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Relationship between Serum Alpha 1 Antitrypsin, Cotinine and Anthropometric Markers in Cigarette Smoked Subjects

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KEYWORDS

Cigarette Smokers, Serum Cotinine, A1AT & Anthropometric Markers

ABSTRACT:

Introduction: - In the United States (US) & other countries cigarette smoking (CS) continues to be the more preventable cause of disease and death. To both tobacco use and exposure to environmental tobacco smoke (ETS), cotinine is widely applied as a marker, because it has a longer half-life (average, 18 to 20 hours) than nicotine (average, 2 to 3 hours). Alpha1-proteinase inhibitor or SERPINA1 are other used words for Alpha1-Antitrypsin (A1AT), and also the SERPIN (an acronym for serine proteinase inhibitor) family of protease inhibitors, prototypical member. Age, Height, Weight, Body Mass Index, Waist Circumference and Waist-hip ratio are simple and valid anthropometric measures for the assessment of risk of obesity & other systemic diseases in smokers The objectives of the present study were to measure the levels of serum alpha 1 antitrypsin, cotinine in cigarette smokers & to study the association between these biochemical markers with anthropometric markers and the duration and number of cigarette smoked.

Materials & Methods:- The present study was carried out in the Department of Biochemistry, Santosh Medical College, Ghaziabad. Prior to estimation anthropometric markers (weight, height, BMI, waist circumference, hip circumference & waist hip ratio) were done in all subjects followed by serum A1AT (alpha 1 antitrypsin) by ELISA (Elabscience, Catalog No: E-EL-H0109), serum cotinine by HPLC (high pressure liquid chromatography).

Results:- The mean serum cotinine level was significantly raised in cigarette smokers as compared to non-smokers whereas mean serum A1AT level was significantly decreased in cigarette smokers as compared to non-smokers. This difference was found to be statistically significant ($p< 0.05$).

Conclusion:- Based on our findings and the other data in the study, we speculate that these biomarkers to the detection of smokers might be useful with a high risk of pulmonary & cardiovascular diseases developed by smoke induced and will help to clinicians to formulate novel treating protocol & follow up for their patients.

Introduction

In most of the world cigarettes are the most common form of tobacco used and each year 443,000 deaths occur in the United States (US). Due to rise of tobacco industry and population growth cigarette smoking (CS) and use of other tobacco products is increasing in the developing world (1).

Tobacco smoking is the inhalation of smoke from burned dried or cured leaves of the tobacco plant, most often in the form of cigarette (2). Different kind of cigarettes (flavoured, hand-rolled, manufactured, filtered and un-filtered), pipes and cigars included as smoked forms of tobacco. The main form of tobacco smoked globally is cigarette smoking, particularly manufactured cigarettes, in some developed & developing countries other forms of smoked tobacco are predominant (3).

In developed countries cigarette smoking, hereafter referred to as “smoking,” is the largest single risk factor for premature death. In the United States approximately one fifth of the deaths are attributable to smoking and 28% of the smoking-attributable deaths involve lung

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cancer, 37% involve vascular disease and 26% involve other respiratory diseases. Approximately 80% of adult smokers initiate their tobacco use before 18 years of age as per WHO estimates. Therefore, the fact that many adult smokers makes smoking a significant public health problem by initiating their smoking habit as in adolescents age (4).

Cigarette smoke is separated into gaseous phase and particulate (tar) phase. a material that is trapped when the smoke stream is passed through the Cambridge glass-fiber filter that retains 99.9% of all particulate material with a size >0.1 μm is defined the tar or particulate phase. The particulate phase of cigarette smoke contains the condensable part of the gas phase. In the particulate phase aldehydes, ketones, organic acid and alcohol are found (5).

Body Mass Index (BMI) and waist-to-hip circumference ratio (WHR) are the simple measures & widely used anthropometric markers in clinical practice. The most widely used method to define thinness and fatness is BMI, a ratio of weight in kilograms divided by height in meters squared (kg/m^2). It has been correlated to morbidity and mortality risk in various populations (Willett WC *et al*., 1999).

