STUDY OF "LUNG AGE" IN PETROL PUMP WORKERS IN RESPECT OF HEALTHY NORTH INDIAN SUBJECTS

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Abstracts: **Background & objectives:** The numbers of motor vehicles and in turn petrol pumps in India are increasing rapidly due to growing urbanization. These petrol pump workers are subjected to significant health hazards, especially on the respiratory system. Hence, Lung age is selected as a newer and easier way of expressing lung damage in petrol pump workers and comparative evaluation with that of healthy general population. **Methods:** A total of 100 male Petrol Pump workers, non-smokers, working for more than 1 year and within 20-50 years of age were selected by Simple Random Sampling from different petrol stations of Bareilly and formed the Study Group. Their percent predicted values of Lung Age were compared with 100 age and sex matched nonsmoker controls from the general population. **Results:** In this study, mean value of Lung Age (percent predicted value) in Study Group (132.37 ± 11.05) was significantly (p=0.001) higher than Control Group (105.85 ± 10.37). In each of the age-group i.e. e Group I (20-<30 years), Group II (30-<40 years) & Group III (40-<50 years), the participants in the study group had significantly higher mean value of lung age than controls. Further, as the duration of exposure increased, Lung age increased significantly among the petrol pump workers. **Interpretation & conclusion**: Deterioration of lung function observed in petrol pump workers in respect of lung age. Ageing and exposure to fuels are additive factors contributing this. Fuel vapour induced airway remodelling, impaired mucociliary clearance, oxidative stress, immunological destruction of bronchial epithelial cells and bronchoconstriction by TRVP1 receptor could play a significant role in this deterioration of lung age. "Lung age" can be widely used for community screening and mass understanding purpose rather than conventional parameters.

Key Words: Lung age, percent predicted values, TRVP1 : Transient Receptor Potential Vanilloid type 1

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Introduction: India is a vast country with a surface area of about 3.3 million square kms and ranked as second most populous country in the world.[1] For the last few decades, Indian cities are witnessing rising urbanization and an increasing usage of private vehicles to maintain the fast pace of modern lives. This precipitous hike in the vehicular traffic density consequently led to the establishment of more and more petrol pumps to cater to the growing demands of the society. Simultaneously, fuel consumption has also risen in successive years. Therefore, to mitigate this enormous and gigantic fuel thirst, the number of petrol stations in India are continuously soaring and approximately 56,190 petrol stations (as of March 2016) are functioning in the country.[2] With its ever growing demand, gradually petrol and diesel has become an essential commodity to sustain our life and certain groups of our society, who serve this fuel, by virtue of their occupation, face an increasing threat of its adverse health effects. Long term exposure to petrol vapours has shown to affect the different physiological systems in the body, with the highest impact on the respiratory system.[3] Petrol-pump workers who are exposed to the petrol fumes exhibit a number of clinical signs and symptoms which may be due to benzene toxicity. Symptoms like chronic cough, wheezing and breathlessness have been reported on exposure to these pollutants.[4, 5]

Lung age can be defined as the ratio of a person's current lung function, to the age at which his or her lung function would be considered normal.[6] Although various epidemiological studies conducted nationally[7,8,9] and internationally [10,11,12] have documented decrements in pulmonary function and various other health problems associated with long-term petrol and diesel exposure; however, no documented study of "Lung age" in petrol pump workers, especially in northern India could be found in literature. Hence, this study was undertaken.

Material and Methods: This was a cross-sectional study carried out in October 2015- September 2016 in the Dept. of Physiology, Rohilkhand Medical College & Hospital after obtaining ethical approval from the Institutional Ethical Committee. Sample size was calculated with the formula $4pq/L^2$; where P (Prevalence) = 50%, Q=100–P= 50%, L (allowable error) = 20% of prevalence=10. A total of 100 male Petrol Pump workers, non-smokers, working for more than 1 year and within 20-50 years of age were selected by Simple Random Sampling from different petrol stations of Bareilly and formed the Study Group. Their percent predicted values of Lung Age were compared with 100 age and sex matched nonsmoker controls from the general population. Those with respiratory disease like tuberculosis, bronchial asthma, COPD; those with chronic disease like diabetes mellitus, hypertension; history of regular medication like sedative or hypnotics; those with major abdominal or thoracic surgery in past or inability to perform pulmonary function test were excluded from the study.

