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Hemoglobin Concentration and Hematocrit (PCV) in Male Adult Smokers

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Abstract

Background: Cigarette smoking is a common problem in Bangladesh and also a major public health problem associated with morbidity and mortality. The prevalence of cigarette smoking has peaked among the adults. Hemoglobin concentration and Hematocrit both are biomarkers of Cardiovascular and Cerebrovascular disease risk factor. **Objective:** The present study has done to find out the cross-sectional association of changes in Hemoglobin concentration and Hematocrit in relation to cigarette smoking and also with different intensity of cigarette smoking. **Methods:** The study population consisted of 105 male adult non-smokers and smokers, aged 20–40 years, of different socio-economic classes. 30 of them were apparently healthy non-smokers, taken as control group for the study, where as the remaining 75 apparently healthy smokers, who are smoking one or more cigarette per day, regularly for at least last one year, considered as the experimental group of this study. Smokers were again divided according to the number of cigarettes they consume per day into three categories. Smoking habit and clinical history & examination performed by pre-tested questionnaire. Unpaired "t" test was performed to find significant statistical difference between non-smokers and different smokers groups. **Results:** The hemoglobin concentration was significantly higher ($p < .01$) in smokers than non-smokers, but it did not significantly differ with intensity of smoking. PCV percentage of smokers was significantly ($p < .01$) higher than non-smokers, but it also did not significantly differ with intensity of smoking. **Conclusion:** The finding in this study suggests that cigarette smoking does changes the Hemoglobin concentration and Hematocrit which are biomarker of cardiovascular and cerebrovascular disease risk factor.

Key words: Cigarette Smoking, Hemoglobin, Hematocrit, Adult Male.

Introduction

Tobacco is the second major cause of death in the world. It is currently responsible for the death of one in ten adults' worldwide (about 5 million deaths each year). If current smoking patterns continue, it will cause some 10 million deaths each year by 2020. Half the people that smoke today – that is about 650 million people – will eventually be killed by tobacco.¹ The Expert Committee observed that tobacco related diseases are on the rise in developing countries.²

In 2003, the smoking prevalence in Bangladesh, among adults was 54.8% in males and 16.6% in females. The prevalence of smoking in age group 18-29 yrs, 30-39 yrs, 40-49yrs is 36.3%, 64.2%, and 70.8% respectively.³

Researchers have demonstrated that certain biomarkers of cardiovascular disease risk factors (eg, hemoglobin, hematocrit, RBC count, WBC count, mean corpuscular volume (MCV), Mean corpuscular hemoglobin (MCH), lipoproteins concentrations, heart rate, respiratory symptoms) known to be affected by smoking are relatively stable over time when amount of smoking is maintained at a constant rate but are altered in response to changes in smoking behavior. This suggests that some of these measures are likely to be sensitive to changes in tobacco toxin exposure.⁴ It is well established that an increased Hematocrit increases the risk of CVD and mortality from CVD⁵.

Other investigators have also found that at all ages; Erythrocyte counts of men are higher in

non-smokers than in smokers. Smokers have larger Erythrocytes than non-smokers in both sexes and at all ages.⁶ In both genders, in comparison to never smokers, smokers have higher values of mean corpuscular volume, hematocrit, hemoglobin, mean corpuscular hemoglobin, and platelet count.⁷

In a study it was found that low and high levels of Hematocrit are associated with increased mortality in the general population.⁸

In an another study it has been observed that, a rapid return towards normal was found in the elevated hemoglobin concentration, Hematocrit (PCV), red cell count, white cell count, neutrophil count, lymphocyte count and platelet count, indicating that at least some of the abnormalities in these parameters are an acute, reversible effect of cigarette smoking rather than being a response to tissue damage.⁹

In this study, we hypothesized that cigarette smoking can alter the Hemoglobin concentration and Hematocrit, which might be associated with intensity of smoking.

Materials and Methods

This is a cross sectional comparative study, which was carried out in the department of Physiology of the Dhaka Medical College from July 2005 to June 2006. Total 105 apparently healthy subjects age ranged from 20–40 years were selected, of whom 30 were non smokers (control – Group A) and 75 were smokers (experimental – Group B) who smoked for at least last one year. The smokers were again sub-grouped into 3 categories accordingly to the number of cigarette smoked per day (Group B₁: Consisted of 25 smokers consuming 1–9 cigarettes / day; Group B₂: Consisted of 25 smokers consuming 10–19 cigarettes/day; Group B₃: Consisted of 25 smokers consuming ≥ 20 cigarettes/day). They were selected from different areas of Dhaka city and different socio-economic classes of subjects were included in the study.