It is also believed that combined use of BMI & WHR parameters of generalized and abdominal obesity may be better in identifying people at risk of cardiovascular disease (CVD) than either of them alone (Ardern CI *et al*., 2003, Meisinger C *et al*., 2006), because they correlate well with each other. World Health Organization include 18.5-24.9 kg/m^2 for normal, 25.0-29.9 for overweight and >30 kg/m^2 for obesity recommended currently cut-offs of BMI (World Health Organization, 1997). International Diabetes Federation (IDF) Criteria for Central Adiposity waist circumference male ≥ 90 cm and female ≥ 80 cm South Asian.

The nicotine absorbed is metabolized to cotinine an average of 70% to 80%. In adult smokers, a nicotine intake of approximately 1 mg can be estimated from a blood cotinine level of 71 nmol/L (12.5 ng/mL) using a conversion factor of 0.08 mg/24 h per nanogram per milliliter under steady-state conditions. The main biomarker used to distinguish tobacco users from people who do not use tobacco is cotinine, which reflects the extent of exposure, not how the exposure was derived (6).

Alpha1-Antitrypsin (A1AT), also referred to as alpha1-proteinase inhibitor or SERPINA1, and is the prototypical member of the SERPIN (an acronym for serine proteinase inhibitor) family of protease inhibitors. The term SERPIN was introduced as by Carrell and Travis in 1985 to describe a superfamily of serine protease inhibitors of mammalian plasma.

A1-Antitrypsin (a1-AT) is synthesized mainly by hepatocytes, a 52-kDa glycoprotein. It protects the alveolar matrix from destruction by neutrophil elastase (NE), a serine protease capable of destroying most of the structural components of the alveolar wall in the lung parenchyma its primary function (7). Accumulation of polymorphonuclear leukocytes and macrophages in the lungs caused by the smoking. A1-AT is abundant in human plasma with concentrations in the 20–53 mM range in addition to its presence in the lung. Due to mutations of the a1-AT gene, the anti-NE protection on the alveolar surface is inadequate, resulting in unopposed proteolytic activity, eventually leading to lung destruction and the development of emphysema by the third or fourth decade of life, when plasma levels of a1-AT are below a protective threshold of approximately 11 mM (8).

Although many studies have been conducted on biochemical markers in cigarette smokers, levels of alpha 1 antitrypsin & cotinine were not well documented. Their involvement & association in cigarette smokers is still not known & the results obtained by other studies are contradictory and have not been extensively studied. These markers have also been implicated in a several lung diseases, including lung, cancer and emphysema etc. Therefore, the objectives of the present study were to measure the levels of serum alpha 1 antitrypsin, cotinine in cigarette smokers & to study the association between these biochemical markers with anthropometric markers and the duration and number of cigarette smoked.

Materials & Methods

The present study was carried out in the Department of Biochemistry, Santosh Medical College, Ghaziabad. Institutional ethical clearance was taken prior to the study (F. No SU/2018/528 {2}.

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Inclusion criteria:-The age group of 18-60 years about 284 healthy cigarette smokers (without any systemic diseases) compared with age & sex matched 284 controls (non-smokers) were included in the study.

Exclusion criteria:- Person with habit of tobacco chewing along with smoking and taking other forms of smoke (bidi, hookah, cigar etc) & patients of tuberculosis, pulmonary disorders, coronary artery diseases, diabetes mellitus, renal failure, chronic liver diseases, thyroid dysfunction, anaemia, malnourished individuals, were excluded from the study.

According to prevalence, used in previous study (9) sample size is calculated

$$n = \frac{Z^2 \times p \times q}{d^2}$$

Where n is the sample size, Z is 1.96 (5% level of significance), p is prevalence, q is 1-p and d is 0.05 (95% of c.f.). According to this formula sample size was 284 for cigarette smokers.

Cigarette smokers comprising of number of cigarettes per day and duration of cigarette smoking was recorded on participant proforma after taking detailed history. Based on number of cigarettes per day and duration of cigarette smoking subjects were classified into different groups i.e. smoking 1-15 cigarette/day <5 years are mild smokers in group I , 15-20 cigarette/day <5 years in group II, 15-20 cigarette/day 5-10 years moderate, and 15-20 cigarette/day >10 years are heavy smokers (10). Out of total 284 cigarette smokers, 129 were in group I, 42 were in group II which were in mild group, 36 were in moderate & 77 were in heavy group smokers.

