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Impact of Tobacco Consumption on Cardiovascular and Respiratory Systems.

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Abstract:
Background: Tobacco consumption significantly harms both the cardiovascular and respiratory systems. It contributes to heart disease, high blood pressure, and stroke by damaging blood vessels and promoting atherosclerosis. In the lungs, tobacco smoke causes chronic inflammation, reduces lung function, and increases the risk of diseases like COPD and lung cancer. Reducing tobacco use through public health efforts is essential to prevent these life-threatening conditions.
Objective: Impact of Tobacco Consumption on Cardiovascular and Respiratory Systems
Methods: A cross-sectional study was conducted among voluntary male participants from Sarojini Naidu Medical College, Agra. The study included smokers aged 18–32 years with a minimum smoking history of one year.
Results: The study demonstrated significant cardiovascular and respiratory impairments associated with tobacco consumption. Among smokers, systolic and diastolic blood pressures were elevated compared to non-smokers, indicating increased cardiovascular strain. Pulmonary function tests revealed reduced lung capacity and airflow obstruction in smokers, with a marked decline in forced expiratory volume (FEV1) and forced vital capacity (FVC) compared to controls.
Conclusion: Tobacco consumption has a significant negative impact on both the cardiovascular and respiratory systems. It contributes to elevated blood pressure, increased cardiac workload, and impaired lung function, leading to a higher risk of heart disease, stroke, COPD, and lung cancer. Preventive measures and smoking cessation are essential to reduce these health risks and improve overall well-being.

Introduction:
Tobacco consumption is considered to be an important public health problem globally by World Health Organization (WHO). With a global morbidity census of 6 million per year, it has to be treated as an urgent matter on priority basis and the tobacco consumption in any form has to be curbed. Tobacco consumer deprives his family of its income when he dies prematurely and raises the cost of health care, and affects the economy of the nation he belongs to when he is alive in a larger scale.[1]

Prevalence of smoking tobacco in India has alarmingly increased from 27% (1998) to 36% (2015).[2] Many countries are already adopting to strict rules to curb free smoking that pollute the breathing air by imposing heavy taxation on tobacco-based products, banning provoking advertisements and even embedding pictorial warnings in media and general locations to create awareness on passive smoking. Inspite of all these measures, still there is an alarming raise in the current tobacco consumers making us to wonder whether there is any strong factor that make them continue smoking. It acts as a trigger and worsens the condition in asthmatic subjects. Though in most of cases, tobacco smoking causes lung cancer, it is alarming to note that it can also cause cancer in other regions of the body such as bladder, blood, pancreas, stomach, colon and cervix.[3]

Surprisingly, Inspite of enormous hike in the price of tobacco and its products, there is always a steady raise in the number of tobacco users in our country. This is attributed mainly to the affordability of the people to buy the tobacco products irrespective of the cost. An empirical analysis on the taxation on the tobacco products by the Indian Government

suggests that the current excise duty and value added tax (VAT) are not sufficient to curb the growing affordability of the people buying tobacco products.[4]

Among all the ingredients, nicotine is considered to be the most important component that makes the smoker addictive which sustains the habit of cigarette smoking. Tobacco addiction is an interplay of pharmacological, conditional, genetical, social and environmental factors.[5]

Effect of nicotine as an addictive agent in brain
In brain, nicotine releases dopamine producing a pleasurable experience and causes reinforcing effects in the mesolimbic area, corpus striatum, frontal cortex, ventral tegmentum of the midbrain and the nucleus accumbens which are the centers for drug induced reward.[6]

Monoamine oxidase gets inhibited by the acetaldehyde in tobacco smoke which in turn reduces the metabolism of dopamine contributing to the addictiveness of smoking.[7]

Effect of nicotine on cardiac autonomic function
Intravenous administration of nicotine causes an acute increase in vascular blood pressure including a raise in heart rate within five to ten minutes of exposure.[8] Hemodynamic effects in a smoker gets stabilized when the nicotine level in a smokers blood gets increased.[19] American lung association states that, nicotine not only releases catecholamines at the adrenergic nerve terminals but also increases sympathetic activity.[9]

Effect of nicotine on respiratory system
Nicotine is rapidly absorbed from smaller airways and alveoli. Concentration of nicotine in blood rises quickly as one completes a cigarette on smoking. During smoking on an average 1.0 mg (range; 0.3 to 2mg) of nicotine is absorbed.[10] Apart from the two sites in the lungs, nicotine has its effects on bronchial epithelial cells, normal fibroblast cells, embryonic lung cells and lung cancer cells.[11]

Material and Methods:
This study was conducted among voluntary participants chosen from Sarojini Naidu Medical College, Agra.

