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RESEARCH ARTICLE

STUDY OF LUNG FUNCTIONS IN TOBACCO CHEWERS

***Dr. Abhijit Ahuja, Dr. Abhay Uppe and Dr. Girija Nair**

Department of Pulmonary Medicine, D Y Patil University School of Medicine, Navi Mumbai, India

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ABSTRACT

Objective: To study pulmonary function indices of tobacco chewers and compare with those of nonusers by PFT & DLCO.
Materials & methods: 60 stable patients attending OPD of D.Y.Patil Hospital, India were recruited in the study, of which 30 were chronic tobacco chewers but non smokers & 30 served as control subjects without any history of tobacco consumption.
Results: In our study we had a total of 60 patients. 29/60 (48.3%) were females and 31/60 (51.7%) were males. The mean FEV1/FVC (% predicted value) for tobacco chewers was 79.8% as compared to mean FEV1 /FVC for non tobacco chewers being 83 %. The mean FEV1 (% Actual/Predicted) of tobacco chewers was 88.03 %. However for non chewers the mean FEV1 (% Actual/Predicted) was higher at 95.96 %. The MMEF 75-25 of tobacco chewers with that of non chewers and we observe that the mean MMEF 75-25 for tobacco chewers is 71.76 % while that for the non smokers is 85.43 %.
Conclusion: In this study, tobacco chewers had low pulmonary function indices which may be due to increased oxidative stress. Considering the large number of tobacco chewers in our country, further studies may be required to identify the effects of tobacco chewing not only locally in the oral cavity but also other systems of the body.

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INTRODUCTION

Tobacco use is a public health concern worldwide as well as in India. Tobacco consumption is mainly done in two forms: smoked tobacco and smokeless tobacco. The tobacco use without burning is referred to as smokeless tobacco (SLT) use. SLT use has increased rapidly throughout the world especially among adolescent boys and young men by considering it as a safe alternative of smoking. Because of vigorous efforts towards increasing awareness of the adverse effects of tobacco, smoking has declined and paradoxically the use of SLT has greatly increased. 'Khaini', tobacco with slaked lime, is one of the most widely used SLT in India. Like cigarettes, the most important constituent of 'khaini is tobacco. This study attempts to find out whether chewing tobacco causes any unfavorable effects to the lungs by using PFT & DLCO. In this study pulmonary function indices of tobacco chewers will be compared with those of nonusers as control subjects. The aim was to study the effects of chewing tobacco on pulmonary function by performing

- PFT by spirometry in tobacco chewers
- DLCO
- Comparing the results with controls (people not chewing tobacco)

MATERIALS AND METHODS

Study design: This is a hospital based prospective study.

Duration of study: 3 years

Sample size: A total of 60 patients were recruited into the study.

Sample selection: Sixty stable patients attending OPD of Dr. D.Y.Patil Hospital were recruited in the study, of which 30 were chronic tobacco chewers who did not smoke & 30 served as control subjects without any history of tobacco consumption.

Inclusion Criteria: All consenting patients were of age 18 years or greater, attending the outpatients clinic or indoor

****Corresponding author: Dr. Abhijit Ahuja,***
Department of Pulmonary Medicine, D Y Patil University School of Medicine, Navi Mumbai, India.

patients of Dr. D.Y. Patil Hospital with the habit of chewing tobacco only, for atleast 3 years.

1)Control & experimental subjects were from same socioeconomic status.

Exclusion Criteria

Exclusion criteria for the subjects included presence of any self reported acute illness, lung diseases like chronic obstructive pulmonary disease, heart failure, malignant, chronic liver or kidney failure, diabetes mellitus, history of heavy alcohol or recreational drug use and smoking habit for at least last three years & biomass (wood chulha) exposure in females. Patients recruited in the study were evaluated by spirometry – PFT & DLCO (Equipment-JAEGER- 694265)

Analysis of Data

All data collected was analyzed based on relevant statistics. The lung function were determined by Pulmonary Function Test & Diffusing Lung Capacity of Carbon Monoxide (DLCO).