All aseptic precautions were taken; with a disposable syringe about five mL of blood was drawn by veinpuncture from a peripheral vein. For the retraction of clot collected blood in clean dry glass tubes was allowed to stand for 30 minutes at room temperature. Then it was centrifuged at 3000 r.p.m. for ten minutes to obtained the serum. The serum was stored at 4°C in the refrigerator for analysis.

Prior to estimation anthropometric markers (weight, height, BMI, waist circumference, hip circumference & waist hip ratio) were done in all subjects followed by serum A1AT (alpha 1 antitrypsin) by ELISA (Elabscience, Catalog No: E-EL-H0109), serum cotinine by HPLC (high pressure liquid chromatography).

Statistical Analysis:- Unpaired "t" test & one way ANOVA were used to analyze all the data for statistical significance, using the SPSS 19.0.2 program for windows.

Results

In present study, out of total 284 cigarette smokers, 272 were males and 12 were females. Mean age of the cigarette smokers and nonsmokers were 40.66±11.08 years and 37.42±9.73 years, respectively.

Table 1: Distribution of age-group among cigarette smokers (n=284).

Age group(yrs)	Male		Female		Total	
	No	%	No	%	No	%
18-29	41	15.07	02	16.67	43	15.14
30-41	75	27.57	02	16.67	77	27.11
42-53	81	29.79	04	33.33	85	29.93
54-60	75	27.57	04	33.33	79	27.82
Total	**272**	**95.77**	**12**	**4.23**	**284**	**100**

Table 2: Anthropometric measurements of cigarette smokers and non-smokers (n=568).

Parameters	Non-smokers (n = 284) Mean± SD	Cigarette smokers (n = 284) Mean± SD	*p* value	t value	95% Confidence interval for mean	
					Lower	Upper
Height(cm)	166.23 ±3.73	165.89 ±3.52	0.425	0.599	158.0	178.0
Weight	63.56±	66.18±	0.0	5.1	48.	76.

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(kg)	6.82	5.16	01	6*	0	0
BMI(kg/m^2)	23.00±2.10	24.09±1.63	0.001	6.93*	17.60	27.90
WC(cm)	78.57±4.51	83.16±4.62	0.189	11.98*	70.0	91.0
HC(cm)	90.92±4.44	89.37±3.76	0.006	-0.446	80.0	98.0
WHR	0.86±0.03	0.93±0.03	0.162*	27.39*	0.84	0.98

The cigarette smokers had significantly higher mean weight, BMI, waist circumference and waist hip ratio as compared to non smokers. These differences were found to be statistically significant ($p< 0.05$).

Table 3: Serum levels of cotinine & A1AT in cigarette smokers and non-smokers (n=568).

Parameter	**Non-smokers (n = 284) Mean ±SD**	**Cigarette smokers (n = 284) Mean ±SD**	**t value**	***p* value**	**95% Confidence interval for mean**		**Normal value**
					Lower	**Upper**	
Serum Cotinine	14.20±5.15	24.20±5.00	2.38	0.013	11.20	36.50	3-20 ng/mL
Serum A1AT	20.80±11.98	13.06±18.00	-1.06	0.027	3.31	77.53	18.59-81.15 ng/mL

The mean serum cotinine level was significantly raised in cigarette smokers as compared to non-smokers whereas mean serum A1AT level was significantly decreased in cigarette smokers as compared to non-smokers. This difference was found to be statistically significant ($p< 0.05$).

Table 4: Distribution of serum cotinine & A1AT levels according to duration & number of cigarette smoked in cigarette smokers (n=284).