Study design: Cross sectional descriptive study
Sample size: total sample size Derived sample size for this study was 195,
The study subjects were grouped into Group I (Low dependency) (n=65), Group II (Moderate dependency) (n=65), and Group III (High dependency) (n=65) and Controls (n=70)-Non users of tobacco

Inclusion criteria:
1. Male smokers aged between 18 to 32 years were selected for the study.
2. Subjects who used to smoke tobacco only and who had an established habit of smoking for a period for not less than one year of smoking were included into the study.

Exclusion criteria:
1. Tobacco users of all other kinds other than smoking tobacco viz smokeless, dipping tobacco, hookah users etc.
2. Smokers who were on medication in the past 30 days
3. Subjects who were diagnosed for any cardiovascular or respiratory illness
4. Known cases of diabetes, hypertension, neurological disorders
5. Habit of alcoholism
6. Subjects who were with the habit of regular exercise or sports activities.

Result:

Parameters (Mean±SEM)	Controls (70)	Group I (65)	Group II (65)	Group III (65)	P value
Age (yrs)	28.26 ± 0.63	29.73 ± 0.54	28.91 ± 0.57	30.46 ± 0.77	0.14
Weight (kgs)	67.00 ± 1.21	65.54 ± 1.23	66.11 ± 1.42	62.50 ± 2.13	0.27
Height (cms)	164.91± 1.21	164.63 ± 0.45	164.76 ± 0.57	161.68 ± 1.27	0.19
BMI (kg/m²)	25.87 ± 1.65	24.15 ± 0.42	24.27 ± 0.44	24.06 ± 0.94	0.64

Table 1. Anthropometric parameters of controls and nicotine dependent smokers

There were no statistically significant differences in age, weight, height, or BMI among the control and experimental groups ($P > 0.05$). This indicates that the groups were comparable in their baseline physical characteristics.

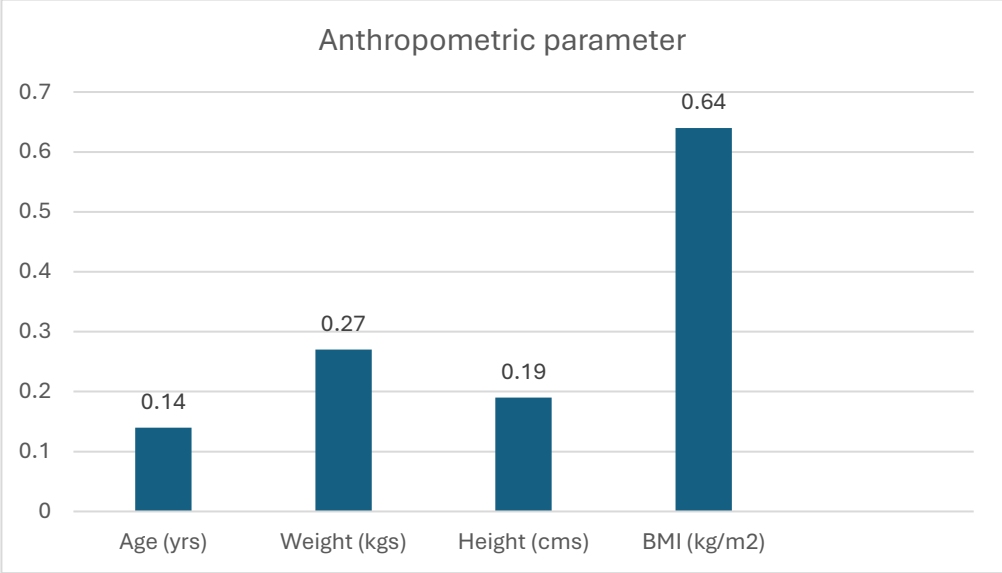

Fig: 1 Anthropometric parameter of controls and nicotine dependent smokers

Parameters (Mean± SEM)	Controls	Group I	Group II	Group III	F Value	P value
FEV_1 (L)	2.61 ± 0.09	1.99 ± 0.12	2.11 ±0.07	2.17 ±0.06	8.561	0.001*
FVC (L)	2.82 ± 0.09	2.50 ± 0.08	2.40 ±0.06	2.47 ±0.07	5.544	0.001*
FEV_1/FVC (%)	86.83 ±0.90	74.58 ±1.58	70.01 ±2.17	79.75 ±1.93	1.937	0.012*
PEFR (L)	6.75 ± 0.21	5.70 ± 0.22	5.72 ±0.20	5.10 ±0.22	10.074	0.001*