After obtaining the informed consent, basic anthropometric measurements were recorded and subjects were made to sit and relax for minimum 5 minutes prior to performing the procedure. The procedure was thoroughly explained to each subject and asked to take full inspiration which was followed by rapid and forceful expiration with closed nostrils in the mouthpiece. RMS HELIOS-401 software provided a detailed analysis of predicted value, derived value and percent predicted values of Lung Age. The Lung Age calculation were repeated thrice on each occasion for each subject and the best reading percent predicted values of Lung Age were selected for analysis as per guidelines of American Thoracic Society. [13, 14, 15]

Data was analyzed with SPSS and results were expressed as mean ± standard deviation (SD). Statistical tests like Independent Sample't' test, One-way ANOVA, Tukey's Post-Hoc Test & Pearson's Correlation test were applied. P value of <0.05 was considered significant.

Result: Table 1 shows that mean height, weight, BMI and age were not significantly different between Study and Control group. But, mean Lung Age (% predicted) in Study Group (132.37 ± 11.05) was significantly (p=0.001) higher than Control (105.85 ± 10.37) depicted in Graph 1.

Graph- 1: Comparison of Lung Age (% predicted) in Study and Control Group

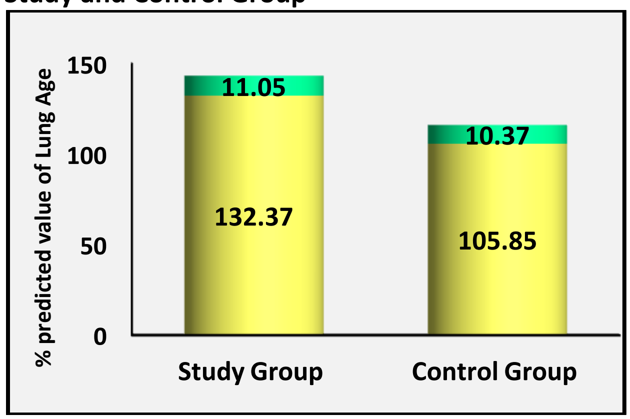


Table 1 : Comparison of anthropometric parameters and Age between Study Group and Control Group

	GROUP	Mean	Std. Deviation	Std. Error Mean	Significance
HEIGHT (meter)	**Study Group**	1.66	0.05	0.01	0.176
	Control Group	1.65	0.06	0.01	
WEIGHT (Kg)	**Study Group**	69.35	7.87	0.78	0.256
	Control Group	68.05	8.25	0.82	
BMI (Kg/m^2)	**Study Group**	25.09	2.53	0.25	0.639
	Control Group	24.92	2.47	0.24	
AGE (years)	**Study Group**	33.93	7.75	0.77	0.115
	Control Group	32.33	6.94	0.69	

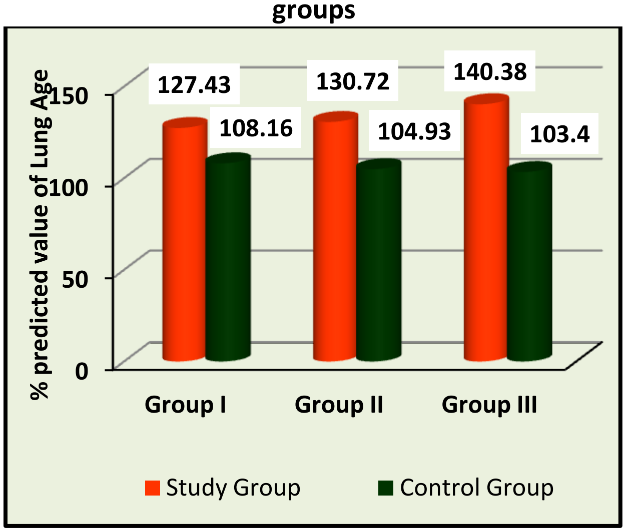
For comparison of lung age, both Study & Control groups were subdivided into Group I, II & III as shown in Table 2. In each age group; mean height,

Table 2 : Anthropometric parameters of different age groups

Age limit	Groups	Study Group				Control Group			
		No.	Height (meter)	Weight (Kg)	BMI (Kg/m^2)	No.	Height (meter)	Weight (Kg)	BMI (Kg/m^2)
20 - <30 years	**Group I**	35	1.65	67.66	24.76	38	1.64	65.61	24.31
30 - <40 years	**Group II**	36	1.66	70.92	25.65	42	1.65	69.05	25.10
40 - <50 years	**Group III**	29	1.67	69.45	24.79	20	1.65	70.60	25.71

weight and BMI of Study group was not significantly different from Control group. But,
Lung age (%pred value) was significantly (p=0.001) higher in all groups of the study group as compared to the control Group (as depicted in Graph 2).

