Original Article

IMPACT OF SMOKING ON ADULTS LUNG AGE AND VENTILATORY FUNCTION

Omar Farouk Helal

Assistant Professor, Physical Therapy Department, Faculty of Applied Medical Sciences, Umm Al Qura University. KSA.

ABSTRACT

Background: Although a large body of evidence exists on the effect of smoking on lung age and pulmonary function, much less attention has been dedicated to using these effects as an effective strategy in smoking cessation.

Objective: The present study was carried out to investigate the impact of smoking on lung age and ventilatory function in adult Saudi in order to use these effects in a future strategy for smoking cessation.

Methods: Eighty one smoker students with their mean age 23.88 ± 2.7 years were enrolled in this study. Every student performed a ventilatory function tests in order to measure lung age, forced vital capacity (FVC), forced expiratory volume at the end of the first second (FEV1), FEV1/FVC ratio and peak expiratory flow rate PEFR.

Results: The result showed significant deterioration in the mean value of FEV1, PEFR and the estimated lung age and a non-significant difference in the mean values of FVC.

Conclusion: Smoking has a significant effect on ventilaroty function and deteriorating estimated lung age.

KEYWORDS: Smoking, Lung Age, Ventilatory function.

Address for correspondence: Dr. Omar Farouk Helal, PT, Assistant Professor, Physical Therapy Department, Faculty of Applied Medical Sciences, Umm Al Qura University. KSA. **Email:** dr.mon5@hotmail.com

INTRODUCTION

Cigarette smoking is a major health problem that is responsible for a wide range of preventable health problems throughout the world. It is estimated that over the next 20 to 30 years, cigarette smoking will result in 10 million deaths annually on a worldwide basis, of which 70% will occur in developing countries. [1]

The World health organization reported that tobacco smoking killed 100 million people worldwide in the 20th century and warned that it could kill one billion people around the world in the 21st century. [2]

Adolescent and teenage smoking has been studied widely, and it has been found in developing countries that nearly one-half of school students who have reached the age of 18 have already established the habit of smoking with some degree of regularity, and it is a rather unrealistic hope on the part of adults to expect that children will abstain until reaching the adult approved age of decision. [3]

Cigarette smoke is a complex mixture of more than 4000 chemicals, many of which exert toxic effects on cellular function and consider it as the single most important risk factor for developing chronic obstructive pulmonary disease (COPD). These obstructions in airways invariably affect the parameters of pulmonary function, e.g. forced vital capacity (FVC), Forced Expiratory Volume in the First Second (FEV1) and Peak Expiratory Flow Rate. [4]

Studies of pulmonary function among asymptomatic smokers have demonstrated that, as a group, they show significant impairment of almost all parameters of lung function, especially those indicating airway obstructions, as compared to matched groups of nonsmokers. [5]

Acute cigarette smoke has a suppressive effect on the number of eosinophils and several inflammatory cytokines, possibly due to the anti-inflammatory effect of carbon monoxide. [6]

In 2005, Selim and Al-Rushood [7] stated that according to recent year for which official data are available, Saudi Arabia imported 36.5 million kg of tobacco, costing an estimated 979 million Saudi riyals (260.64 million US dollars) for a population of about 15 million people. Lung cancer, a smoking-related disease, is a leading cause of cancer deaths among Saudi males, suggesting that cigarette smoking is becoming an important public health problem among men in Saudi.

Smoking cessation is the only effective treatment, slowing down the accelerated decline in ventilatory function. One strategy to motivate smokers to quit is to assess whether they have abnormal lung function, which is a strong independent risk factor for chronic obstructive pulmonary disease (COPD), lung cancer, cardiovascular disease, stroke, and all-cause mortality. [8]

One way to discuss abnormal lung function results with 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. [9]

Recently, Parkes, et al., 2010 and Kotz et al., 2011 [10-11] demonstrated that using lung age to communicate lung function to smokers in the primary care setting enhances smoking abstinence at one year, but these authors were not able to demonstrate any negative impact of normal lung age.