Table 2. Values of spirometry in controls and nicotine dependent smokers

FEV_1, FVC, and PEFR showed decline from controls to that of Group I, Group II and Group III of smokers which were statistically significant

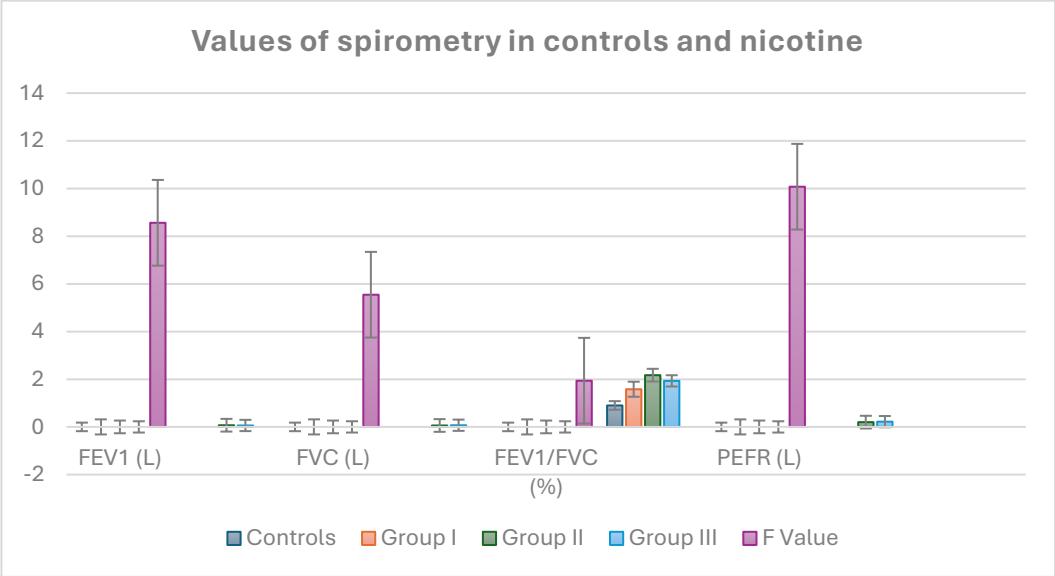

Fig: 2 Values of spirometry in controls and nicotine dependent smokers

FEV_1 (L)	Control	Group I	Group II	Group III
Control	-	0.61 ± 0.13	0.49 ± 0.13	0.43 ± 0.13
		p<0.000*	p<0.001*	p<0.006*
Group I	61± 0.13	-	0.12± 0.13	0.18± 0.13
	p<0.000*		p<0.791	p<0.515

Group II	_0.49 ± 0.13	0.12 ± 0.13	-	0.06 ± 0.13
	p<0.001*	p<0.0791		p<0.969
Group III	_0.43 ± 0.13	0.18 ± 0.13	0.06 ± 0.13	-
	p<0.006*	p<0.515	p<0.969	

Table no. 3 FEV_1 in controls and nicotine dependent smokers

FVC (L)	**Control**	**Group I**	**Group II**	**Group III**
Control	-	0.32 ± 0.11	0.42 ± 0.11	0.35 ± 0.11
		p<0.027*	p<0.001*	p<0.012*
Group I	0.32 ± 0.11	-	0.10 ± 0.11	0.03 ± 0.11
	p<0.027*		p<0.799	p<0.993
Group II	0.42 ± 0.11	0.10 ± 0.11	-	0.74 ± 0.11
	p<0.001*	p<0.799		p<0.920
Group III	0.35 ± 0.11	0.03 ± 0.11	0.07 ± 0.11	-
	p<0.012*	p<0.993	p<0.920	

Table no. 4 FVC in controls and nicotine dependent smokers

FVC was significantly reduced in all experimental groups compared to the control group ($p < 0.05$). However, there were no significant differences in FVC among the experimental groups themselves ($p > 0.05$), suggesting uniform impairment.