Parameter		**1-15C/D <5years Mean± SD**	**15-20C/D <5years Mean± SD**	**15-20C/D 10years Mean ±SD**	**15-20C/D >10years Mean± SD**	***p* value**
Serum cotinine (mg/dl)		24.16±5.00	24.66±5.00	23.71±5.00	24.62±5.00	0.348
95% confidence interval for mean	**Lower**	21.79	22.76	25.10	22.73	
	Upper	24.29	25.59	27.46	25.50	
	A1AT	13.68±20.75	12.34±17.41	12.21±8.28	13.19±15.86	0.098
	Lower	12.41	10.03	10.34	11.80	
	Upper	14.26	12.71	14.80	19.48	

The mean serum cotinine level was significantly raised in cigarette smokers when adjusted with duration and number of cigarette smoked as compared to non-smokers. This difference was found to be statistically significant ($p< 0.05$).

Table 5: ANOVA analysis & unpaired 't' test of serum cotinine & A1AT level according to duration & number of cigarette smoked in cigarette smokers.

Parameter	**1-15 C/D <5**	**15-20C/**	**15-20C/**	**15-20C/**	

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		years	D <5 years	D 5-10years	D >10years	
		Sum of squares	Mean square	F value	*p* value	F Critical
Serum Cotinine	**Between groups**	391.490	130.497	5.565	0.001	**3.032**
	Within groups	6542.533	23.448			
A1AT	**Between groups**	1509.215	503.072	3.96	0.098	
	Within groups	99833.705	357.827			

The p value was <0.05 which signifies significant variation in levels of serum cotinine & A1AT when compared among each other in terms of group means.

Table 6: Correlation between serum A1AT with serum Lp(a) & cotinine levels in cigarette smokers and non smokers (n=568).

Parameter	Correlation analysis	SerumCotinine
Serum A1AT	**Correlation coefficient (r)**	-0.212
	S.E.OF 'r'	0.041
	t statistic	-5.159
	***p* value**	0.000
	R -square	0.043

A1AT had negative correlation with cotinine (r=-0.143; $p< 0.000$) and (r=-0.212; $p<0.000$) in cigarette smokers & non-smokers. Linear relationships were observed between the parameters.

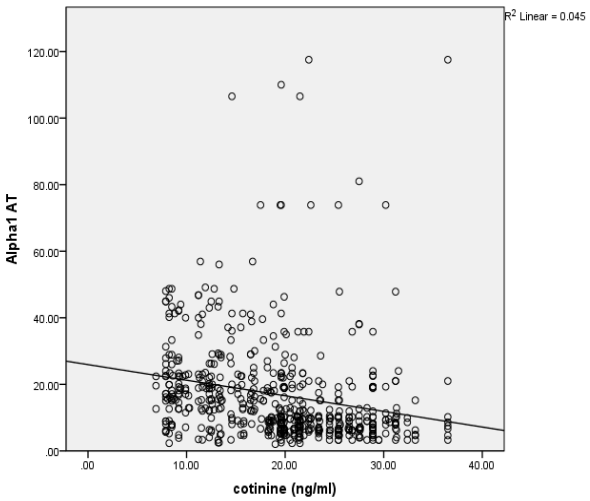

Fig. 1: Correlation of serum A1AT with serum cotinine in cigarette smokers & non-smokers

Discussion

Cigarette smoking that has spread all over the world is a reprehensible habit. Considerable research attests the adverse effects of chronic smoking on human health. In the development of many cardiovascular, pulmonary and ocular diseases and also neurological disorders smoking has been implicated (11).

Cigarette consumption has risen over the past two decades in India and most other countries. Reports reveal that the smoking rate is on continuous increasing. WHO estimates suggest that if the current pattern of smoking continues, this 21st century is about to see 1 billion tobacco deaths (12).

In the present study, Out of total 284 cigarette smokers, 272 were males & 12 were females. 15.14% of the cigarette smokers were in the age group 18-29 years followed by 27.11% within 30-41 years, 29.93% within 42-53 years & 27.82 within 54-60years (**Table 1**). According to study prevalence our study indicates that male are more prone for smoke related diseases than females. Same results were also obtained by other

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studies. Our finding is similar to the Townsend *et al* who reported in the sub-Saharan Africa that the prevalence of cigarettes use was higher among males than females across all countries in the region.

20% of men aged 18 and over smoked compared with 17% of women according to ash fact sheet in 2016. Smoking prevalence is highest among young adults 23% of those aged 16-24 and 24% among the 25-34 age groups. Smoking continues to be lowest among people aged 60 and over. Although they are more likely than younger people to have ever been smokers, they are more likely to have stopped smoking.

