Content available at: iponlinejournal.com

IP Indian Journal of Immunology and Respiratory Medicine

Journal homepage: www.innovativepublication.com

Original Research Article

# Assessment of serum total IGE levels in smokers, non smokers and ex smokers and its relation to lung function, airway symptoms and atopic predisposition

**Swetha Madas[1], C Malliakarjuna Reddy[2,*], M Hima Bindu[2]**

[1]*Dept. of Pulmonary Medicine, RVM Institute of Medical Sciences and Research Centre, Telangana, India*
[2]*Dept. of Microbiology, Malla Reddy Institute of Medical Sciences, Hyderabad, Telangana, India*

ARTICLE INFO

*Article history:*
Received 09-09-2019
Accepted 13-09-2019
Available online 09-12-2019

*Keywords:*
Atopy
Allergens
Immunoglobulin E
Smoking

ABSTRACT

**Introduction:** Many studies have shown an association of smoking with many of the health consequences such as stroke, cardiovascular diseases, chronic respiratory illness, lung cancer and impaired pulmonary function. Increased levels of IgE is found in these people, especially in addition to allergic diseases.
**Materials and Methods:** 310 patients, both men and women above the age of 18 years were included into the study. Blood was collected for absolute eosinophil count and detection of IgE levels. Pulmonary Function test was done for all the patients.
**Results:** Out of the 310 patients 22.3%were male smokers and 5.2% were female smokers. The difference in the total serum IgE count was highly significant in the smokers, with the mean being 329.23 ± 71.3 IU/ml, 28.94 ± 9.2 IU/ml in non smokers, 194.3 ± 23.55 in ex-smokers and 199.3 ± 23.64IU/ml in passive smokers. The Eosinothil count was also significantly high in the smokers, followed by ex smokers, passve smokers in comparison to the non smokers. The FVC was the lowest in the smokers and in the normal range in the non smokers.
**Conclusion:** Smoking is one of the most common modifiable risk factors to a number of diseases prevalent in man. It is associated with the increase in IgE levels, making the patient more sensitive to allergens.



## 1. Introduction

About one-third of the world's population are tobacco smokers, consisting about 1.1 billion over the age of 15 years.[1] Many studies have shown an association of smoking with many of the health consequences such as stroke, cardiovascular diseases, chronic respiratory illness, lung cancer and impaired pulmonary function.[2–4]

Immunoglobulin E (IgE) is said to be associated with allergy such as allergic rhinitis, atopy, asthma, atopic dermatitis, all of which are caused during an increase in the Th2 immune response. These antibodies, which are specific to foreign substances are produced by B cells, during the process of sensitization.[5]

The cause of increase of the total serum Immunoglobulin E in children is not fully known. It is however reported that IgE is higher in men than in women.[6,7] IgE is supposed to sensitise the mast cells and basophil by its binding activity towards the high affinity receptors for the IgE.[8] When there receptors are cross linked by the allergens, they granulate and vasoactive amines chemokines, lipid mediators are liberated.[8]

There is a strong association reported between serum total IgE and male sex, airway hyperresponsiveness, early life wheezing bronchitis, parenteral allergic predisposition, positive skin prick test, atopic dermatitis and hay fever. These are considered to be the predictors of elevated IgE.[9]

One of the most common causes of preventable death is smoking. Tobacco smoke contains very high levels of carbon monoxide and nitrogen oxide and other carcinogen, which have adverse effects on health.[10] Increased levels of IgE is found in these people, especially in addition to allergic diseases.[11]

* *Corresponding author.*
*E-mail address*: cpmreddy@gmail.com (C. Malliakarjuna Reddy).

https://doi.org/10.18231/j.ijirm.2019.048

## 2. Materials and Methods

This study was done by the Department of Pulmonology at RVM Institute of Medical Sciences and Research Centre over a period of 2 years from February 2017 to January 2019. 310 patients, both men and women above the age of 18 years who had come to our department for check up were included into the study, after the nature was thoroughly explained to them and their relatives and an Informed Consent was taken. This study was approved by the Institutional Ethical Committee.