RESULTS

In our study we had a total of 60 patients. 29/60 (48.3%) were females and 31/60 (51.7%) were males (Figure 1).

Table 1. Distribution According to Gender

Gender	Frequency	Percent
Male	31	51.7%
Female	29	48.3%
Total	60	100.0%

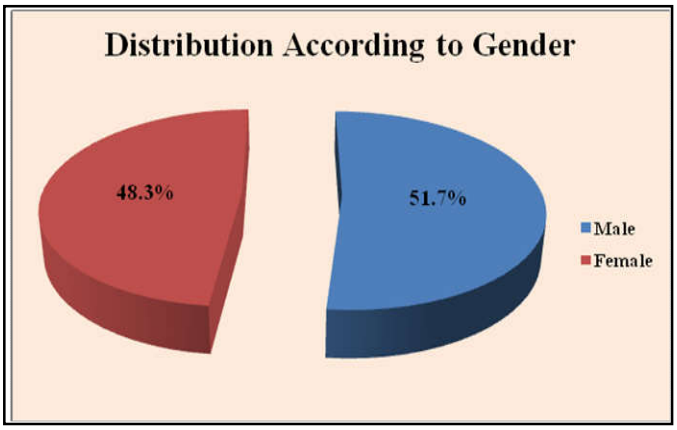


Figure 1.

The mean FEV_1/FVC (% predicted value) for tobacco chewers was 79.8% as compared to mean FEV_1 /FVC for non tobacco chewers being 83 % (Figure 2). The statistical difference (p value) between the mean FEV_1/FVC of tobacco chewers and non chewers is 0.026 (Table 5) which is significant. On comparing the MMEF 75-25 of tobacco chewers with that of non chewers and we observe that the mean MMEF 75-25 for tobacco chewers is 71.76 % while that for the non smokers is 85.43 %, the statistical difference (p value) being 0.024 (Table 3) which is significant.

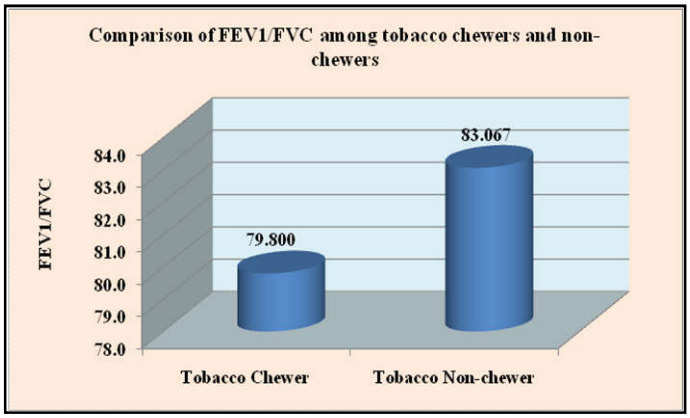


Figure 2.

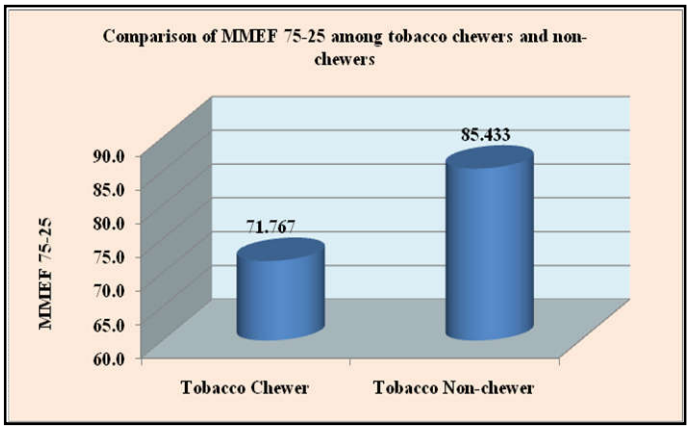


Figure 3.