Graph- 2: Comparison of Lung Age in three age groups



'One-way ANOVA' test results of comparison of lung age among Group I, II & III of study goup showed that there was a statistically significant difference between these groups {F (2,97)=14.699, p=0.001}(effect size 0.233). Post-Hoc analysis

result of the same between different age groups are shown in Table 3. 'Pearson Correlation test' result between Lung age and age groups revealed a moderate strong significant positive (r=0.462, p=0.001, n=100) correlation.

Table 3 : Post-Hoc Tukey's Test of Lung Age among the Age Groups of Petrol pump workers

Parameter	group	Comparison	Sig
LUNG AGE	Group I	Group II	0.335
	Group II	Group III	.001**
	Group I	Group III	.001**
*'p value' < 0.05 considered as significant			

Further to find out effect of duration of exposure Study group was again divided into three subgroups as depicted in Table 4. 'One-way ANOVA' test results of comparison of anthropometric parameters among three Group A, Group B and Group C of Study Group revealed non-significant difference but there was a statistically significant difference (Table 4) of mean Lung Age between

Table 5: Tukey's Post-Hoc analysis of Lung Age between different exposure groups

Parameter	group	Comparison	Sig
LUNG AGE	Group A	Group B	.029*
	Group B	Group C	.006**
	Group A	Group C	.001**

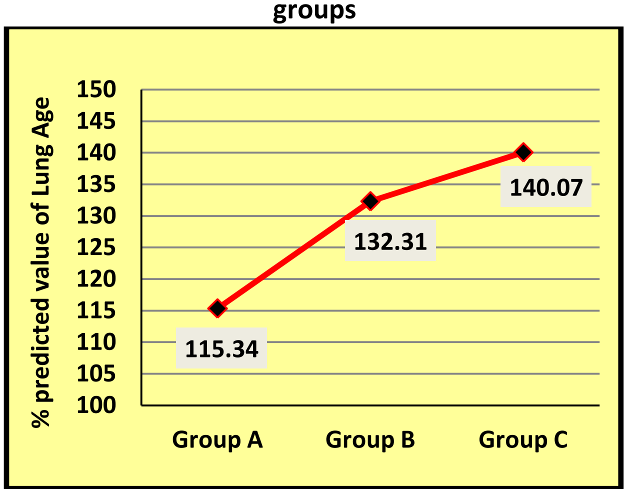
Table 4 : Comparison of mean anthropometric parameters among three duration of exposure groups

Age groups	Exposure	Height (meter)	Weight (Kg)	BMI (Kg/m^2)	Mean Lung Age (% pred)
Group A	>1year- 3 years	1.64	66.76	24.70	115.34
Group B	>3 years- 5 years	1.66	72.06	25.89	132.31
Group C	>5 years- 7 years	1.67	69.73	24.73	140.07
ANOVA Significance (p)		0.53	0.07	0.09	**0.001**

these groups {F(2,97)=95.045, p=0.001}(effect size 0.151). The post-hoc analysis results of the same are depicted in Table 5.

'Pearson Correlation test' results between Lung age and duration of exposure groups of the total 100 petrol pump workers revealed Lung Age (r=0.51, p=0.001, n=100) had a significant positive correlation (Graph 3).

Graph- 3: Change in Lung Age in three exposure groups



Discussion: The concept of "lung age" was developed in 1985 as a way of making spirometry data easier to understand by the common people and also as a potential psychological tool to show smokers the apparent premature ageing of their lungs.[6] Lung age is a way of conceptualizing the deterioration of lung function and a way of expressing lung damage rather than using mathematical concepts of a percentage of the expected value of FEV_1 for height, age and gender.[16] Thus a way to discuss abnormal lung function results with people or patients is to use, the lung age concept, which relates a person's current lung function, to the age at which his/her lung function would be considered normal. Thus, an elevated lung age signifies poor lung function as if the lungs have aged beyond the patient's chronological age.[17,18]