The smokers who were included into the study, were cigarette or bidi smokers or both. Their relatives, who were in close proximity to them during their smoking period were categorised as the passive smokers. Those who used to smoke, but have quit smoking for more than 6 months were considered as ex smokers and those who never smoked were considered non-smokers. Patients with eczema, allergic rhinitis, asthma, unstable cardiac or renal conditions were excluded from the study. Subjects with positive ova/cyst in stool were also excluded from the study.

Detailed demographic data was collected from the patients. Their smoking history, respiratory symptoms such as persistent cough, shortness of breath, wheezing was taken into consideration. Stool was collected for microscopy to rule out ova and cyst.

Blood was collected in vacutainers for complete blood picture, haemoglobin, erythrocyte sedimentation rate, absolute eosinophil count etc. Physical activity was taken as at least 30 mins of moderate exercise 5 times a week. Alcohol consumption was also taken into account, as also was the allergic disposition of the patients. BMI was calculated from the height and weight of the patient.

Non smokers were those who never smoked cigarettes in a ny form in their whole life, while smokers were those who were currently smoking atleast 1 cigarette per day. Ex smokers were those who had smoked earlier, but for the past 1 year had not smoked even one cigarette and passive smokers were those who lived in close proximity to the smokers.

Serum was taken from the patient and IgE was analysed using ELISA. The IgE detected was against pollen, weeds, animals such as dogs, insects and fungi.

## 3. Results

Out of the 310 patients who were included into the study, 149 were males and 161 were females (Figure 1). 69(22.3%) were male smokers and 16(5.2%) were female smokers. 83 females were non smokers while 54 of them were passive smokers, where in most cases, their husbands were smokers.33 males were passive smokers, where in there were fathers/ brothers who were regular smokers in the house (Figure 1).

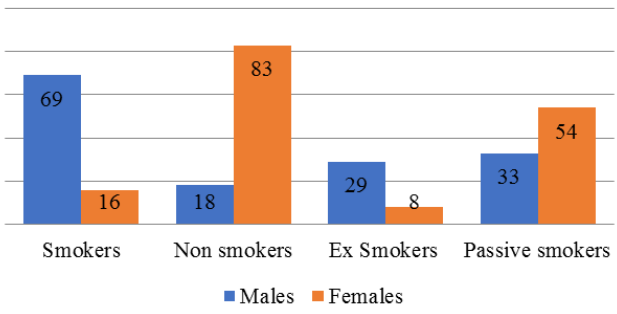


**Fig. 1:** Distribution of smokers, non smokers, ex smokers and passive smokers, and Non smokers

The mean age of the non smokers was 41.9 ± 1.5, while for smokers it was 43.7 ± 2.4, for ex smokers 47.3 ± 4.2, the passive smokers 49.1 ± 2.3, with no significance. The mean BMI was 24.2 ± 0.3 in the non smokers, 22.9 ±0.8 among the smokers, 23.5 ± 0.8 among the ex smokers and 24.1 ± 0.5 among the passive smokers. No significance was observed even in the waist circumference in all the four categories. A moderate physical activity of atleast 30 minutes daily at a time was observed in less than 10% in almost all the categories (Table 1).

The difference in the total serum IgE count was highly significant in the smokers, with the mean being 329.23 ± 71.3 IU/ml, 28.94 ± 9.2 IU/ml in non smokers, 194.3 ± 23.55 in ex-smokers and 199.3 ± 23.64IU/ml in passive smokers. The absolute eosinophil count was also highly significant. In the smokers it was 346 ± 21.54 per mm3, ex smokers, it was 290 ± 42.13 per mm3, 221 ± 34.53 mm3 in the passive smokers and 193 ± 37.3 per mm3 in the non smokers. Even the carbon monoxide presence in the breath was significant in the smokers with 16.3 ± 2.97 ppm, 4.23 ±1.12 ppm in the non smokers, 5.33 ± 2.24 in the ex smokers, and 7.94 ± 2.48 in the passive smokers (Table 2).

