these two factors has not been consistent.

Mukhtar and Mohan Rao (18) were of the opinion that exposure to industrial atmosphere may lead to decrease in VC.

Schwartz et al (19) reported a negative correlation between pleural thickening and FVC. Our observations suggest that lung volumes and flow rates decrease with increase in the duration of exposure to industrial pollutants.

In conclusion, it seems that exposure to different chemicals, noxious gases and particulate matter of FACT was injurious to the normal pulmonary function. Significant decrease in lung volumes and flow rates among non-smokers and smokers than that of normal controls was observed. But the difference between smokers and non-smokers was insignificant. It was found that with increased duration of exposure to toxic pollutants, lung volumes and flow rates were significantly reduced in all workers.

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REFERENCES

1. American Thoracic Society (1987). Standardisation of spirometry – 1987 update. *Am Rev Respir Dis* 136: 1285–1298.
2. Waldron HA. Lecture Notes on Occupational Medicine, 3rd edn. Oxford, Blackwell Scientific Publications 1985: 47–163.
3. Berne RM, Levey MN. Physiology, 2nd edn. The C. V. Mosby Publications 1988: 575–645.
4. Dosman J, Bode F, Urbanetti J, Martin R, Macklem PT. The use of helium-oxygen mixture during maximum expiratory flow to demonstrate obstruction in small airways in smokers. *J Clin Invest* 1975; 55: 1090–1099.
5. West JB. In: Best and Taylor's Physiological Basis of Medical Practice, 11th edn. Baltimore, Williams Wilkins Publications 1985: 586–629.
6. Astrand PO, Rodalh K. Textbook of Work Physiology–Physiological Bases of Exercise, 3rd edn. McGraw-Hill Publications 1986: 209–267.
7. Governa M, Comai M, Valenteno M, Antonicelli L, Rinaldi F, Pisani E. Ventilatory function in rubber processing workers: acute changes over the work-shift. *Br J Ind Med* 1987; 44(2): 83–89.
8. Joshi VS. A study of forced vital capacity and expiratory flow rates in urea plant workers. M. D. Thesis, Baroda, M. S. University 1994.
9. Miller A, Lilis R, Godbold J, Chem E, Wu X, Selikaff JJ. Spirometric impairments in long-term insulators: Relationships to duration of exposure, smoking and radiographic abnormalities. *Chest* 1994; 105: 175–182.
10. Baser ME, Tockman MS, Kennedy TP. Pulmonary function and respiratory symptom in polyvinyl chloride fabrication workers. *Am Rev Respir Dis* 1985; 131(2): 203–208.
11. Kolarzyk E, Pach J, Targosz D, Gauszka Z, Stala Z. Evaluation of respiratory function in coke workers. II. Influence of 8-hour exposure on ventilation efficiency. *Folia Med Cracov* 1990; 31(4): 143–159.
12. Kreiss K, Greenberg LM, Kogul SJH, Lezolte DC, Irvin CG, Cherniack RM. Hard-rock mining exposure affect smokers and non-smokers differently. *Am Rev Respir Dis* 1989; 139: 1487–1493.
13. Korn RJ, Dockery DW, Speizer FE, Ware JH, Ferrus BG. Occupational exposures and chronic

respiratory symptoms – A population-based study. *Am Rev Respir Dis* 1987; 136: 298–304.

14. Mikaelsson B, Stjernberg, Nils, Gosta WL. The prevalence of bronchial asthma and chronic bronchitis in an industrialised community in Northern Sweden. *Scand J Soc Med* 1982; 10: 11–16.

15. Lewin-Kowalik J, Krause M, Poborska K, Barski JJ, Gorka D, Gorka B, Kwiek S, Larysz-Brysz M, Haber-Garlinska J. Occupational exposure of workers in chemical factories and selected parameters of the respiratory system. *Med Pr* 1994; 45(3): 227–238.

16. Gupta BN, Rastogi SK, Husain T, Mathur N, Pangtey BS. A study of respiratory morbidity and pulmonary function among solders in the electronics industry. *Am Ind Hyg Assoc J* 1991; 52(2): 45–51.

17. US Public Health Service. Cancer and chronic lung disease in the workplace. A report of the Surgeon General. Rockville, MD, US Department of Health and Human Service, Public Health Service, Office on Smoking and Health 1985: 1–512.

18. Mukhtar MR, Mohan Rao GM. Respiratory effects of occupational exposure to asbestos. *Indian J Physiol Pharmacol* 1996; 40(1): 98–102.

19. Schwartz DA, Fourtes LA, Galven JR, Schmidt LE, Leistikow BN, La Marse FP, Merchant JA. Asbestos-induced pleural fibrosis and impaired lung function. *Am Rev Resp Dis* 1991; 141: 321–326.