In present study prevalence of cigarette smoking among people aged 30 years and above and this was much higher among males (95.77%) than females (4.23%). A clustered community –based study in 2001 with about 50% of adult males between 30-60 years found to be smokers and very few females admitting to smoking similar finding has been reported from Delhi by Chhabra *et al* (13). The young often smoke because their peers smoke reported by these studies in urban areas. Their most common reason was their film hero who smokes. In the rural areas many people were unaware of the hazards of smoking (14).

In developing & developed countries females start smoking later than males due to the smoking has been considered socially unacceptable for women (with exceptions in certain areas of India, Nepal, Papua New Guinea, northern Thailand, and for Maoris). Religious constraints may be the reason, for example to buy cigarettes in Muslim countries women have had less spending power than men; traditional methods of smoking are adhere by rural women, e.g. hubble-bubble pipes, and are therefore a lower dosage of tobacco they exposed; and women use tobacco in other forms as chewing tobacco is used in some areas, such as parts of India and the Middle East (Subramanian, 2004). Where it is culturally less acceptable for women to smoke, there may be significant underreporting of smoking among women in countries (15).

Health effects associated with reproductive health such as problems associated with pregnancy, use of oral contraceptive, menstrual function, and cancers of the cervix and bladder are more prone in women smoker or who exposed with smokers. irregular menstrual cycles and increased menstrual discomfort may also caused by smoking. Women who are smokers may also have an earlier menopause, which increases chances of getting osteoporosis, heart disease and other conditions for which estrogen provides a protective effect. The risk of sudden infant death syndrome may also increase when a pregnant woman smokes.

Age, Height, Weight, Body Mass Index, Waist Circumference and Waist-hip ratio are simple and valid anthropometric measures for the assessment of risk of obesity & other systemic diseases in smokers. In the present study the cigarette smokers had significantly higher mean BMI, waist circumference and waist hip ratio as compared to non- smokers **(Table 2)**.

Some studies reported that measures of abdominal adiposity such as WC and WHR are better & simple markers of cigarette smoked in smokers, other studies claim that central adiposity measures such as WC and WHR do not provide additional prognostic information than BMI alone.

The amount of visceral adipose tissue (VAT) is indicated by Waist circumference or waist-to-hip ratio (WHR). WHR is higher in smokers than in nonsmokers indicated by several cross-sectional studies. The number of pack years of smoking is positively associated with WHR and between WHR and the number of cigarettes smoked there is a dose-response relation.WHR is negatively associated with the time since smoking cessation in former smokers. (16)

A study showed that CS may reduce testosterone concentrations in men. In male dogs, smoking induced a large reduction in serum testosterone concentrations. Overall, these results suggest that, in addition to excess cortisol, an imbalance between male and female sex hormones and a decrease in testosterone in males could play a role in the effect of smoking on VAT (17).

Several studies reported that smoking was associated with lower weights and BMI. Smoking is also known to be associated with insulin resistance and type 2 diabetes. In Caucasian men and women, current smokers had lower mean BMI, WC, and body fat percentage, compared with non-smokers. Age-adjusted mean WC and body fat increased with cigarettes smoked per day among smokers, while not significantly related to BMI. Basterra-Gortari *et al*., demonstrated

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greater weight gain among active than never smokers over a 50 month follow-up period.

Nicotine is a known appetite suppressant and many studies show that nicotine decreases food intake in mice (18). Thus, one would expect that smokers with slower nicotine metabolism may potentially have lower BMI, but our hypothesis was not supported.

It is possible that persons who like cigarettes may also like foods that are rich and high in fat or sugar, which counteracts any appetite suppressant characteristics of nicotine. Heavy smokers tend to have greater body weight than light smokers or nonsmokers remain unanswered. One explanation could be that heavy smokers are more likely to adopt behaviors favoring weight gain (eg, low physical activity, unhealthy diet, and high alcohol intake) than are light smokers or nonsmokers. Smokers eat less fruit and vegetables adopt unhealthy patterns of nutrient & calorie intake, than do nonsmokers (19).