The smokers had a higher positivity to allergens by skin prick test (15.2%). The most common was to insects (4.5%), followed by fungal spores and tree and plant pollen (2.9%) each. Only 4 members among the non smokers were allergic to allergens. The most common was pollen seen in 3 pf them. 9.7% of the ex smokers and 16 of the passive smokers showed positivity to allergens (Table 3).

The difference in the pulmonary function test among the four categories was highly significant. The forced vital capacity among the ex-smokers was 75.21 ±2.87%, which was the least compared to smokers which was 84.52 ± 3.54%, 89.62 ± 5.13% in passive smoking and 93.21 ± 13.2% in the non smoking category. The forced expiratory volume in the first second was 70.23 ± 17.23% among the smokers, 51.35 ± 17.36% among the ex smokers, 91.87 ± 27.87% among the passive smokers and 94.7 ±28.65% among the non smokers. The FEV1/FVC ratio was maximum in the non smokers with 99.2 ±29.94%, but

**Table 1:** Demographic details of the patients

| Details | Non smokers | Smokers | Ex smokers | Passive |
|---|---|---|---|---|
| Mean age | 41.9 ± 1.5 | 43.7 ± 2.4 | 47.3 ± 4.2 | 49.1 ± 2.3 |
| Mean BMI | 24.2 ± 0.3 | 22.9 ±0.8 | 23.5 ± 0.8 | 24.1 ± 0.5 |
| Waist circumference (in cm)(kg/m2 | 77.4 ± 2.5 | 78.3 ± 3.8 | 76.0 ± 6.1 | 77.3 ± 7.3 |
| Alcohol (>once /month) | 5 (1.6%) | 68 (21.9%) | 22 (7.1%) | 11(3.5%) |
| Physical activity | 21 (6.8%) | 19 (6.1%) | 23 (7.4%) | 32 (10.3%) |

**Table 2:** IgE, AEC and CO levels in patients

| Details | Non smokers | Smokers | Ex smokers | Passive | p-value |
|---|---|---|---|---|---|
| IgE (IU/ml) | 28.94 ± 9.2 | 329.23 ± 71.3 | 194.3 ± 23.55 | 199.3 ± 23.64 | <0.001 |
| Absolute eosinophil count per mm3 | 193 ± 37.3 | 346 ± 21.54 | 290 ± 42.13 | 221 ± 34.53 | <0.001 |
| Breath CO (ppm) | 4.23 ±1.12 | 16.3 ± 2.97 | 5.33 ± 2.24 | 7.94 ± 2.48 | <0.001 |

**Table 3:** Positive skin prick test to common allergens

| Allergens | Positive in Non smokers | Positive in Smokers | Positive in Ex smokers | Positive in Passive |
|---|---|---|---|---|
| Pollen | 3(1%) | 9(2.9%) | 7(2.3%) | 2(0.6%) |
| Weeds | 0 | 7(2.3%) | 4(1.3%) | 4(1.3%) |
| Fungi | 0 | 9(2.9%) | 7(2.3%) | 5(1.6%) |
| Insects | 1(0.3%) | 14(4.5%) | 9(2.9%) | 4(1.3%) |
| Animals | 0 | 8(2.6%) | 3(1%) | 1(0.3%) |
| Total | 4(1.3%) | 47(15.2%) | 30(9.7%) | 16(5.2%) |

**Table 4:** Pulmonary function test among the patients

| PFT | Non smokers | Smokers | Ex smokers | Passive | p-value |
|---|---|---|---|---|---|
| FVC % | 93.21 ± 13.2 | 84.52 ± 3.54 | 75.21 ±2.87 | 89.62 ± 5.13 | <0.001 |
| FEV1 % | 94.7 ±28.65 | 70.23 ± 17.23 | 51.35 ± 17.36 | 91.87 ± 27.87 | <0.001 |
| FEV1/FVC% | 99.2 ±29.94 | 85.23 ±1.7 | 66.23 ± 8.73 | 91.23 ± 19.47 | <0.001 |
| FEF25-75 % | 95.08 ± 23.12 | 55.82 ± 11.36 | 37.72 ± 10.83 | 77.23 ± 8.37 | <0.001 |
| FEF max % | 105.37 ± 22.87 | 76.34 ± 38.11 | 41.98 ±4.84 | 83.36 ± 17.26 | <0.001 |

only 66.23 ± 8.73 in the ex smokers, 85.23 ±1.7 in the smokers, and 91.23 ± 19.47 in the passive smokers (Table 4)

