Evaluation of Peak Expiratory Flow Rate in Tea Garden Factory Workers in Dibrugarh District, Assam

Authors
Dr (Mrs) Rumi Konwar¹, Dr (Mrs) Wasima Jahan²
¹Asstt. Prof. of Physiology, Assam Medical College, Dibrugarh
²Prof. and Head, Deptt of Physiology, Assam Medical College, Dibrugarh

Corresponding Author
Dr Rumi Konwar
Asstt Prof of Physiology, Assam Medical College, Dibrugarh (786001), India
Email: rumz6034@gmail.com, Mobile no. 9508430288

ABSTRACT
Introduction: Tea which is brewed from the tea plant Camellia sinensis is an indispensable beverage used the world over. Manufacture of black tea involves several labour-intensive processes: sifting and blending being the dustiest processes in the tea industry thereby exposing the workers involved to the hazards of dust inhalation.

Materials and Method: The present study was carried out to evaluate the Peak Expiratory Flow Rates (PEFR) of tea garden factory workers in Dibrugarh district of Assam in India and study the effect of duration of exposure to pollutants in them. A Mini Wright Peak Flow Meter was used. Study population comprised of 210 male factory workers (cases) divided into 3 groups (70 in each) according to their duration of exposure as Group 1: < 1 year, Group 2: 1–2 years, Group 3: > 2 years to 10 years duration of exposure. A control group was taken comprising of 70 male field workers with no exposure to dusty environment. Results analysed applying ANOVA.

Results: PEFR values of Group 3, when compared to the control group, showed a highly significant decrease (p < 0.01). When the PEFR value was compared among the 3 groups, it showed a highly significant decline (p < 0.01) with increase in duration of exposure.

Conclusion: The study reveals that the PEFR values of the tea factory workers were lower than that of the Control group and that with increased duration of exposure to dust within the factory, there was a decline in their PEFR values.

Keywords: Peak Expiratory Flow Rate, Tea Garden Factory Workers, dust.

INTRODUCTION
A person, apart from the environmental pollution, is exposed to the polluted air by virtue of his/her occupation and one of the most affected organs are the lungs. Exposure to hazardous materials and processes at the workplace can cause or exacerbate a multitude of diseases. Physicians commonly treat the sequelae of such diseases in the practice of medicine; however, unless the underlying connection with hazardous exposure is identified and mitigated, treatment of manifestations rather than the cause, at best only ameliorates the
condition. At worst, the neglect of hazardous exposures may lead to both failure of treatment and failure to recognize a public health problem with wide significance \[1\]

Identification of an environmental/occupational etiology of an illness may have important economic ramifications for the patient (e.g. awarding of workers compensation, which covers medical bills as well as lost wages). The assessment is important because removal of a patient from a harmful environment is often the only intervention that might prevent further significant deterioration or lead to improvement in the patient’s condition. The identification of an environment associated disease in a single patient may lead to primary preventive strategies affecting other similarly exposed people who have not yet developed disease.\[2\] The tea industry is one such dusty industry wherein the tea factory workers are exposed to a dusty environment. Tea, which is an indispensable beverage used world over, is brewed from the tea plant Camellia sinensis. The tea industry of Assam produces \(\sim\)51% of tea produced in India and about 1/6th of tea produced in the world. Manufacture of black tea involves several labour-intensive processes. Sifting and blending are the dustiest processes in the tea industry and the workers involved are exposed to the hazard of inhaling the dust. Inhalation of tea dust is known to give rise to both acute and chronic respiratory symptoms. A large number of studies on lung function on different types of factory workers are available, but similar studies among teagarden factory workers are not much available and hence this study was carried out amongst the tea garden factory workers in Dibrugarh district of Assam, India.

**AIMS AND OBJECTIVES**

- To assess the Peak Expiratory Flow Rate in tea garden factory workers.
- To study the effect of duration of exposure to pollutants on PEFR in the tea garden factory workers.