Ventilatory function tests can provide important clinical information, yet they are vastly underused. They are designed to provide objective, quantifiable measures of lung function which are used to evaluate and monitor disease that affect heart and lung, including airflow obstruction, restrictive disorders, exercise limitation and bronchial hyperactivity and the information obtained from these tests enable the practitioner to recognize impairment , follow the progress of disease, determine patient s' responses to therapy, and to monitor the effect of environmental, occupational and therapeutic exposure. [12]

Smoking is a community health problem which unfortunately has become part of our youth culture. The success of the community interventions for smoking cessation and changing smoking attitudes is to know the priority of smoking as a public health problem and to make efforts to limit and eradicate smoking.

So the purpose of our study was to clarify the adverse effects of smoking on lung age and ventilatory function in adult Saudi students in order to utilize it as an encouragement tool for smoking cessation.

MATERIALS AND METHODS

Subjects:

One Hundred Healthy, male, adult (aged 18 – 28 years) light smokers for more than two years were eligible for study entry. Subjects were excluded if they had diabetes mellitus, blood pressure >160/95mmHg, or if they were on medication, had any history of participating in any regular exercise training for at least 4 months prior to the study, or were participating in another clinical trial.

Subjects were recruited via friends across the University and signed the standard informed consent form to participate in the study following the consent of the Faculty Ethics Committee.

We actually start the study with 100 male healthy subjects. All participants were assessed using a structured pre tested respiratory questionnaire to obtain their clinical and anthropometric data. Unfortunately 19 subjects dropped out and excluded from the study.

Study materials:

1- Standard weight and height scale (weight scale, made in Germany) was used to measure the weight and height of each participant and

consequently the body mass index (BMI = weight "kg"/height2"m2") was calculated. [13]

2- Computerized pulmonary function testing (PFA) apparatus (Carello Quark Cosmed Srl pulmonary function test-Italy) was used to evaluate Ventilatory function (FVC, FEV1, FEV1/FVC, PEFR) and lung age among the participated students (Figure 1).

Fig. 1: Cosmed PFT Apparatus.

Procedure:

Cosmed PFT apparatus preparation:

Calibration of the Cosmed PFT apparatus was done daily before the measurement takes place, then subject physical data [name, age (years), height (cm), weight (kg)] was entered to allow the Cosmed PFT apparatus flow screen to calculate the predicted values.

Preparation of the subject:

The subjects to be tested were lightly clothed and instructed not to eat before the test by 2 hours. The position of the subjects at the time of the test was standing position, then learn the subject what is meant by the various commands "breathe naturally", "take a deep breath" and "blow all the air out" was necessary explained before starting the test. The test was explained to each patient individually in simple terms and demonstrated for them before applying its steps.

At the beginning, subject's name, age, weight, height, race and sex were recorded in the computerized Cosmed PFT apparatus. The patient connected to flow sensor, then nose clip was placed around the subject's nose to prevent air from passing through the nose during application, then he put a new mouthpiece into his mouth; it was held by the subject teeth and enclosed firmly by the lips to prevent air leakage.

The patient then breathes few times normally before the test done.

The maneuver was explained by the instructor to the participants prior to testing (breathe normally for several cycles, and then perform a maximal inspiration, followed by a maximum forced exhalation).

Subjects were asked to breathe at first normally out of the mouthpiece. The instructor pressing the start-button at the moment of the subject is ready before start test. Then the subject was asked to take a slow and deep inspiration to fill his lungs completely with air and then expire (blow out) as much as he can into the mouthpiece, with nose clip is on. In order to complete the FVC testing maneuver and meet the acceptability and reproducibility criteria, subjects must expel air forcibly and completed in at least three attempts six seconds each (Figure 2. A and B).

Fig. 2: Pulmonary function testing.

(A)

(B)

The measured values show the subject performance of three successive trials on the operator screen, then the best one was selected. In this operating mode the device measures the FVC, FEV1, FEV1/ FVC, PEFR and lung age.

Statistical Analysis

Statistical analysis was conducted for this study using SPSS software version 16. All values were presented as a mean $\pm$ standard deviation (SD). Paired t-tests were used to assess any differences in spirometric data collected before and after the exercise and differences were considered significant at $p > 0.05$.

RESULTS AND TABLES

Eighty-one smoker students from were enrolled in this study. Their age ranged from 16 to 25 years with a mean of 23.88 ± 2.7 years the height ranged from 158.00 cm to 185.00 cm, with a mean of 170.75 ± 6.28 cm, and weight ranged from 46.00 to 94.00 kg, with a mean of 72.32 ± 11.53 kg and BMI ranged from 16.3 to 33.12 kg/m^2 with a mean of 24.82 ± 3.88 kg/m^2. Table (1).