## 4. Discussion

There have been several studies which show that tobacco smokers have lower levels of Immunoglobulin G and A, but a higher level of Immunoglobulin E, predisposing them to respiratory tract infections such as allergic rhinitis, wheezing, atopic dermatitis etc.[6,12–16]

The serum IgE levels were higher in the smokers in our study compared to the non smokers. The ex smokers had a higher IgE level, but it was considerably lower than the non smokers. The passive smokers also had a significantly higher level of IgE than the non smokers. This study was corroborated by another study by Goel et al, who also observed an elevated IgE levels in the smokers compared to non smokers and past smokers.[17] These IgE levels were not associated with the amount of cigarette smoked. Yet another study by Vollmer et al supports these results.[18] The mean IgE levels in smokers was much higher than the non smokers and ex smokers in a study by Bahna et al.[19]

There was a greater levels of absolute eosinophil count in the present study in smokers than in the non smokers which was corroborated by a study by Schneider et al.[20]

There have been suggestions that the cigarette smoking increases the permeability of the mucosa, thereby granting easier access of the allergens into the subepithelial lymphoid tissues. This increased permeability is said to enhance atopy.[21]

There is evidence that smoking also hampers in the airflow to the lungs.[18,22] In the present study, we observed a marked reduction of the airflow in the smokers compared to the non smokers and the passive or ex smokers. In a study by Goel et al, there was no significant relation between the IgE levels and the FVC and FEV%.[17]

In the present study, there was a significant reaction to skin test for various allergens in smokers compared to the non smokers and the passive smokers. A similar reaction was observed in a study by Goel et al,[17] wherein also, the smokers showed a positive reaction to various allergens,

especially to Drosophila and cockroach. Jarvis et al showed a higher sensitivity of the smokers to the house dust mites than the non smokers.[23] A study by Kim et al reported that the current smoking status was an independent factor for the increased IgE levels especially to those specific to cockroach, which increased with the increase in number of cigarettes smoked per day.[24] In a meta analysis study, both active and passive smokers had lower pulmonary function and were associated with symptoms of asthma, including wheezing.[25] The FEV/FVC % was also lower in smokers than those who never smoked.[25]

## 5. Conclusion

Smoking is one of the most common modifiable risk factors to a number of diseases prevalent in man. It is associated with the increase in IgE levels, making the patient more sensitive to allergens Stopping smoking can lead to healthier patients, better lifestyle and longer life.

## 6. Source of funding

None.

## 7. Conflict of interest

None.

## 8. Acknowledgement

None.