**MATERIALS AND METHOD**

Present study was conducted on 210 apparently healthy male tea garden factory workers (cases) and 70 apparently healthy male field workers (control) from different tea gardens of Dibrugarh. Only male workers aged 20 – 50 years were selected as the number of females working within the tea factory were very few. Prior to conducting this study the subjects were made to understand in brief why the study was carried out and only those who were cooperative were considered and relevant physical examination was done.

**Inclusion Criteria**

1. Healthy male tea garden factory workers with no previous history of respiratory or other significant illnesses were taken.
2. Duration of exposure to dust amongst the factory workers was noted and divided into 3 groups (70 cases in each):
   - Group 1: \(<1\) year
   - Group 2: \(1–2\) years
   - Group 3: \(>2\) upto 10 years duration of exposure
3. Control group: comprised of male field workers of the tea gardens with no exposure to dusty environment by virtue of their occupation.

**Exclusion Criteria:** Tea garden factory workers with respiratory symptoms or history of any known lung diseases like asthma, TB etc. & any known cardiac dysfunctions were excluded from the study.

Mini Wright Peak Flow Meter was used to measure PEFR. The apparatus is small, simple, portable, easy to handle and the most commonly used instrument the world over. The manoeuvres were explained and demonstrated to the subjects. Each subject was given a few trial runs of at least two until he did the procedure correctly. The subject thus, inspired as deeply as possible and blew out into the Wright Peak Flow Meter as hard and rapidly as he could in one sharp, short blast. Three readings were taken and the highest reading was accepted in each case. They were all examined in standing posture. Anthropometer was used to measure height and weighing machine used to
obtain the weight. Body surface area in square meters was calculated using DuBois formula.

Du Bois formula:

\[ \text{BSA} = (W^{0.425} \times H^{0.725}) \times 0.007184 \]

**Statistical analysis** and the p value obtained using ANOVA technique.

### RESULTS

**TABLE–1:** Shows the Mean Values of the Physical Parameters and PEFR Value of Male Tea Garden Factory Workers

| PHYSICAL PARAMETERS | TOTAL CASES (n = 210) | MEAN ± S.D. |
|---------------------|-----------------------|-------------|
| Age (years)         | 28.58                 | 6.22        |
| Height (cms)        | 159.46                | 5.44        |
| Weight (Kg)         | 61.11                 | 4.33        |
| BSA (sq.m)          | 1.63                  | 0.07        |
| PEFR (Lit/min)      | 441.00                | 37.26       |

In the above Table–1, physical parameters (mean values ± SD) and PEFR value of 210 male workers in tea garden factories have been shown.

**Table 2:** Shows the Effect of Duration of Exposure on PEFR in Different Groups

| GROUP   | No. OF CASES | DURATION OF EXPOSURE (years) | PEFR (Lit/min) | MEAN ± S.D. |
|---------|--------------|-------------------------------|----------------|-------------|
| 1       | 70           | < 1                           | 460            | 39.07       |
| 2       | 70           | 1—2                          | 458            | 18.37       |
| 3       | 70           | > 2                           | 405            | 18.60       |
| CONTROL | 70           | –                             | 466            | 14.02       |

It was seen that the mean PEFR values of Group 1, 2 and 3 was 460, 458 and 405 liters/min respectively. The mean PEFR in the control group was found to be 466 liters/min.

Results were analyzed applying ANOVA and it was found that, on comparing with the Control group the decline in Group 1 and 2 was insignificant (p > 0.05); however the decline in Group 3 was highly significant (p < 0.01). Also when results of all the three groups were compared among themselves it was found that the decline in the values of PEFR from Group 1, Group 2 to Group 3 respectively were found to be highly significant (p <0.01).

