

## ORIGINAL RESEARCH

**Smoking Effect's on Heart Rate at Rest, During Exercise and Recovery in Adults**<sup>1</sup>Dr. Bharat Bhushan Tayal, <sup>2</sup>Dr. Nishant Tayal, <sup>3</sup>Dr. Komal Tayal, <sup>4</sup>Dr. Uma Tayal<sup>1</sup>Associate Professor, Department of Physiology, NCR Medical College, Meerut, Uttar Pradesh, India<sup>2</sup>Associate Professor, Department of Medicine, LLRM Medical College, Meerut, Uttar Pradesh, India<sup>3</sup>Associate Professor, Department of Obstetrics and Gynaecology, LLRM Medical College, Meerut, Uttar Pradesh, India<sup>4</sup>Professor, Department of Pathology, NCR Medical College, Meerut, Uttar Pradesh, India**Corresponding author:**Dr. Nishant Tayal

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**Abstract**

**Introduction** - In elderly and middle-aged populations, there is a known correlation between smoking, aberrant heart rate (HR) values, and poor cardiovascular health but there is scarcity of literature about research done among young adults. The aim of present study is to evaluate the smoking effects on heart rate at rest, during exercise and recovery in adults.

**Material and methods**- 325 young adult students were chosen as a sample based on their body mass index, level of physical activity, smoking habit, and health. Every participant completed a maximum Bruce treadmill test, and their heart rates were monitored before, during, and after the exercise session ended.

**Results**- Compared to non-smokers, smokers' resting heart rates were noticeably greater. Smokers' heart rates increased during exercise considerably more slowly in both male and female smokers. Both male and female smokers fell short of their age-predicted maximal heart rate. In comparison to non-smokers, the actual maximum HR attained (HR<sub>max</sub>) was considerably lower in smokers, both male and female. Both male and female smokers had considerably decreased heart rate reserves. Only female smokers experienced a considerable attenuation of the HR drop during recovery. Compared to men, women displayed a higher HR response during sub-maximal exercise and a higher resting HR.

**Conclusion** – It was discovered that smoking had an impact on young smokers' heart rates (HR), elevating HR during rest, decreasing HR rise during exercise, and hindering HR<sub>max</sub> attainment. Furthermore, smoking was linked, albeit only in females, to a lessened HR fall during recovery.

**Keywords** – adult, exercise, heart rate, heart rate recovery, heart rate reserve, smoking, young

**Introduction**

One of the major global causes of early death is smoking-related disorders.[1] The European Society of Cardiology reports that smoking causes 13% of cardiovascular deaths in women and 28% of cardiovascular deaths in men between the ages of 35 and 69.[2] Long-term smoking raises the risk of coronary artery disease and myocardial infarction among other impacts on the circulatory, pulmonary, and cardiovascular systems.[3]

The relationship between heart rate (HR) and cardiovascular health is well-established. A highly significant, non-invasive, and simple-to-measure indicator of myocardial function is HR.[4-6] HR reaction during the workout[7] and a decrease in HR after exercise [8] are also excellent indicators of autonomic heart function. Numerous studies indicate that the results of reduced HR fall during recovery and blunted HR elevation during progressive exercise (chronotropic incompetence) are significant indicators of underlying autonomic dysfunction linked to higher rates of cardiovascular morbidity and death. Elevated resting heart rate (HR<sub>rest</sub>) and irregular heart rate responses before or after physical activity could predispose individuals to cardiovascular disease before symptoms appear and help identify high-risk individuals early on.[9-12] The importance of HR in clinical practice for evaluating cardiovascular function has not been underappreciated, despite these indications.4,5

There is a correlation between smoking and greater HR<sub>rest</sub> [13] and chronotropic incompetence.[14,15] Furthermore, populations with a blunted HR increase during exercise or an attenuated HR fall during recovery had a considerably greater smoking prevalence. In general, the association between smoking and cardiovascular disease may be explained by the HR reactions to cigarette smoking. [16] Nonetheless, despite the fact that smoking's effects on HR appear to vary with age, relatively little is known about how smoking affects HR in young adults.