In the present study, mean lung age (% predicted value) in Study Group was (132.37 ± 11.05) significantly (p=0.001) elevated than Control Group (105.85 ± 10.37). Similar finding was reported by Al-Jaddan S A N *et al.*[10]This implies that lung of the petrol pump workers have significantly deteriorated beyond their age. Particles generated from petrol and diesel exhaust are extremely small and can carry a much larger fraction of toxic compounds, such as hydrocarbons and metals on their surface. Also, they can remain airborne for long periods of time and deposit in greater numbers and deeper into the lungs. Hence chronic exposure to them can lead to chronic inflammation of respiratory tract and lung parenchyma. These would contribute to the substantial decrease in lung functions.[19]Also, exposure to these hydrocarbons and nanoparticles ultimately leads to hemorrhagic alveolitis, interstitial inflammation, intra-alveolar hemorrhage and edema, bronchial necrosis and vascular necrosis causing defective lung parenchyma culminating to reduced lung function.[20,21]Toxicologists have attributed the central role in mediating airway inflammation caused by chemical irritants, air pollutants and tissue damaging stimuli to modulation of TRPV1(transient receptor potential vanilloid receptor). These stimuli appear to alter protein conformation and stability, which results in ion influx and disruptions of structural gating. The activation of TRPV1 receptors on sensory fibers and some non-neural cells (e.g. respiratory epithelia) produces calcium and sodium influx and the corresponding release of tachykinin neuropeptides (substance P, neurokinin A, and calcitonin-gene-related peptide). Various resident immune cells (e.g. macrophages), peripheral target cells (endothelia, epithelia), and tissues (smooth muscle) respond to these neuropeptides and mediate the tissue response characteristic of neurogenic inflammation resulting reduction in lung peformance.[22]

In the present study, it was also found that as the age of the workers increased,lung age increased significantly. That implies that petrochemical vapours have an additional role to lung aging for decrease in lung function. This reduction in lung function may be due to destruction of Clara cells, non-ciliated cuboidal epithelial cells that secrete important defense markers and serve as progenitor cells after injury and which make up a large portion of the epithelial lining in the latter portions of the conducting airway. This impedes the natural clearance mechanism of lung.[23]Organic diesel exhaust particles also induce apoptosis and necrosis in bronchial epithelial cells via mitochondrial pathway.[24,25] DEPs also may promote expression of the T_H2 immunologic response phenotype that had been associated with asthma and allergic disease. Further, these exhaust particles appeared to be of greater immunologic effects in the presence of environmental allergens than they do alone.[26] Fuel vapour and vehicular exhaust in addition to air pollutants may cause a change in receptor sensitivity of bronchial smooth muscles. This is supported by a study which emphasized the fact that environmentally relevant polyaromatic hydrocarbons can impede β_2-adenergic receptor mediated airway relaxation.[27] Derangement of other proteins or messengers involved in contraction and relaxation of bronchial smooth muscle may also contribute in this mechanism. Studies found that, perturbations in Ins(1,4,5)P3 (inositol 1,4,5 triphosphate) accumulation, its metabolism and intracellular binding may underlie changes in airway smooth muscle contractility and increased cAMP accumulation, may be due to altered receptor/G protein modulation of adenylate cyclase activity, as well as to altered binding of Ins(1,4,5)P3 to its Ca^{++} mobilizing intracellular receptor are responsible for changes in airway relaxation of smooth muscle.[28]

It was also revealed that as the duration of exposure increased, lung age increased significantly in each exposure group of petrol pump workers. It signifies progressive deterioration of lung function with increased exposure to petrol and diesel fumes. This gradual deterioration of lung function with increased duration of exposure could be attributed to fuel vapour and pollutants induced remodeling of airways. Chronic irritation and inflammation is often associated with increase in collagen deposition and increased recruitment of interstitial inflammatory cells with macrophages. This reduces the elasticity of lung tissues.[29] Continuous accumulation of fuel particles in peri-bronchial lymphoid and connective tissues also lead to varying degrees of bronchial wall thickening and remodeling of terminal and respiratory bronchioles which may be a probable cause for the decrease in pulmonary function in obstructive form.[9]

Conclusion: With the growing body of scientific evidence, it is well known today that Petrol pump workers are vulnerable to develop impairment in lung function as evident in the increased lung age found in the present study also. Aging further increases their susceptibility to lung dysfunction. Also, it can also be deduced that gradual increase in exposure over the years increases the extent of decrement in lung function suggesting a cumulative effect. As lung age as a parameter is simple to understand this can be employed easily for regular monitoring and follow up of petrol pump workers' respiratory health. "Lung age" can be used as an easy way to understand lung damage which can be widely used for community screening and mass understanding purpose rather than conventional parameters.

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