Number of biochemical markers like nicotine, cotinine and carbon monoxide in the expired air and carboxyhaemoglobin in blood have been used to validate claims of non-smoking. Levels of nicotine and carbon monoxide/carboxyhaemoglobin are easier to determine but can be raised through exposures unrelated to smoking such as traffic emissions and diet. Cotinine is possibly the best marker for situations where accuracy is paramount. It is one of the most frequently used biomarkers for exposure to environmental tobacco smoke in body fluids and widely practical as a biomarker of nicotine uptake and exposure to both active and secondhand tobacco smoke (20).

In present study the mean serum cotinine level was significantly raised in cigarette smokers as compared to non-smokers and also increased when smokers adjusted with age & duration & number of cigarette smoked **(Table 3, 4 & 5)**.

The present study also shows the correlation of cotinine with serum A1AT level in mild group I, mild group II, Moderate & heavy cigarette smokers respectively. Cotinine had negative correlation serum A1AT ($r=0.458$, $p<0.006$) in mild group II & moderate cigarette smokers respectively Linear relationships were observed between the parameters **(Table 6)**.

Balhara YPS, Jain R 2013 detected urinary cotinine by the highest sensitivity and specificity method for smoking using ELISA kits of Calbiotech (21). Kulza M, *et al* 2012 found that the concentration of salivary cotinine was increased in cigarette smokers, detected by using high performance liquid chromatography with diode array detection. Mean concentrations of cotinine was found to be highly increased suggested that saliva cotinine is useful in the assessment of tobacco smoke (22). Nuca C, *et al* 2012 By used NicAlert™ Saliva tests and found that 44.06% were active smokers, 16.43% were non-smokers and 39.50% were passive smokers (23).

In the Coronary Artery Risk Development in (Young) Adults study (CARDIA), serum cotinine levels were higher in black smokers than in white smokers, despite lower estimated daily nicotine exposure among black smokers (24).

Cotinine levels have earlier been used to validate the smoking status of an individual. These biomarkers has also been used in epidemiological studies, to assess the effects of tobacco use on human health, as measures to estimate the exposure to environmental tobacco smoking, and for assessment of the efficacy of interventional methods on cessation of smoking.

Alpha-1 antitrypsin deficiency is a genetic disorder that causes deficiency of the protein, alpha-1 antitrypsin (A1AT). The lack of this protein may cause lung disease over time. Alpha-1 antitrypsin deficiency is often undetected for many years, and although treatable, the disease is in curable.

In our study the mean Serum A1AT level was significantly decreased in cigarette smokers as compared to non-smokers. The mean serum A1AT level also was significantly decreased in cigarette smokers when adjusted with duration and number of cigarette smoked as compared to non-smokers. This difference was found to be statistically significant ($p< 0.05$) **(Table 2,4 & 5)**.

Different studies demonstrated change in A1AT activities in serum of patients with COPD; which are more commonly associated with cigarette smoking habit (25). One more study also shows increased A1AT activity in cigarette smokers compared to healthy nonsmokers which suggest its role in mediating some of

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the chronic health hazards of smoking like COPD, TB and other lung diseases; which were found to be associated with an increased levels of A1AT and also reported positive correlation between pack size and A1ATactivity (26).

A1AT level was lower in COPD patients with smoking as compared to COPD patients without smoking. The present study shows the significant difference in serum AAT level between the two groups ($p<0.05$). This study supports the data of previous studies F. Ogushi *et al*. 1991,Oliver Senn, Erich W Russi, Christian Schindler *et al*. 2008 and Deore Deepmala *et al*. 2012 (27, 28).

Higashimoto *et al* reported in a cross-sectional study that serum alpha-1 antitrypsin levels were inversely correlated with cigarette smoke in Japanese patients with COPD (29). Furthermore, Senn*et al* reported that serum alpha-1 antitrypsin levels were inversely correlated with cigarette smoke and FEV1 in the general population (30).