### DISCUSSION

In the present study, the effect on the PEFR due to exposure to dust within the factory according to their duration of exposure was compared with a control group as well as compared within Groups 1, 2 and 3 themselves. In healthy adult males depending on their size and age the normal PEFR is 350 – 800 L/min [3]. The present study values was found to be within normal range (Table 1), though on the lower side of the normal range. This could be due to the difference in body build. The PEFR values showed a significant decrease (p < 0.01) when Group 3 and Control group’s values were compared (Table 2); while the other two groups reported no significant (p > 0.05) decrease.

When the effect on PEFR among the 3 groups (Group 1,2 &3) were analyzed using ANOVA it was found that the mean values of PEFR significantly decreased (p <0.01) with increase in duration of exposure. This finding matches with a study carried out in Sri Lanka on “Ventilatory function of factory workers exposed to tea dust” by Jayawardana et al, 1997 where factory workers exposed to tea dust for a period of at least 5 years appear to be at risk of developing ventilatory dysfunction. He found lower values of lung function parameters when compared to Control group’s values [4].

Eugenija Zuskin and Zdenka Skuric, University of Yugoslavia (1984) carried out a similar study on respiratory function in five group of tea workers employed in processing different type of tea. They found significant decline in ventilatory functions in workers exposed to tea dust.[7]. KC Sarkar, et al. Department of Physiology, North Bengal Medical College conducted a study on 64 tea garden factory workers in West Bengal in March 2010. They found
the value of vital capacity, FVC, FEV1, FEV25-75%, PEFR were significantly lower in workers having chronic exposure to tea dust [8].

A related study on PEFR conducted by Sanjay. P. Zodpey and R.R. Tiwari (1997) on flour mill workers [6], to see the effect on PEFR values with increased duration of exposure to flour mill grain dust, found lower values of PEFR among workers exposed for longer duration. They concluded that lower values are probably due to hypertrophy of the mucosal cells due to irritation by grain dust and smoke resulting in increased secretion of mucous and formation of mucosal plugs which cause obstruction to the exhaled air.

CONCLUSION

The present study reveals that the PEFR values of the tea factory workers were lower than that of the Control group. It also reveals that with increased duration of exposure to dust within the factory, there was a decline in PEFR values of the factory workers. In the course of this study, it came to light the fact, that, studies on lung function in tea garden workers are very few. The results of the study cannot be taken as representative of characteristics of a region. Assam, being in itself, a mini India, values are likely to vary from place to place and region to region. The findings of the present study, therefore, should be considered as preliminary and this calls for further detailed study and statistical analysis with a large sample size. However, it is hoped that this study will be of help for further studies on this topic thereby helping in assessing the health status of the workers and take remedial or preventive measures as needed, such that a healthy environment for the workers is ascertained.