Table 1: General characteristics of the participating students (mean $\pm$ SD).

Character	Mean	SD
Age (years)	21.53	1.6
Weight (Kg)	72.32	11.53
Height (cm)	170.75	6.28
BMI (Kg/cm^2)	24.82	3.88
Smoking duration (years)	4.35	2.01
Cigarette number (cig/day)	12.8	5.73

Ventilatory function, including (FVC, FEV_1 and PEFR) and lung age were evaluated for all participants.

Paired t-test was applied to test the hypothesis that there were non-significant differences between the ventilatory function (FVC- FEV_1- FEV_1/ FVC -PEFR) and lung age values obtained from smoker students and those obtained from predicted measures for normal; nonsmoker students.

The results of the study showed the effect of smoking on ventilatory function and lung age as appeared in the differences between the measured values for smokers and predicted values for matched normal subjects.

The result revealed that there was a significant decrease in the mean of FEV_1 values in smokers' students as compare with the predicted values of normal, healthy matched subjects, as F-value was (7.874) and p-value (0.006). Table (2) and Figure (3).

The results showed non-significant differences between the mean values of FVC in smokers' students as compared with the predicted values of normal, healthy matched subjects, as F-value was (0) and p-value (0.989). Table (2) and Figure (3).

Table 2: The Mean, SD and 't' value with statistical significance of the evaluated parameters of the students.

Parameters	Smokers Students Values	Predicted normal values	t-value	P-value
FVC	4.93 ± 0.65	4.93 ± 0.38	0	0.989**
FEV1	4.22 ± 0.29	4.44 ± 0.63	7.874	0.006*
PEFR	548.42 ± 127	575.86 ± 63.31	3.027	0.084**
Lung age	23.88 ± 2.7	21.51 ± 1.7	44.871	0.000*

Level of significance at $P < 0.05$. * = significant ** = non-significant

As shown in table (2) and figure (4) there was non-significant difference in mean value of PEFR (L/min) when comparing the measured values in smokers, students with the predicted values of normal, healthy matched subjects; F-value was (3.027) and p-value (0.084).

The results showed a significant difference between the mean values of the lung age values in smokers' students as compared to the actual age values, as F-value was (44.871) and p-value (0.00). Table (2) and Figure (5).
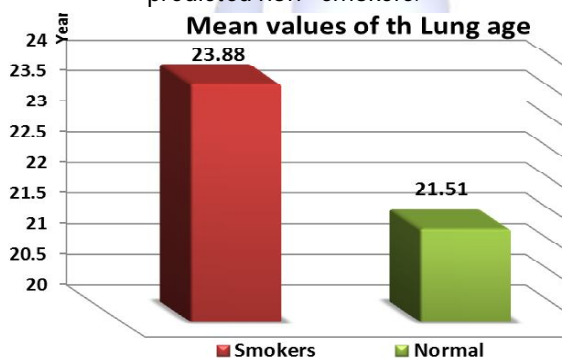
Fig. 3: FVC and FEV1 mean values for smokers and predicted non-smokers.
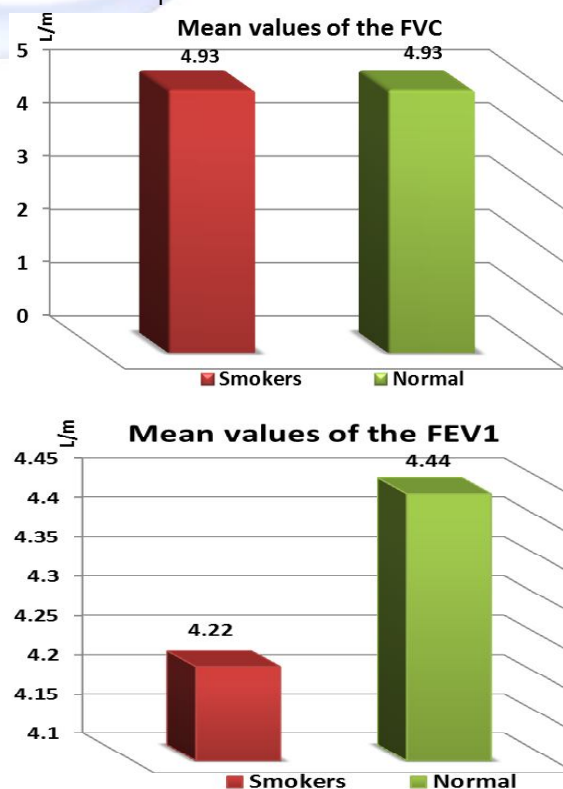
Fig. 4: PEFR mean values for smokers and predicted non-smokers.
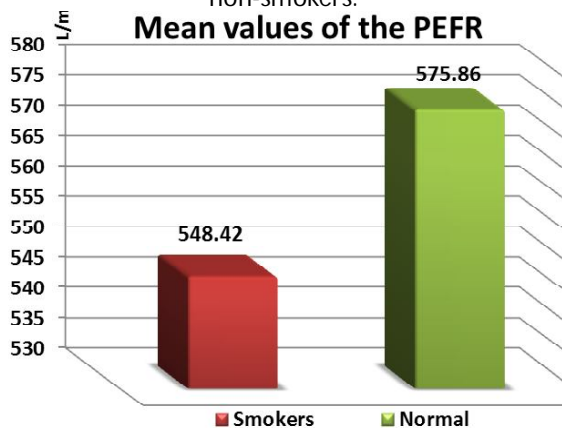
Fig. 5: Lung age mean values for smokers and predicted non –smokers.