Hence the aim of present study is to evaluate the smoking effects on heart rate at rest, during exercise and recovery in adults.

### **Material & methods**

The present cross sectional study was conducted among young college students for period of one year. The ethical permission was taken from ethical review board of allied institution before commencement of study. Informed consent was taken from students after explaining them the complete procedure of study.

The students were selected randomly by consecutive sampling. Smokers were defined as those who had smoked 20 or more cigarettes per day for at least three smoking years. Non-smokers had never smoked Total 400 students were selected on the basis of following inclusion and exclusion criteria:

**Inclusion criteria-** Healthy college students, 20-29 years of age, of normal weight ( $18.5 \text{ kg/m}^2 \leq \text{BMI} \leq 24.9 \text{ kg/m}^2$ ), and normotensive (systolic blood pressure: SBP<140mm Hg; diastolic blood pressure: DBP<90 mm Hg) with a low consumption of coffee (up to two cups per day) and alcohol (up to seven drinks per week) were included.

**Exclusion criteria-** Students with elevated heart rate at rest, metabolic diseases, such as diabetes, hyperlipidaemia and thyroid disease, physical disability, recent illness, pregnancy and a history of alcohol or drug abuse, were excluded.

A standardised self-addressed questionnaire was given to all 400 college students, but only 325 out of them submitted the completely filled form. Therefore in the final sample only 325 students were taken in the study.

Every test and measurement was carried out in the morning, in an environment with consistent humidity and temperature. The physical activity level of the subjects was evaluated using the short International Physical Activity Questionnaire (IPAQ-Gr). After ten minutes of rest, the individuals were measured supine for baseline HR<sub>rest</sub>, SBP, and DBP. Every participant used the usual Bruce maximal treadmill test for their workout. The subjects' ability to exercise was inferred indirectly from the duration of the maximal exercise test. The American Heart Association's criteria were adhered to throughout the exercise testing protocols.[17] The individuals fasted for at least six hours prior to the exercise test, abstaining from alcohol, caffeine, and heavy meals. Subjects did not use the handrails as support during the testing. The individuals who managed to proceed to the fourth phase of the Bruce regimen

engaged in running-style exercise. There was no usage of age-predicted target HRs as predefined endpoints. When symptoms such extreme weariness, dyspnea, intense leg pain, or exhaustion set in, the test was stopped at maximum effort. After the exercise test ended, all subjects were immediately put to sleep for a five-minute recuperation period.

HR measurements were performed using a 12-lead ECG. Following ten minutes of rest, the patients were asked to lie supine in order to acquire HR<sub>rest</sub>. HR measurements (such as HR<sub>1</sub>) were obtained at the conclusion of each minute during the Bruce test. HR<sub>submax</sub> was defined as the HR value at a predetermined sub-maximal aerobic activity (e.g., end of stage one - HR<sub>3</sub>, end of stage two - HR<sub>6</sub>). As a representative measure of the HR response during exercise, the percentage HR rise up to a certain minute ( $\Delta HR\%$ ) was computed as follows: Where  $\Delta HR_v = HR_v - HR_{rest}$ ,  $\Delta HR_v\% = (\Delta HR_v / HR_{rest}) \times 100$ . Furthermore, the percentage rise in heart rate ( $\Delta HR_{6-3}\%$ ) between the third and sixth minutes of exercise was calculated. The actual maximum HR (HR<sub>max</sub>) was defined as the highest HR attained at maximal effort. The age-predicted HR<sub>max</sub> was determined as 220 minus the participant's age. In order to account for age and HR<sub>rest</sub>, heart rate reserve (HRR) and the percentage of the age-predicted HRR achieved (HRR%) were used and computed as follows:  $HRR = HR_{max} - HR_{rest}$  and  $HRR\% = [HRR / (\text{age-predicted } HR_{max} - HR_{rest})] \times 100$ . [18] The HR recovery (HR<sub>rec</sub>) was recorded during the 5-min post-exercise period. Two indexes of HR<sub>rec</sub> were computed: 1) the HR difference between HR<sub>max</sub> and a given min of recovery,  $\Delta HR_{recv} = HR_{max} - HR_{recv}$ , as a measure for comparison with previously published data; and 2) the percentage HR decline until a given min of recovery,  $\Delta HR_{recv}\% = (\Delta HR_{recv} / HR_{max}) \times 100$ . For the purposes of the present study, HR values at rest, during sub-maximal exercise, at peak exercise, and during the first two minutes of recovery were used for comparisons between groups.