DISCUSSION

In India it has been estimated that roughly one third of women and two-thirds of men use tobacco in one form or another. In prevalence surveys in eight rural areas of India, smokeless tobacco use was upto 53% among men and upto 49% among women. (WHO 1997) About 35–40% of tobacco consumption in India is in smokeless forms. The various forms are chewed, sucked or applied to teeth and gums.

Table 2. Comparison of FEV1/FVC among tobacco chewers and non-chewers

		N	Mean	SD	SEM	t-stat	df	p-value
FEV1/FVC	Tobacco Chewer	30	79.800	6.810	1.243	2.277	58	0.026*
	Tobacco Non-chewer	30	83.067	3.921	0.716			

Table 3. Comparison of MMEF 75-25 among tobacco chewers and non-chewers

		N	Mean	SD	SEM	t-stat	df	p-value
MMEF 75-25	Tobacco Chewer	30	71.767	29.604	5.405	2.318	58	0.024
	Tobacco Non-chewer	30	85.433	12.905	2.356			

Generally sun- or air cured smokeless tobacco can be used by itself in unprocessed, processed or manufactured form. It can be used with lime, with areca nut or in a betel quid (*pan*). The use of unprocessed tobacco, the cheapest form, varies in different parts of India. (Gupta and Ray, 2003)

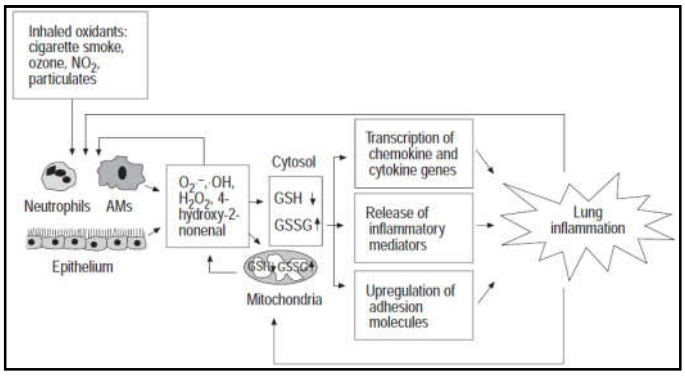
This study was done to identify if tobacco chewing leads to any change in lung functions.

1. Our findings in this study showed that there was a decrease in FEV1/FVC ratio in tobacco chewers as compared to those didn't chew tobacco.
2. FEV1 indices were also found to be decreased in tobacco chewers as compared to non chewers.
3. $MMEF_{75-25}$ volumes were also decreased in tobacco chewers as against non chewers.
4. However, the difference in FVC & $DLCO_{SB}$ was not found to be statistically significant.

These changes may be attributed to imbalance between formation of reactive oxygen species and antioxidants which may contribute to inflammation & chronic airway limitation. Rahman and MacNee proposed that inflammation is an important protective response to cellular/tissue injury. The purpose of this process is to destroy and remove the injurious agent and injured tissues, thereby promoting tissue repair. When this crucial and normally beneficial response occurs in an uncontrolled manner, the result is excessive cellular/tissue damage that results in chronic inflammation and destruction of normal tissue. Reactive oxygen species (ROS), such as the superoxide anion liberated by phagocytes recruited to sites of inflammation, are proposed to be a major cause of the cell and tissue damage, including apoptosis, associated with many chronic inflammatory diseases. Lung cells, in particular alveolar epithelial type II cells, are susceptible to the injurious effects of oxidants. It has been shown that lung cells release inflammatory mediators and cytokines/ chemokines such as tumour necrosis factor-a (TNF-a), interleukin (IL)-1 and IL-8 in response to oxidative/nitrosative stress. The release of cytokines/chemokines induces neutrophil recruitment and the activation of key transcription factors such as nuclear factor-kB (NF-kB) and activator protein-1 (AP-1), thereby augmenting the inflammatory response and tissue damage. As a result, the acute and chronic alveolar and/or bronchial inflammatory response is a fundamental process involved in the pathogenesis of many lung diseases such as asthma, chronic obstructive pulmonary disease (COPD), acute respiratory distress syndrome (ARDS), idiopathic pulmonary fibrosis (IPF) and cystic fibrosis (CF).