DISCUSSION

This study was conducted to assess the impact of smoking on ventilatory function and lung age in sedentary smoking students. This study was conducted on 81 volunteer smokers who agreed to participate in the study, and was selected randomly from a University students.

Forced expiratory volume in 1 sec. (FEV1) and forced vital capacity (FVC) are measurements of air forced out of the lungs. These measurements are used to gauge the pulmonary function. [14]

Our study provides a conclusion about the difference between the ventilatory function and lung age of smoker students compared to non-smoker students. The study proves the association of smoking with deterioration of lung functions and lung aging.

Results of ventilatory function and lung age of smokers' students were compared with predicted values for normal matched non-smokers and showed significant difference in the FEV1, PEFR and lung age between the smoker students' values and predicted normal values. On the contrary; the study revealed non-significant difference in FVC between the smokers and non-smokers.

Cigarette smoking has been identified to be the most important determinant of ventilatory impairment [15]. Smoking impairs the growth of forced expiratory volume in one second (FEV1) in children[16] and cause an accelerated decline in FEV1, in adults. [15, 17]

The concept of "lung age" (the age of the average person who has an FEV1 equal to the individual) was developed as a way of making spirometry data easier to understand and also as a potential psychological tool to show smokers the apparent premature ageing of their lungs. [18]

Abnormal lung age is a clear message that the lungs are undergoing accelerated deterioration that would be slowed if the smoker stopped[19].

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 FEV1 for height, age and gender. [20]

The results of this study showed clearly that ventilatory function; specifically FEV1; significantly deteriorate in smokers students. Furthermore; smokers' lungs showed more advanced age than matched normal non-smokers. Walter et al., [21] found that Adult smokers experience faster longitudinal pulmonary function decline than nonsmokers and that this accelerated decline returns to the normal rate of aging-related decline following smoking cessation, even if the cessation is intermittent. [21]

The results of this study concerning ventilatory function were previously supported by a study by Jawed et al., [17] indicated the association of smoking with deterioration of lung functions as well as the number of cigarettes smoked/day.These results were further supported by a cross-sectional survey among the 20 to 40 years old smokers that showed association of cigarette smoking with deterioration in FEV1/FVC ratio and the onset of respiratory complaints which was dosed dependent. [22]

The significant differences between smokers' ventilatory function and non-smokers' predicted values were supported by a study showed that there are the significant difference in the mean spirometric values of FEV1/FVC of the smokers

and the non-smokers of age 18-30 years.[23]

Viegi et al.,[24] also showed that the prevalence of pathological pulmonary function tests is significantly higher in males and in subjects exposed to active and/or passive smoking. . The reduction of FVC may be an early marker of the morphological changes first occurring in the small airways of subjects exposed to smoke.