Statistical analysis of the data was performed using the IBM SPSS version 25.0 software package. Age, BMI, BP, HR values and maximal exercise test duration were presented as mean  $\pm$  standard deviation. Analysis of variance for personal, baseline and exercise data, and the chi-square test were used to examine possible differences between groups. Level of significance was p less than 0.05

## Results

Out of total 325 students 175 were male and 150 were female. In male category 130 were smoker and 45 were non smoker whereas in female category 45 were smoker and 105 were non smoker. Non-significant differences were found between smokers and non-smokers regarding age, height, weight, BMI and physical activity level in both sexes. In addition, non-significant differences were found for resting SBP and DBP between smokers and non-smokers of both sexes. However, smokers had significantly higher HR<sub>rest</sub> compared with non-smokers in both female (p=0.002) and male (p=0.003) students. The results of exercise time duration were also significant in female and male category as shown in table 1.

**Table 1 showing baseline characteristics of study population**

Variable	Male (175)		Female (150)	
	Smoker (130)	Non smoker (45)	Smoker (45)	Non smoker (105)
Age (years)	24.1 $\pm$ 1.8	23.2 $\pm$ 1.2	22.4 $\pm$ 1.5	22.2 $\pm$ 1.9
Height (cm)	177.3 $\pm$ 5.0	177.1 $\pm$ 4.8	167.2 $\pm$ 3.2	165.1 $\pm$ 3.1
Weight (kg)	70.3 $\pm$ 5.3	70.2 $\pm$ 4.7	58.3 $\pm$ 5.6	58.2 $\pm$ 3.4
BMI (kg/m <sup>2</sup> )	24.2 $\pm$ 1.5	24.1 $\pm$ 1.4	21.3 $\pm$ 1.8	20.9 $\pm$ 1.7
HR <sub>rest</sub> (bpm)*	70.1 $\pm$ 5.8	66.2 $\pm$ 5.7	75.8 $\pm$ 5.5	71.2 $\pm$ 4.2
SBP <sub>rest</sub> (mm/Hg)	124.7 $\pm$ 8.2	125.2 $\pm$ 7.8	117.2 $\pm$ 7.3	119.3 $\pm$ 8.4

DBPrest (mm/Hg)	77.3±6.5	76.4±6.7	73.1±6.5	73.2±6.4
Physical activity	Low	Low	Low	Low
IPAQ score	336	351	325	340
Exercise test duration (min)*	9.8±0.2	11.4±0.7	8.6±0.6	9.3±0.3

There were no discernible variations in HR<sub>submax</sub> between smokers and non-smokers in females. Nonetheless, compared to non-smokers, female smokers showed a noticeably smaller HR increase up until a specific minute of sub-maximal activity. Furthermore, for female smokers, the percentage HR increase from the third to the sixth minute of exercise was much lower. Furthermore, in female smokers, the rate of HR increase slowed down as exercise intensity rose. Their resting heart rate was higher than nonsmokers', but by the end of the second stage (6 minutes), this had decreased. While non-smokers attained their age-predicted HR<sub>max</sub>, female smokers did not. Smokers had a considerably reduced heart rate reserve ( $p < 0.001$ ) than non-smokers. Furthermore, smokers exhibited a considerably lower proportion of HRR attained (HRR%) ( $p < 0.001$ ). Female non-smokers attained 100.2% of their age-predicted HRR, compared to 95% for female smokers. During the first two minutes of recovery, female smokers' absolute HR reduction and percentage HR decline were both considerably lower than those of non-smokers. During the initial minute following physical activity, female smokers'  $\Delta\text{HRrec1\%}$  was 20.5%, whereas non-smokers' was 25.3% ( $p = 0.003$ ). During the second post-exercise minute, female smokers'  $\Delta\text{HRrec2\%}$  was 29.9%, while non-smokers' was 34.3% as shown in table 2.