A study demonstrated an increase in total erythrocyte count, total leukocyte count, PCV, Hb levels, neutrophil percentage and decrease in the percentage of monocytes and lymphocytes and biochemical parameters (eg increase in blood cholesterol levels, blood glucose levels) which was seen in gutka consumers. They concluded that these negative effects of tobacco use may increase the possibility of developing other health related complications like respiratory diseases, cardiovascular diseases, diabetes, etc. (Roan Mukherjee *et al*., 2013) Pramod Kumar Avti *et al*. evaluated the effects of long term use of aqueous extract of gutkha (a form of smokeless tobacco) on the antioxidant defense status and histopathological changes in liver, lung, and kidney of male Wistar rats. Histopathological findings suggested that administration of aqueous extract of smokeless tobacco (AEST) at the high dose for 2 weeks or at the low dose for 32 weeks could cause mild to moderate inflammation in liver and lungs. They concluded that a decrease in the antioxidant defense system and long-term inflammation caused by smokeless tobacco may be risk factors for gutkha-induced pathogenesis. In the lungs, it was observed that the high dose of AEST for 2 weeks caused a mild degree of interstitial inflammation, whereas long-term administration of the low dose of AEST for 32 weeks caused a moderate degree of interstitial inflammation. (Pramod Kumar Avti *et al*., 2006) Oxidative injury, as a result of exposure to oxidants from diverse sources including endogenous oxidants, may at least in part be related to reduced FEV1 and, therefore, contribute to airflow obstruction. (Macnee and Rahman, 1999)

Rahman and MacNee evaluated oxidative stress and regulation of glutathione in lung inflammation. Reactive oxygen species (ROS) and reactive nitrogen species (RNS) are generated by several inflammatory and immune and various structural cells of the airways. An imbalance of oxidant/antioxidant in favour of oxidants contributes to the pathogenesis of several inflammatory lung diseases. The presence of oxidative stress in the airspaces and the blood initiates a number of early events during pulmonary inflammation. Inflammatory cells are sequestered into the pulmonary microcirculation and recruited to the airspaces as a result of the generation of mediators such as IL-8. Once recruited, inflammatory cells become activated and generate ROS in response to a sufficient level of a secretagogue stimulus (Rahman and MacNee, 2000)



Mechanisms of oxidant-mediated lung inflammation:. The inflammatory response is mediated by oxidants which are inhaled and/or released by the activated neutrophils, alveolar macrophages (AMs) and epithelial cells leading to depletion of the antioxidant reduced glutathione (GSH). Activation of transcription of the pro-inflammatory cytokine and chemokine genes, upregulation of adhesion molecules and increased release of pro-inflammatory mediators are involved in the inflammatory responses. GSSG: oxidized glutathione: decrease; increase. Cigarette smokers have higher death rates for chronic bronchitis and emphysema; they also have a higher prevalence of lung-function abnormalities, respiratory symptoms, and all forms of chronic obstructive airway disease. Tobacco smoke has two phases – gas phase and tar phase. Gas

phase has ~10^{15} reactive species particularly nitric oxide. The tar phase also has an equally abundant number of reactive oxygen and nitrogen species (ROS, RNS), including phenols and quinone. This results in greater pro-inflammatory activity resulting in emphysema. (MacNee *et al*., 1989) Overall, tobacco smoking accounts for a large percentage of risk of developing COPD. So we conducted our study to identify whether tobacco chewing, too could affect lung function and cause obstructive airway disease.