The decrease in FVC reflects a small airway narrowing, with gas trapping, determined by loss of elastic load or airway thickening whereas the decrease in FEV1 reflects smooth muscle contraction in the large airways. [25]

Furthermore, the results of deteriorated smoker's PEFR in this study were supported by a study by Willemse et al.,[26] that clearly found lower FVC, FEV1 and mid-expiratory flow rate (FEF25-75) in smokers as compared to non-smokers.[26]

Deteriorated ventilatory function observed in smokers can be further explained on the basis of the defensive lung reflex in response to smoking; resulting in narrowing of the airways and a consequent rise in airway resistance. [27]

Another explanation for abnormal smoker's ventilatory function is that the smoking of cigarettes by humans is believed to cause oxidative stress by several mechanisms, including direct damage by radical species and the inflammatory response induced by smoking. Oxidative stress was found to cause apoptosis and lung injury. [28,29]

Smoking induces an oxidative burden by disturbing the oxidant- antioxidant balance and leads to cellular damage in the lungs. Oxidative stress caused by cigarette smoking can result in the destruction of the alveolar wall, leading to airway enlargement. Moreover, increased oxidative stress can trigger proinflammatory cytokines, which are increased in the lungs of smokers. [30]

Substantial evidence suggests the accelerated decline of lung function in cigarette smokers' results from smoke-induced inflammatory processes. Inflammation begins with an increased number of macrophages in the first and second generation respiratory bronchioles. [21]

Airway epithelial cells (AEC) are important regulators of inflammation in the airway. They have a function in host defense and play a significant role in airway inflammation by releasing NO, a potentially important mediator of airway inflammation, as well as releasing other mediators and recruiting inflammatory cells. Cigarette smoke interferes with and inhibits the normal function of AEC by a variety of mechanisms. Some of these include decreases in the level of exhaled NO, enhanced release of pro-inflammatory cytokines, and inhibition of the airway repair process.[31]

Exposure to cigarette smoke activates an inflammatory cascade in the airway epithelium, resulting in the production of a number of potent cytokines and chemokines, with accompanying damage to the lung epithelium, increased permeability, and recruitment of macrophages and neutrophils to the airway. [32]

A positive association between pulmonary surfactant and airway diameter has been described; surfactant proteins were observed to inhibit pulmonary inflammation. Production of particular surfactant proteins is inhibited by tobacco smoking. [21]

CONCLUSION

The results of the present study clarify the deteriorating effects of smoking on lung functions and showed that the ventilatory function of smoker students are significantly lower than the predicted ventilatory function of age matched non-smoker subjects. Furthermore; smoker lung tends to be older than age matched non-smoker subject.

REFERENCES

1. Lee PN, Forey B, Fry JS. Revisiting the association between environmental tobacco smoke exposure and lung cancer. 2001; (10): 384-398.
2. WHO report: Tobacco could kill one billion by 2100. Science Daily, 2008 Aug; 24:71.
3. Horn D. Behavioral aspects of cigarette smoking. Journal of Chronic Diseases 1963;16: 383-39.
4. Nawafleh HA, Abo Zead S, Al-Maghaireh D F. Pulmonary function test: The value among smokers

and nonsmokers.Health science journal. 2012 ; 6: 703-713.

5. Robert V, Jay H. Smoking-Related Interstitial Lung Diseases. Clin Chest Med 2012; 33: 165–178.

6. Vaart HVD, Postma D S, Timens W, Hacken NHTT. Acute effects of cigarette smoke on inflammation and oxidative stress. 2013; 31: 713-721.

7. Selim M M and Al-Rushood R. M.. Statistical investigation of prevalence of smoking among rural primary and secondary school students in Aflaj area, Kingdom of Saudi Arabia. International Journal of Basic and Applied Science 2005; 11(2): 47-56.

8. Mannino, D, Aguayo, S, Petty, T, and Redd, S. Low lung function and incident lung cancer in the Unites States. Data from the First National Health and Nutrition Examination Survey Follow-up. Archives of Internal Medicine 2003; 163: 1475–1480.

9. Kaminsky DA, Marcy T, Dorwaldt A,Pinckney R, DeSarno M., Solomon L,Hughes JR. Motivating smokers in the hospital pulmonary function laboratory to quit smoking by use of the lung age concept. Nicotine and tobacco research 2011 ;13 : 1161–1166.

10. Parkes G, Greenhalgh T, Griffin M, and Dent R. (Effect on smoking quit rate of telling patients, their lung age: The step2quit randomized controlled trial. British Medical Journal 2008; 336: 598–600.