**Table 2 showing analysis of the effects of smoking on heart rate values at rest, during sub-maximal exercise, at peak exercise and during recovery in young females**

Variable		Smoker	Non smoker	P value
Rest	HR <sub>rest</sub> (bpm)	75.8±5.5	71.2±4.2	0.001
Submax exercise	HR <sub>3</sub> (bpm)	131.2±7.2	130.4±7.7	0.324
	$\Delta\text{HR3\%}$ (%)	78.2±12.6	84.2±13.8	0.001
	HR <sub>6</sub> (bpm)	165.2±7.8	165.3±8.2	0.237
	$\Delta\text{HR6\%}$ (%)	117±16.2	138.1±17.1	0.001
	$\Delta\text{HR6-3\%}$ (%)	24.1±3.2	26.3±4.1	0.002
Peak exercise	HR <sub>max</sub> (bpm)	191.0±2.3	197.2±2.1	<0.001
	HRR (bpm)	115.2±6.2	129.5±5.3	<0.001
	HRR% (%)	96.1±2.1	101.3±1.5	<0.001
Recovery	$\Delta\text{HRrec1}$ (bpm)	40.1±6.8	51.2±6.5	<0.001
	$\Delta\text{HRrec1\%}$ (%)	21.3±3.2	24.3±3.1	0.003
	$\Delta\text{HRrec2}$ (bpm)	56.4±6.1	67.1±3.2	<0.001
	$\Delta\text{HRrec2\%}$ (%)	28.2±3.2	33.6±2.4	0.001

At the end of the third minute, male smokers' HR<sub>submax</sub> was considerably higher than that of non-smokers', but at the end of the sixth minute, the differences were not statistically significant. Compared to non-smokers, male smokers' heart rate increased less until the conclusion of the sixth minute of submaximal exercise. Furthermore, for male smokers, the percentage HR increase between the third and sixth minutes of exercise was much lower. In male smokers, the rate of HR increase slowed as exercise intensity rose. Their heart rate was higher than non-smokers' at rest, but it decreased in the third and sixth minutes. The age-predicted HR<sub>max</sub> for smokers and non-smokers was comparable. Nonetheless, when comparing male smokers to non-smokers, the real HR<sub>max</sub> was considerably lower ( $p < 0.001$ ) for smokers. Male smokers' heart rate reserve was noticeably lower than that of non-smokers, and they were unable to achieve their age-predicted HR<sub>max</sub>. In contrast to male non-

smokers, who attained 101.4% of their age-predicted HRR, male smokers only attained 97.2%.HR recovery values were not found to be significantly different between smokers and non-smokers in males as shown in table 3.

**Table 3 showing analysis of the effects of smoking on heart rate values at rest, during sub-maximal exercise, at peak exercise and during recovery in young males**

Variable		Smoker	Non smoker	P value
Rest	HRrest (bpm)	70.1±5.8	66.2±5.7	0.002
Submax exercise	HR3 (bpm)	127.3±6.7	120.3±7.6	0.003
	ΔHR3% (%)	76.3±13.6	85.4±13.2	0.126
	HR6 (bpm)	150.2±6.2	147.8±8.4	0.231
	ΔHR6% (%)	107.2±15.2	120.3±15.4	0.001
	ΔHR6-3% (%)	18.2±5.2	21.1±4.3	0.001
Peak exercise	HRmax (bpm)	192.3±2.1	199.7±3.0	<0.001
	HRR (bpm)	121.7±7.2	132.3±5.4	<0.001
	HRR% (%)	98.1±2.6	102.3±3.1	<0.001
Recovery	ΔHRrec1 (bpm)	40.2±6.1	51.2±6.3	0.137
	ΔHRrec1% (%)	21.3±2.4	25.4±2.5	0.264
	ΔHRrec2 (bpm)	59.2±8.6	68.2±6.3	0.351
	ΔHRrec2% (%)	31.2±3.2	35.4±2.7	0.287

## Discussion

The current study discovered that smoking had an impact on young adult smokers' resting and exercising heart rates in both male and female students. Smokers showed higher resting heart rates, a delayed increase in heart rate during physical activity, difficulty achieving their age-predicted maximum heart rate, and a lessened decrease in heart rate during recovery among female smokers.