These findings could also explain the role of AAT in the development of COPD; its decreased levels in the blood resulting from systemic inflammation, meeting the elevated levels of alveolar neutrophil elastase and proving to be a marker for increased risk of COPD development, as a result of low-grade lung inflammation. Direct exposure of α1-antitrypsin to gas phase cause loss of elastase inhibitory capacity leading to formation of reactive free radicals from smoke that inactivate a 1-antitrypsin by oxidizing methionine 358 terminal amino acid (31).

Conclusion:- Based on our findings and the other data in the study, we can say that these biochemical parameters might be helpful biomarkers for the detection of cigarette smokers with a high risk of developing smoke induced diseases i.e. pulmonary & cardiovascular diseases.

Cigarette smoking may increased serum cotinine represents exposure of smoke, decreased serum A1AT modify the deleterious effect of smoking on these markers, indicating genetic susceptibility to smoking-related diseases and shows the relationship between biomarkers.

Limitations: - The study was limited to the population residing in and around Haldwani and their involvement in the study especially in case of females. Lack of fund, time and manpower prevented the inclusion of a large study group and other sensitive biochemical markers of cigarette smoke.

Future cross-sectional, longitudinal and mechanistic studies are needed to determine how CS, anthropometric markers, A1AT & cotinine are useful in large populations of cigarette smokers with the inclusion of clinically relevant endpoints are needed to extend these findings.

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References:-

1. U.S. Environmental Protection Agency. Quality Criteria for Water 1986. Washington: U.S. Environmental Protection Agency, Office of Water Regulations and Standards, 1986. Publication No. EPA 440/5-86-001.
2. IARC (2004a). Tobacco smoke and involuntary smoking. IARC Monogr Eval Carcinog Risks Hum, 83: 1–1438.PMID:15285078.
3. Guerin MR. Chemical composition of cigarette smoke. In: Gori GB, Bock FG, editors. Banbury Report No. 3: A Safe Cigarette? Cold Spring Harbor (NY): Cold Spring Harbor Laboratory, 1980:191–204.
4. Warren CW, Jones NR, Peruga A, Chauvin J, Baptiste J-P, de Silva VC, el Awa F, Tsouros A, Rahman K, Fishburn B, et al: Global Youth Tobacco Surveillance, 2000–2007. MMWR 2008, 57(SS01):1–21.
5. Pryor WA, Stone K: Oxidants in cigarette smoke. Radicals, hydrogen peroxide, peroxynitrate, and peroxynitrite. Ann N Y Acad Sci 1993;686:12-27; discussion 27-18.
6. Isaac PF, Rand MJ. Cigarette smoking and plasma levels of nicotine. Nature. 1999;236:308-310.