REFERENCES

1. Howard Hu, Frank E Speizer, Influence of environmental and occupational hazards on disease, Chapter 5, Harrison’s Principles of Internal Medicine, 15th edition, pg 19.
2. Frank E. Speizer, Environmental Lung Disease, Harrison’s Principles of Internal Medicine, 15th edition, pg 1467.
3. Cotes. J. E., 2nd Edition, Lung Function, Assessment and application in Medicine, Pg: 2–6, 60–61, 74, 86–88, 331.
4. PL Jayawardana and M Udupihille, Ventilatory function of factory workers exposed to Tea dust, Occup. Med. Vol. 47, No. 2, pp105–109, 1997.
5. Rajnarayan R. Tiwari etal. Peak Expiratory Flow Rate in Handloom weavers, Indian J Physiol Pharmacol 1998; 42(2): 266–270.
6. Sanjay P. Etal.: Peak expiratory flow rate in flour mill workers. Indian J Physiol Pharmacol 1998; 42(4): 521–526.
7. Zuskin E, Skuric Z, Respiratory function in tea workers. Br J Ind Med.1984;41:88–93.
8. Sarkar KC, Chakraborty S, Mukherjee AK,MandalDS,MandalS (Department of Physiology, North Bengal Medical College, Sushrutanagar), Study of Pulmonary Function in Tea Industry workers in North Bengal.J Indian Medical Study association. 2010;108(3):144,146–7
9. RM Castellan, et al.Pulmonary functions and symptoms in herbal tea workers, Chest 1981; 79; 81–85.
10. Cotes. J. E., 2nd Edition, Lung Function, Assessment and application in Medicine, Pg: 2–6, 60–61, 74, 86–88, 331.
11. Engel S. (1962) Lung structure. C. C. Thomas, Springfield, Illinois.
12. Thoracic Society of Great Britain (1950) The nomenclature of bronchopulmonary anatomy. Thorax 5, 222–228.
13. Von Hayek H. (1960) The human lung, translated by V. E. Krahl. Hafner Publ. Co. Inc., New York.
14. Wilson T. A. (1967) Design of the bronchial tree. Nature 213, 668–669.
15. Khurana I. (2006) Pulmonary Diffusion, Textbook of Medical Physiology, 1st edition, 400, 435.
16. Guyton and Hall, 11th edition, Textbook of Medical Physiology, Pg 496–497, 474, 483, 479, 471.
17. Cole RP: CO2 and lung mechanical or gas exchange function. Crit Care Med 32: 1240, 2004.

18. West JB: Pulmonary Physiology and Pathophysiology: An Integrated, Case-Based Approach. Philadelphia: Lippincott Williams and Wilkins, 2001.

19. West JB: Pulmonary Physiology—The Essentials. Baltimore: Lippincott Williams and Wilkins, 2003.

20. Guazzi M: Alveolar–capillary membrane dysfunction in heart failure: evidence of a pathophysiological role. Chest 124: 1090, 2003.

21. Hilaire G, Duron B: Maturation of the mammalian respiratory system. Physiol Rev. 79: 325, 1999.

22. Clements J. A. (1962) Surface phenomena in relation to pulmonary function. Physiologist 5, 11–28.

23. Pattle R. E. (1965) Surface lining of lung alveoli. Physiol. Rev. 45, 48–79.

24. Wright JR: Pulmonary Surfactant: a front line of lung host defense. J Clin Invest 111: 1453, 2003.

25. Guyton AC, Parker JC, Taylor AE, et al: Forces governing water movement in the lung. In: Fishman AP, Renkin EM(eds): Pulmonary Edema. Baltimore: Waverly Press, 1979, p 65.

26. Guyton AC, Taylor AE, Granger HJ: Circulatory Physiology II. Dynamics and control of the body fluids. Philadelphia: WB Saunders, 1975.

27. Carr MJ, Undem BJ: Bronchopulmonary afferent nerves. Respiratory 8: 291, 2003.

28. Coulson FR, Fryer AD: Muscarinic acetylcholine receptors and airway diseases. Pharmacol Ther 98: 59, 2003.

29. Levitzky MG, Pulmonary Physiology, 7th edition

30. Campbell E.J.M., et al. (1963) Clinical Physiology, 2nd edition. Blackwell Scientific Publications, Oxford.

31. Comroe J. H. Jr, et al. (1962) The lung, clinical physiology and pulmonary function tests, 2nd edition. Year Book. Medical Publishers Inc., Chicago.

32. Dejours P. (1966) Respiration, translated by L. E. Farhi. Oxford University Press, New York and London.

33. Fenn W. O. and Rahn H. (eds) (1964) Handbook of Physiology Section 3: Respiration. Vols I and 2. American Physiological Society, Washington DC.

34. Cyril A. Keele, Eric Neil and Norman Joels(1985), Lung volumes and capacities, Samson Wright’s Applied Physiology, Thirteenth edition, 157.

35. Bouhuys, A. (1977). The physiology of breathing. Frune and Stratton, New York.

36. William F. Ganong, Pulmonary Function, Review of Medical Physiology, 22nd edition, 652, 661.