Our study's findings that the HRrest of females was higher than that of males are consistent with the majority of studies' average HRrest differences between the sexes.[19,20] Additionally, our results show that young smokers, both male and female, have considerably greater HRrest than non-smokers. These findings are consistent with evidence from young populations that have already been published[21,22] and with several HR-related studies of middle-aged, healthy groups where smoking has been linked to higher resting HR values.[23,24] Both autonomic dysfunction and specific changes in cardiac autonomic regulation are linked to smoking. More precisely, smoking reduces vagal drive over time by acting on peripheral sympathetic sites, which raises catecholamine levels in the blood and enhances sympathetic outflow. Even in young heavy smokers, there is a sympathetic predominance that is linked to decreased baroreflex function and a significant rise in HRrest.[25]

According to our data, there were no differences in the sub-maximal HR values of young smokers and non-smokers, with the exception of male smokers' HR3 values, which were significantly higher. Studies on the impact of smoking on HRsubmax in young, healthy persons are scarce. The findings are contradictory; smoking was shown to raise men's heart rates at a fixed sub-maximal workload in certain studies [14], but there were other studies that suggested smokers had lower heart rates at sub-maximal exercise [26,27], and other research that found no differences in heart rates.[28]

When compared to non-smokers, smokers in the current study—both male and female—showed a noticeably slower HR increase during treadmill testing. Despite the fact that no subgroup under investigation showed signs of chronotropic incompetence, a notably higher proportion of smokers fell short of their age-predicted HRmax. Male smokers exhibited a lower HRmax, which is comparable to the difference between the smoking and non-smoking

groups in middle-aged, healthy men that Sandvik et al reported.[11] Our findings are consistent with several HR-related epidemiological studies conducted in middle-aged or younger, healthy populations, where smoking was consistently linked to a lower HRR, a lower HRmax attained, or an impeded HR rise during exercise.[29] Our data analysis revealed that female smokers had more noticeable impacts from smoking on  $\Delta HR\%$ , HRmax, HRR, and HRR%. These gender-specific results corroborate the notion that smoking may put young women at higher cardiovascular risk than it does men, as reported by Lauer and Sidney.[14,26]

No indications of aberrant HRrec were found in any of the subgroups that our study looked at. Young smokers of both sexes did, however, experience a lessened HR decrease during recovery; nevertheless, these alterations were only statistically significant for female smokers. Research on the relationship between smoking and HRrec in young adults is quite rare. Young smokers showed a lessened heart rate drop during submaximal exercise, according to Kobayashi et al[30] In our investigation, smoking was associated with a lower heart rate following maximal activity. On the other hand, smoking prevalence was considerably lower in the slower HRrecquartiles in the CARDIA trial.[31] The disparity between the results could be explained by the significant variations in the research designs of the studies that were just described, such as the workload intensity at the end of exercise (maximal vs. sub-maximal) and the recovery strategy (cool down vs. abrupt stop of activity). The size of the sample and the non-randomised design are the main limitations. Our target population consisted of healthy young higher education students. Socioeconomic status, smoking years, dietary habits, as well as other factors might differ from the general population which also serves as key factors.

### Conclusion

It has been discovered that smoking has an impact on young smokers' heart rates, which increases at rest, decreases during exercise, and hinders their capacity to reach the age-predicted HRmax. Furthermore, smoking was linked, albeit only in females, to a lessened HR fall during recovery. In terms of gender differences, women's resting heart rates were higher than men's, and they also displayed a greater HR response during sub-maximal activity.

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