Subjects suffering from any acute or chronic respiratory illness, hypertension, diabetes mellitus, angina, endocrine, hepatic, allergic disorders, any infectious or debilitating illness etc. and subjects with history of recent hospitalization and surgery were also excluded. Persons taking the drugs such as antibiotic, steroids, thiazide diuretics, aspirin etc. or taking radiotherapy and subjects who drink alcohol were also excluded. Passive smokers were not included for control group.

All the subjects were explained about the aims and objectives of the study and the test procedure were briefed and written consent was taken before performing the test. A detailed history of smoking habit and health of each subject was obtained by using a pre-tested questioner and clinical examination sheet. Clinical examinations of these subjects were done on the first day of the visit. The subjects were advised overnight fasting and not to engage in unusual physical exercise and smoking before reporting on the next morning.

On the reporting day all the blood samples were collected at morning between 8.00 am to 9.00 am. With all aseptic (70% alcohol) precaution a venepuncture was done with a 3ml disposable syringe in the antecubital fossa applying a tourniquet, which was released when the actual blood sampling began. 2 ml venous blood was drawn from each sample, in sitting position. After collection the sample of blood was mixed in a vial containing anticoagulant- EDTA and transported to the Physiology laboratory of Dhaka Medical College in an air conditioned bus within one hour of collection. All the hematological analyses were done within 5 hours of sample collection.

With the collected samples Hemoglobin concentration was done by Cyanmethaemoglobin Method and Hematocrit was done by Macro (Spell's Wintrobe's) method for each study subjects in the laboratory of Physiology of Dhaka Medical College and Hospital, Dhaka.

After the collection of data these were checked, verified, edited for consistency to reduce error. All the results of laboratory investigations were analyzed by SPSS 12.0 programmer and significance tests were done by unpaired student's "t" test.

Results

Estimation of Hemoglobin

The results are shown in Table I A & B and Figure 2.

The means ($\pm$ SD) of Hemoglobin were 10.9 ± 1.26 gm/dl and 12.9 ± 3.27 gm/dl in group A and group B respectively. Again the means ($\pm$ SD) of Hemoglobin were 12.4 ± 2.99 , 12.5 ± 3.59 , 13.8 ± 3.14 gm/dl in B₁, B₂ and B₃ groups respectively.

The difference of means ($\pm$ SD) of Hemoglobin was significant ($p < .01$) between group A and B.

The results were also significant between group A and smoker group B₁ ($p < .05$), B₂ ($p < .05$), and B₃ ($p < .001$).

Again the differences of mean of Hemoglobin among the smokers groups B₁ vs. B₂ ($p > .05$), B₁ vs. B₃ ($p > .05$), and B₂ vs. B₃ ($p > .05$) were not statistically significant.

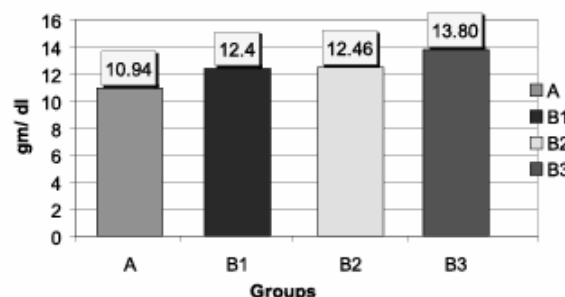
TABLE I A: Mean And $\pm$ sd of Hemoglobin Levels

Groups	N	Minimum gm/dl	Maximum gm/dl	Mean gm/dl	Std. Deviation
A	30	7.40	12.90	10.9400	± 1.25687
B	75	6.60	20.20	12.8840	± 3.26873
B ₁	25	8.80	20.20	12.4000	± 2.98761
B ₂	25	6.60	18.40	12.4600	± 3.59026
B ₃	25	7.80	18.40	13.7920	± 3.13660

Table I B: Statistical Analysis of Hemoglobin Levels between Different Groups:

Groups	"P" Value	Significance Level	Remark
A vs. B	.0021	$P < .01$	Significant
A vs. B ₁	.018	$P < .05$	Significant
A vs. B ₂	.035	$P < .05$	Significant
A vs. B ₃	.0001	$P > .001$	Significant
B ₂ vs. B ₃	.17	$P > .05$	Not Significant
B ₁ vs. B ₃	.11	$P > .05$	Not Significant
B ₁ vs. B ₂	.95	$P > .05$	Not Significant

Figure 2 : Mean ($\pm$ SD) of Hemoglobin levels in non-smoker's and smoker's groups.