FEV_1/FVC (%)	**Control**	**Group I**	**Group II**	**Group III**
Control	-	12.24 ± 2.37	16.81 ± 2.37	7.07 ± 2.37
		p<0.000*	p<0.000*	p<0.017*
Group I	12.24 ± 2.37	-	4.56 ± 2.42	5.16 ± 2.42
	p<0.000*		p<0.236	p<0.144
Group II	16.81 ± 2.37	4.56 ± 2.42	-	9.73 ± 2.42
	p<0.000*	p<0.236		p<0.000*
Group III	7.07 ± 2.37	5.16 ± 2.42	9.73 ± 2.42	-
	p<0.017*	p<0.144	p<0.000*	

Table 5 EV_1/FVC in controls and nicotine dependent smokers

PEFR (L)	**Control**	**Group I**	**Group II**	**Group III**
Control	-	1.04 ± 0.30	1.02 ± 0.30	1.65 ± 0.30
		P<0.004*	P<0.005*	P<0.000*
Group I	1.04 ± 0.30	-	0.02 ± 0.31	0.60 ± 0.31
	P<0.004		P<1.00	P<0.215
Group II	1.02 ± 0.30	0.02 ± 0.31	-	0.62 ± 0.31
	P<0.005	P<1.000		P<0.187
Group III	1.65 ± 0.30	0.60 ± 0.31	0.62 ± 0.31	-
	P<0.000*	P<0.215	P<0.187	

Table 6. PEFR in controls and nicotine dependent smokers

Parameters (Mean± SEM)	**Controls**	**Group I**	**Group II**	**Group III**	**F Value**	**Pvalue**
SBP (mmHg)	126.82 ± 0.97	129.45 ± 1.28	123.83 ± 1.11	131.10 ± 2.88	4.051	0.008*
DBP (mmHg)	77.28 ± 0.90	81.21 ± 1.16	77.45 ± 0.92	76.68 ± 1.84	1.144	0.014*
HR (beats /min)	80.11 ± 1.14	78.54 ± 1.11	82.56 ± 0.76	81.15 ± 1.65	2.061	0.106
MABP (mmHg)	93.79 ± 0.77	97.29 ± 1.09	92.91 ± 0.89	94.82 ± 0.55	3.444	0.017*

RPP	10186.91± 183.43	10166.69± 175.09	10215.96 ± 122.76	11972.90 ± 285.29	8.081	0.001*

Table 7. Cardiovascular parameters in controls and nicotine dependent smokers.

Significant differences were observed among the groups in systolic blood pressure (SBP) (p = 0.008), diastolic blood pressure (DBP) (p = 0.014), mean arterial blood pressure (MABP) (p = 0.017), and rate pressure product (RPP) (p = 0.001). Group III showed the highest SBP (131.10 ± 2.88 mmHg) and RPP (11972.90 ± 285.29), indicating increased cardiovascular workload. DBP was significantly higher in Group I compared to controls. Heart rate (HR) differences were not statistically significant (p = 0.106). Overall, smoking was associated with elevated blood pressure parameters and increased myocardial workload, especially in Group III.

Diastolic blood pressure (DBP) was significantly higher in Group I smokers compared to the control group (p < 0.030), indicating early cardiovascular effects of smoking. No significant differences in DBP were found between the control group and Groups II or III. Differences between smoker groups were not statistically significant, though Group I showed a trend toward higher DBP compared to Groups II and III. Overall, smoking appears to raise DBP mainly in the initial stages.

TABLE:8 Diastolic pressure (DBP) in controls and nicotine dependent smokers

HR/min	Control	Group I	Group II	Group III
Control	-	1.57 ± 1.45	2.45 ± 1.66	1.04 ± 1.94
		p<0.702	p<0.457	p<0.950
Group I	1.57 ± 1.45	-	4.02 ± 1.66	2.61 ± 1.94
	p<0.702		p<0.077	p<0.535
Group II	2.45 ± 1.66	4.02 ± 1.66	-	1.40 ± 2.11
	p<0.457	p<0.077		p<0.909
Group III	1.04 ± 1.94	2.61 ± 1.94	1.40 ± 2.11	-
	p<0.950	p<0.535	p<0.909	

No statistically significant differences in heart rate were observed between the control group and any of the smoker groups (Group I, II, or III), with all p values > 0.45. Although Group II showed a trend toward a higher heart rate compared to Group I (p = 0.077), this was not statistically significant. Overall, smoking did not significantly affect resting heart rate in this study.