Conclusion

Oxidant injury appears to underlie a number of inflammatory, obstructive, and fibrotic lung disorders (Heffner and Repine, 1989; Crystal, 1991). Since the lung surface is exposed constantly to oxidants from ambient air, the respiratory tract lining fluids act as an interface between the environment and toxic exposure (e.g., tobacco) on one side and respiratory tract epithelial cells on the other side (Cross *et al*., 1994). Additional oxidative burden arrives from endogenous or ingested compounds with oxidant activity. There is a potential role of oxidative stress in airway obstruction. Inactivation of antiproteases, inflammation, infection, direct cell damage, and disturbances in the antioxidant defense are primary mechanisms that appear to be involved in tobacco chewers (Li *et al*., 1996; Pritchard *et al*., 1996) Oxidative stress can cause inflammation of respiratory epithelial cells and morphologic changes that may lead to airway obstruction and ultimately alter pulmonary function (Bast *et al*., 1991; Cantin and Crystal, 1985). Smokeless tobacco produces oxidative stress resulting from imbalance between formation of reactive oxygen species and antioxidants, contribute chronic airway limitation (Schunemann *et al*., 2001). Thus in my study, tobacco chewers have low pulmonary function indices which may be due to increased oxidative stress. Considering the large number of tobacco chewers in our country, further studies may be required to identify the effects of tobacco chewing not only locally in the oral cavity but also other systems of the body.

REFERENCES

Bast A, Haenen GRMM, Doelman CJA. 1991. Oxidants and antioxidants: state of the art. *Am J Med*., 91(suppl):2S-13S.

Cantin A, Crystal RG. 1985. Oxidants, antioxidants, and the pathogenesis of emphysema. *Eur J Respir Dis*., 66:7-17.

Cross CE, van der Vliet A, O'Neill CA, *et al*. 1994. Oxidants, antioxidants, and respiratory tract lining fluids. *Environ Health Perspect*., 102:184-91.

Crystal RG. 1991. Oxidants and respiratory tract epithelial injury: pathogenesis and strategies for therapeutic intervention. *Am J Med*., 91(suppl):39S-44S.

Gupta PC. and Ray CS. 2003. Smokeless tobacco and health in India and South Asia. *Respirology*, 8:419e31.

Heffner JE. and Repine JE. 1989. Pulmonary strategies of antioxidant defense. *Am Rev Respir Dis*., 140:531-54.

Li XY, Gilmour PS, Donaldson K, *et al*. 1996. Free radical activity and pro-inflammatory effects of particulate air pollution (PM10) in vivo and in vitro. *Thorax*, 51:1216-22.

Macnee W, Rahman I. 1999. Oxidants and antioxidants as therapeutic targets in chronic obstructive pulmonary disease. *Am J Respir Crit Care Med*. 160(suppl):S58-65

MacNee W, Wiggs BB, Berzberg AS, Hogg JC. 1989. The effect of cigarette smoking on neutrophil kinetics in human lungs. *N Engl J Med*., 321: 924±928.

Pramod Kumar Avti *et al*. 2006. Smokeless Tobacco Impairs the Antioxidant Defense in Liver, Lung, and Kidney of Rats, Toxicological Sciences 89(2), 547–553.

Pritchard RJ, Ghio AJ, Lehmann JR, *et al*. 1996. Oxidant generation and lung injury after particulate air pollutant exposure increase with the concentrations of associated metals. *Inhal Toxicol*., 8:457-77.

Rahman I., W. 2000. MacNee Oxidative stress and regulation of glutathione in lung inflammation. *Eur Respir J*., 16: 534±554

Roan Mukherjee *et al*. 2013. Assessment of the effects of smoking and consuming gutka (Smokeless Tobacco) On Selected Hematological And Biochemical Parameters: A Study On Healthy Adult Males Of Hazaribag, Jharkhand, *IJPCBS*, 3(4), 1172-1178.

Schunemann J, Freudenheim JL and Grant BJB. 2001. Epidemiologic evidence linking antioxidant vitamins to pulmonary function and airway obstruction. *Epidemiol Rev.,* 23: 248–261

World Health Organization. 1997. Tobacco or Health, a Global Status Report. WHO, Geneva.

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