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www.jchr.org

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7. Alper, C. A., Raum, D., Awdeh, Z. L., Petersen, B. H., Taylor, P. D., and Starzl, T. E. (2000) Clin. Immunol. Immunopathol. 16, 84–89.
8. McElvaney, N. G., Cervantes-Laurean, D., Wehr, N., Gabrielle, K., Barnes, P., Robinson, W., Moss, J., and Levine, R. L. (1996) Am. J. Res. Crit. Care Med. 153, 737.
9. Imtiaz D, Kandpal SD, Juyal R, Shrotriya VP, Singh AK. A study on prevalence and [9]pattern of smoking among rural population in dehradun district of Uttarakhand. Natl J Community Med. 2014;5(4):440-43.
10. Nagaraj, Satish Kumar D, Paunipagar PV. Study of serum malondialdehyde and [10]vitamin C in smokers. Journal of Scientific & Innovative Research. 2014;3(6):569-71.
11. Esterbauer H., Striegl, G., Puhl H. and Rotheneder. M. (2012) Contrinuous monitoring of in vitro oxidation of human low density lipoprotein. Free radical Res. Commun 6: 67-75.
12. G.k. Gajalakshmi, Prabhat J, Kent Ranson and Son Nguyen (2000). Global patterns of smoking and smoking attributable moxt&lity (Internet).
13. Chhabra SK, Rajpal S, Gupta R. Patterns of smoking in Delhi and comparison of chronic respiratory morbidity aming beedi and cigarette smokers. Indian J Chest Dis Allied Sci. 2001; 43:19-26.
14. Muttapppallymyalil, J; Sreedharan, J & Divakaran, B. (2010). Smokeless Tobacco Consumption among School Children. Indian J Cancer.47:19-23.
15. Subramanian, S. V. (2004). Patterns and Distribution of Tobacco Consumption In India: Cross Sectional Multilevel Evidence From The 1998-9.National Family Health Survey. Am J Public Health. 92, 102–116.
16. Pasquali R, Vicennati V. Activity of the hypothalamic-pituitaryadrenal axis in different obesity phenotypes. Int J Obes Relat Metab Disord 2000;24(suppl):S47–9.
17. Meikle AW, Liu XH, Taylor GN, Stringham JD. Nicotine and cotinine effects on 3 alpha hydroxysteroid dehydrogenase in canine prostate. Life Sci 1988;43:1845–50.
18. Xie XT, Liu Q, Wu J, Wakui M. Impact of cigarette smoking in type 2 diabetes development. Acta Pharmacol Sin. 2009;30(6):784-787.
19. Mineur, Y.S., Abizaid, A., Rao, Y., et al., 2011. Nicotine decreases food intake through activation of POMC neurons. Science 332 (6035), 1330–1332. http://dx.doi.org/10.1126/science.1201889 (10).
20. Kvaavik E, Meyer HE, Tverdal A. Food habits, physical activity and body mass index in relation to smoking status in 40–42 year old Norwegian women and men. Prev Med 2004;38:1–5.
21. Balhara YPS, Jain R. A receiver operated curve-based evaluation of change in [6]sensitivity and specificity of cotinine urinalysis for detecting active tobacco use. Journal of Cancer Research and Therapeutics. 2013;9(1):84-89.
22. Kulza M, Wozniak A, Przybylowska SM, Czarnywojtek A, Kurhanska-Flisykowska [4]A, Florek E. Saliva cotinine determination using high-performance liquid chromatography with diode - array detection. Przegl Lek. 2012;69(10):837-40.
23. Nuca C, Amariei C, Badea V, Zaharia A, Bucur L, Arendt C. Salivary cotinine [11]- biomarker of tobacco consumption in the assessment of passive smoking prevalence. FARMACIA. 2012;60(5):662-74.
24. Wagenknecht LE, Cutter GR, Haley NJ, et al. Racial differences in serum cotinine levels among smokers in the Coronary Artery Risk Development in (Young) Adults study. Am J Public Health 1990; 80:1053-56.
25. Hedstrom AK, Hillert J, Olsson T, Alfredsson L. Smoking and multiple sclerosis susceptibility. Eur JEpidemiol. 2013;28:867-74.
26. Ramasamy R, Murugaiyan SB, Arulkumaran U, Sathiya R, Velu KV, Gopal N. Comparative study of adenosine deaminase activity, insulin resistance and lipoprotein (a) among smokers and healthy non-smokers. Int J Res MedSci. 2016;4:1950-3.
27. Ogushi F, R. C. Hubbard, C. Vogelmeier, G. A. Fells, and R. G. Crystal. Risk Factors for

Emphysema Cigarette Smoking Is Associated with a Reduction in the Association Rate Constant of Lung al-Antitrypsin for Neutrophil Elastase. The Journal of Clinical Investigation. 1991 87; 1060-65.

28. Oliver Senn1, Erich W Russi, Christian Schindler, Medea Imboden, Arnold von Eckardstein, Otto Brändli. Circulating alpha1-antitrypsin in the general population: Determinants and association with lung function. Respiratory Research. 2008, 9:35.
29. Higashimoto Y, Yamagata T, Honda N, et al. Clinical and inflammatory factors associated with body mass index in elderly patients with chronic obstructive pulmonary disease. Geriatr Gerontol Int. 2011;11 (1):32–38. doi:10.1111/j.1447-0594.2010.00629.
30. Senn O, Russi EW, Schindler C, et al. Circulating alpha1-antitrypsin in the general population: determinants and association with lung function. Respir Res. 2008;9:35. doi:10.1186/1465-9921-9-35.
31. Somayajulu, Rao R, Reddy. Serum Alpha 1-Antitrypsin in smokers and non-smokers. Ind J Clin Biochem 1996; 11(1):70.