11. Kotz D, Wesseling G, Aveyard P, and Van Schayck O. Smoking cessation and development of respiratory health in smokers screened with normal spirometry. Respiratory Medi-cine 2010; 105, 243–249.

12. Hyatt R E, Scanlon P D, Nakamura M. Interpretation of Pulmonary Function Tests: A Practical Guide, 3rd Edition. 2009. Lippincott Williams and Wilkins.

13. Jones RL and Nzekwu MM. The effects of body mass index on lung volumes. Chest 2006; 130:827-833.

14. Saxena S and McBean D. An investigation into the effects of smoking on physical fitness parameters in adolescents. Br J Sports Med 2010; 44 (Suppl 1): i30-i36.

15. Peat JK, Woolcock AJ, Cullen K. Decline of lung function and development of chronic airflow limitation: a longitudinal study of non-smokers and smokers in Busselton, Western Australia. Thorax 1990;45:32-7.

16. Tager IB, Munoz A, Rosner B, Weiss ST, Carey V, Speizer FE. Effect of cigarette smoking on the pulmonary function of children and adolescents. Am Rev Respir Dis 1985; 131:752-759.

17. Jawed S, Ejaz S, Rehman R. Influence of smoking on lung functions in young adults. J Pak Med Assoc 2012; 62 (8) : 772- 775.

18. Morris JF, Temple W. Spirometric "lung age" estimation for motivating smoking cessation. Prev Med 1985; 14: 655-62.

19. Parkes G, Greenhalgh T, Griffin M, Dent R. Effect on smoking quit rate of telling patients, their lung age: the Step2quit randomized controlled trial. BMJ 2008 ; 336 (15): 598- 600.

20. Pride NB. Smoking cessation: effects on symptoms, spirometry and future trends in COPD. Thorax 2001; 56 (90002):7ii-10.

21. Walter R, Gottlieb D J, and O'Connor, G T."Environmental and genetic risk factors and gene-environment interactions in the pathogenesis of chronic obstructive lung disease", Environ Health Perspect 2000;108 (Suppl 4): 733–742.

22. Isabel U, Alberto C, María QJ, Nerea M, Xavier B, Jordi S. Smoking habit, respiratory symptoms and lung function in young adults. Eur J Public Health 2005; 15: 160-5.

23. Gold DR, Wang X, Wypij D, Speizer FE, Ware JH, Dockery DW. Effects of cigarette smoking on lung function in adolescent boys and girls. N Engl J Med 1996; 335: 931-7.

24. Viegi G, Pedreschi M, Pistelli F, et al. Prevalence of airway obstruction in a general population. Chest 2000; 117: 339-345S.

25. Corsico A, Milanese M, Baraldo S, et al. Small airway morphology and lung function in the transition from normality to chronic airway obstruction. J Appl Physiol 2003; 95:441–7.

26. Willemse BW, Hacken N. H., Rutgers B., Postma D.S, and Timens W. Association of current smoking with airway inflammation in chronic obstructive pulmonary disease and asymptomatic smokers .Respir. Res. 2005; 6(1): 38.

27. Sterling GM, "Mechanism of Bronchoconstriction caused by cigarette smoking". Brit.Med.J. 1967 ; 3: 275-277.

28. Halliwell B, Gutteridge JMC: Free Radicals in Biology and Medicine 4th edition. Oxford: Oxford University Press; 2007.

29. Banerjee S, Maity P, Mukherjee S, Sil A K, Panda K, Chattopadhyay D, and Chatterjee I B. Black tea prevents cigarette smoke-induced apoptosis and lung damage. J Inflamm. (Lond) 2007; 4: 3.

30. Devereux G. ABC of chronic obstructive pulmonary disease: Definition, epidemiology, and risk factors", BMJ 2006 ; 332:1142-1144.

31. Wei X.M, Kim H S, Kumar R K, Heywood GJ, Hunt JE, McNeil H, P, and Thomas PS. Effects of cigarette smoke on degranulation and NO production by mast cells and epithelial cells. Respir. Res 2005; 6(1):108-114.

32. Hellermann G R, Nagy S. B, Kong X, Lockey RF, and Mohapatra S. S. Mechanism of cigarette smoke condensate-induced acute inflammatory response in human bronchial epithelial cells", Respir. Res 2002; 3(1): 22-26.