Table 9 Heart rate (HR) in controls and nicotine dependent smokers.

DBP(mmHg)	Control	Group I	Group II	Group III
Control	-	3.92 ± 1.41	−0.16 ± 1.62	0.60 ± 1.89
		p<0.030*	p<1.00	p<0.989
Group I	3.92 ± 1.41	-	3.76 ± 1.62	4.53 ± 1.89
	p<0.030*		p<0.096	p<0.080
Group II	0.16 ± 1.62	3.76 ± 1.62	-	0.76 ± 2.05
	p<1.00	p<0.096		p<0.982
Group III	−0.60 ± 1.89	4.5 ± 1.89	−0.76 ± 2.05	-
	p<0.989	p<0.080	p<0.982	

Heart rate showed a decrease in Group I and an increase Group II and no change in Group III smokers compared to that of controls. Between the groups, there was no significance in the changes between the three groups of smokers and the controls and among the three groups of smokers

MABP (mmHg)	Control	Group I	Group II	Group III
Control	-	3.49 ± 1.34	0.88 ± 1.54	1.02 ± 1.80
		p<0.049*	p<0.939	p<0.941
Group I	3.49 ± 1.34	-	4.38 ± 1.54	2.47 ±1.80
	p<0.049*		p<0.025*	p<0.518
Group II	0.88 ± 1.54	4.38 ± 1.54	-	1.91 ± 1.95
	p<0.939	p<0.025*		p<0.762
Group III	1.02 ± 1.80	2.47 ± 1.80	1.9 ± 1.95	-
	p<0.941	p<0.518	p<0.762	

Table: 10 Heart rate (HR) in controls and nicotine dependent smokers.

MABP showed an increase in Group I and III and a decrease in Group II smokers compared to that of controls. Between the groups, the increase in Group I smokers was significant ($p<0.049$) and that of Group III smokers was non-significant ($p<0.941$) against the controls. The decrease in Group II smokers was significant against the increase in Group I smokers ($p<0.025$). Between Group I and Group III, the increase was not significant ($p<0.518$)

Discussion: Tobacco, the major but preventable risk factor for non-communicable disease that is taking toll in both the developed and developing nations. This could be well prevented suitably with policy implementations like creating awareness towards the adverse effects of smoking, public smoking ban, increasing taxation on tobacco products and more importantly encouraging smokers to quit smoking.

In the respiratory system, tobacco damages airway linings, impairs mucus clearance, and leads to chronic conditions like bronchitis, emphysema, COPD, and lung cancer. Overall, it reduces oxygen supply to the body, causing fatigue and breathing difficulties.

Decrement in the FEV1/FVC values with respect to the increasing age in adults is a well-documented observation.129 Less than 50% FEV1/FVC ratio and maximum voluntary ventilation (MVV) in a COPD patient predict his high risk for post operative complications.

The trend in the systolic blood pressure recorded among the different groups of tobacco users shows a gradually raising systolic blood pressure which predominantly shows a raise which supports the raising trend of sympathetic activities as seen in the HRV indices.

Conclusion: The study indicates that the level of nicotine dependency serves as a useful indicator of both cardiac autonomic and respiratory function in smokers. A proportionate and significant decrease was observed in ventilatory function as nicotine dependence increased. Interestingly, reduced FEV1 values were seen in the low nicotine-dependent group, which is particularly concerning as these individuals were mostly young and early-stage smokers. In contrast, moderate and high nicotine-dependent smokers showed better FEV1 values than their low-dependent counterparts. The FEV1/FVC ratio did not show significant differences across different levels of nicotine dependency. However, PEFR was found to decrease consistently with increasing dependency. In terms of cardiac autonomic function, sympathetic activity (measured by LF activity) generally decreased with higher nicotine dependence, but a significant rise was noted among those with high dependency.

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