Estimation of PCV

The results are shown in Table II A & B and Figure 3. The means (SD) of PCV were 40.6 4.34 and 45.0 7.38 percentage in group A (control-non smokers) and B (experimental-smokers) respectively. Again the means (SD) of PCV were 45.0 6.55, 47.5 8.30, 42.6 6.60 in B₁, B₂ and B₃ groups respectively.

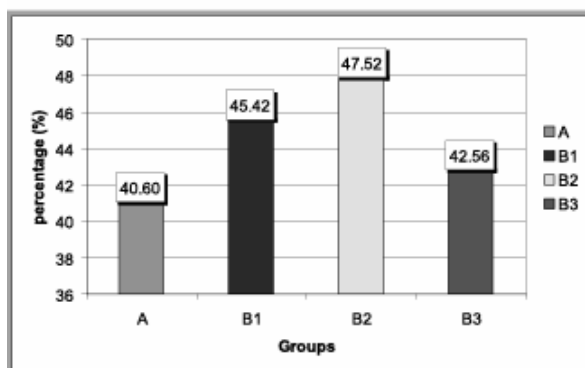
The difference of mean (SD) of PCV was significant ($p < .01$) between group A and B.

The results were also significant between group A and smoker's group B₁ ($p < .01$), B₂ ($p < .001$). But not statistically significant in between group A and B₃ ($p > .05$).

Again the differences of mean of PCV among the smokers groups B₁ vs. B₂ ($p > .05$), B₁ vs. B₃ ($p > .05$) were not statistically significant. But was significant between B₂ vs. B₃ ($p < .05$).

Table II A : Mean And $\pm$ Sd Of Pcv Percentages

Groups	N	Minimum (%)	Maximum (%)	Mean (%)	Std. Deviation
A	30	30.00	48.00	40.6000	± 4.34384
B	75	26.00	60.00	45.0133	± 7.38423
B ₁	25	34.00	60.00	44.9600	± 6.55413
B ₂	25	26.00	60.00	47.5200	± 8.29719
B ₃	25	35.00	54.00	42.5600	± 6.59596

Figure 3 : Mean ($\pm$ SD) of PCV levels in non-smoker's and smoker's groups.**Table-II B : Statistical Analysis of PCV Percentages Between Different Groups**

Groups	"P" Value	Significance Level	REMARK
A vs. B	.0028	$P < .01$	Significant
A vs. B ₁	.0047	$P < .01$	Significant
A vs. B ₂	.0002	$P < .001$	Significant
A vs. B ₃	.19	$P > .05$	Not Significant
B ₂ vs. B ₃	.024	$P < .05$	Significant
B ₁ vs. B ₃	.20	$P > .05$	Not Significant
B ₁ vs. B ₂	.23	$P > .05$	Not Significant

Discussion

The Hemoglobin concentration values given in Table-1A show a significantly higher ($p < .01$) in smokers than non-smokers. But it was not significantly ($P > .05$) differ with intensity of cigarette smoking per day. But the mean high hemoglobin level was estimated in the heavy smokers group. This result is in agreement with Paolo Boffetta⁸ et al., Van tile¹⁰ et al., Helman & Rubenstein¹¹, and Friedmen.¹²

The Table-II B shows that PCV percentage of smokers was significantly ($p < .01$) higher than non-smokers. This result is consistent with the finding of some of the studies.^{10,11,13,14}

Helman and Rubenstein¹¹ suggested that, the elevated Hb and Hematocrit in smokers could be interpreted as a compensatory response to chronic carbon monoxide-induced hypoxia. However, in all other instances of hypoxia-induced polycythemia, the erythron reacts by producing more erythrocytes, not bigger erythrocytes. Perhaps, then, it is a combination of effects. For example, carbon monoxide-induced hypoxia produces a demand for more erythrocytes. The demand cannot be met because of cyanide-induced diversion of vitamin B₁₂ away from nucleoprotein synthesis. However, there is no impairment of hemoglobin synthesis. The result would be little change in erythrocyte number but increases in erythrocyte size and hemoglobin content. The similar suggestions were also stated by Van tile¹⁰ et al.

In the present study we had some potential limitations which arise primarily due to lack of scope and resources, the tests could not be done on the spot of blood collection, which would give better results. The Hematocrit was done in traditional Macro (Spell's Wintrobe's) method rather than using an auto analyzer. Blood cells were more or less hemolysied during transport. The sample included in the study only on basis of questionnaire and clinical examination.

Conclusion

Cigarette smoking increase both the hemoglobin concentration and Hematocrit as a compensatory response to chronic carbon monoxide-induced hypoxia, which both are biomarkers for cardiovascular and cerebrovascular disease risk factors.

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Conflict of interest: none declared.

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