## References

1. World Health Organization (WHO). Tobacco or Health: A Global Status Report. Geneva: World Health Organization; 1997 ;.
2. Glantz SA. Passive smoking and heart disease: mechanisms and risk. *JAMA*. 1995;273(13):1047–1053.
3. Lai HK. Secondhand smoke and respiratory symptoms among adolescent current smokers. *Pediatr*. 2009;124(5):1306–1310.
4. Wang C. Effects of in utero and childhood tobacco smoke exposure and 2-adrenergic receptor geno-type on childhood asthma and wheezing. *Pediatr*. 2008;122(1):107–114.
5. Gadermaier E, Levin M, Flicker S, Ohlin M. The human IgE repertoire. *Int Arch Allergy Immunol*. 2014;163:77–91.
6. Jensen EJ, Pedersen B, Schmidt E, Dahl R. Serum IgE in nonatopic smokers, nonsmokers, and recent exsmokers: relation to lung function, airway symptoms, and atopic predisposition. *J Allergy Clin Immunol*. 1992;90(2):224–233.
7. Sherrill DL, Halonen M, Burrows B. Relationships between total serum IgE, atopy, and smoking: a twenty-year follow-up analysis. *J Allergy Clin Immunol*. 1994;94(6):954–962.
8. Larche M, Akdis CA, Valenta R. Immunological mechanisms of allergen-specific immunotherapy. *Nat Rev Immunol*. 2006;6:761–771.
9. Taye B, Enquselassie F, Tsegaye A, Amberbir A, Medhin G, Fogarty A. Association between infection with Helicobacter pylori and atopy in young Ethiopian children: A longitudinal study. *Clin Exp Allergy*. 2017.
10. Chang CM, Corey CG, Rostron BL, Apelberg BJ. Systematic review of cigar smoking and all cause and smoking related mortality. *BMC Public Health*. 2015;15:390.
11. Bousquet PJ, Cropet C, Klossek JM, Allaf B, Neukirch F, et al. Effect of smoking on symptoms of allergic rhinitis. *Ann Allergy Asthma Immunol*. 2009;103:195–200.
12. Gulsvik A, Fagerhoi MK. Smoking and immunoglobulin levels. *Lancet*. 1979;1:449.
13. Mili F, Flanders WD, Boring JR, Annest JL, Destefano F. The association of race, cigarette smoking, and smoking cessation to measures of the immune system in middle-aged men. *Clin Immunol Immunopathol*. 1991;59:187–200.
14. Wolfe WH, Miner JC, Michalek JE. Immunological parameters in current and former US Air Force personnel. *Vaccine*. 1993;11:545–547.
15. Mcmillan A, Douglas JP, Archbold GP, Mccrum EE, Evans AE. Effect of low to moderate levels of smoking and alcohol consumption on serum immunoglobulin concentrations. *J Clin Pathol*. 1997;50:819–822.
16. Burrows B, Halonen M, Berbee RA, Lebowitz MD. The relationship of serum immunoglobulin E to cigarette smoking. *Am Rev Respir Dis*. 1981;124:523–525.
17. Goel N, Singh BP, Arora N, Kumar R. Effect o smoking on atopic predisposition and sensitization to allergens. *Ind J Chest Dis Allied Sci*. 2008;50:329–362.
18. Vollmer WM, Buist AS, Johnson LR, Mccamant LE, Halonen M. Relationship between serum IgE and cross sectional and longitudinal FEV1 in two cohort studies. *Chest*. 1986;90:416–423.
19. Bahna SL, Heiner DC, Myhre BA. Immunoglobulin E pattern in cigarette smokers. *Allergy*. 1983;38(1):57–64.
20. Schneider M, Hilgers RH, Allergy SJ. total IgE and eosinophils in East and West: serious effects of different degrees of helminthiasis and smoking. *Eur J Med Res*. 2002;21:763–771.
21. Chhabra SK, Rajpal S, Gupta R. Patterns of smoking in Delhi and comparison of chronic respiratory morbidity among bidi and cigarrete smokers. *Indian J Chest Dis Allied Sci*. 2001;43:19–26.
22. Shadick NA, Sparrow D, O'Connor GT, DeMolles D, Weiss ST. Relationship of serum IgE concentration to level and rate of decline of pulmonary function: the normative ageing study. *Thorax*. 1996;51:787–792.
23. Jarvis D, Chinn S, Luczynska C, Burney P. The association of smoking with sensitization to common environmental allergens: results from European Community Respiratory Health Survey. *J Allergy Clin Immunol*. 1999;104:934–940.
24. Kim YS, Kim HY, Ahn HS. The Association between Tobacco Smoke and Serum Immunoglobulin E Levels in Korean Adults. *Intern Med*. 2017;56:2571–2577.
25. Yoo S, Kim HB, Lee SY. Effect of active smoking on asthma symptoms, pulmonary function, and BHR in adolescents. *Pediatr Pulmonol*. 2009;44:954–961.

## Author biography

**Swetha Madas** Associate Professor

**C Malliakarjuna Reddy** Associate Professor

**M Hima Bindu** Associate Professor

**Cite this article:** Madas S, Malliakarjuna Reddy C, Bindu MH. Assessment of serum total IGE levels in smokers, non smokers and ex smokers and its relation to lung function, airway symptoms and atopic predisposition. *Indian J Immunol Respir Med* 2019;4(4):210-213.