

## The effect of cigarette smoking in plasma fibrinogen level and platelet count in male Smokers in Nnewi, Anambra State, Nigeria

ONWURAH OW<sup>1,\*</sup>, EZE HT<sup>2</sup>, EZEAGWUNA DA<sup>3</sup>, NWACHUKWU EP<sup>4</sup>, EZE CG<sup>1</sup>, ONWUASUANYA UF<sup>5</sup> and UZOEWULU NG<sup>3</sup>

<sup>1</sup> Department of Hematology, Nnamdi Azikiwe University Teaching Hospital, Nnewi, Anambra State, Nigeria.

<sup>2</sup> Department of Internal Medicine, College of Health Sciences, Nnamdi Azikiwe University, Awka, Anambra State, Nigeria.

<sup>3</sup> Department of Microbiology & Parasitology, Nnamdi Azikiwe University Teaching Hospital Nnewi, Anambra State, Nigeria.

<sup>4</sup> Department of Chemical Pathology, Nnamdi Azikiwe University Teaching Hospital, Nnewi, Anambra State, Nigeria.

<sup>5</sup> Department of Medical Laboratory Science, Nnamdi Azikiwe University, Awka, Anambra State, Nigeria.

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### Abstract

Cigarette Smoking has been shown to induce hypercoagulability and a hyper thrombotic state, possibly by increased platelet aggregation and adhesiveness as a result of its nicotine content. During tissue and vascular injury, Fibrinogen (factor I) is converted enzymatically by thrombin to fibrin and subsequently to a fibrin-based blood clot. This study was aimed to determine the effect of cigarette smoking in plasma fibrinogen and platelet levels in male smoker in Nnewi, Anambra state. Apparently healthy 80 male cigarette smokers and 80 male non cigarette smokers, aged between 18 and 40 years, resident in Nnewi were recruited for this study. Four milliliters of blood were collected from each participants (test and control samples), 2ml in trisodium citrate anticoagulated tubes and 2ml in EDTA for the estimation of plasma fibrinogen levels and platelets count respectively. Fibrinogen was estimated using the method described by Claus. Platelets were estimated using Mythic 22 Automated Hematological Analyzers. The results showed a significant difference between the mean plasma Fibrinogen level in cigarette smokers and non-cigarette smokers ( $365.90 \pm 66.79$  vs  $203.02 \pm 47.40$ ;  $p = 0.001$ ). There was no significant difference observed in the mean platelets count of cigarette smokers when compared to the mean values of non-cigarette smokers ( $242.80 \pm 94.70$  VS  $202.84 \pm 76.31$ ;  $p = 0.564$ ). The study concluded that cigarettes smoking elicits an increase in the plasma fibrinogen level.

**Keywords:** Fibrinogen level; Platelet count; Cigarette smokers; Nnewi

### 1. Introduction

Fibrinogen (factor I) is a glycoprotein that circulates in the blood of vertebrates. During tissue and vascular injury it is converted enzymatically by thrombin to fibrin and subsequently to a fibrin-based blood clot. Fibrinogen functions primarily to occlude blood vessels and thereby stop excessive bleeding. However, fibrinogen's product, fibrin, binds and reduces the activity of thrombin [1]. Platelets, also called thrombocytes are a component of blood, which function along with the coagulation factors to arrest bleeding from blood vessel injury by clumping, thereby initiating a blood clot [2]. Platelets have no cell nucleus: they are fragments of cytoplasm that are derived from the megakaryocytes of the bone marrow, and then enter the circulation as a biconvex discoid structures with 2–3  $\mu\text{m}$  in diameter [3].

\*Corresponding author: ONWURAH OW

Department of Haematology, Nnamdi Azikiwe University Teaching Hospital, Nnewi, Anambra State, Nigeria.

Cigarette smoking is one of the largest causes of preventable death worldwide. It is the leading cause of coronary artery disease [4]. Smoking is an independent risk factor for renal failure, it increases heart rate, blood pressure, blood clot formation and promotes fatty deposit in the arteries. Compared to non-smokers, smokers have an increased risk of having protein in urine twice more and likely to develop kidney cancer. Smoking has been reported to exert a significant effect on almost all the hematological parameters including hematocrit, plasma fibrinogen, hemoglobin, red blood cell (RBC) count and white blood cell (WBC) count with several deleterious effects on the properties of blood [4,5]. Carboxyhemoglobin and plasma viscosity are raised by smoking, and cardiac output is decreased [5].

Cigarette smoke contains a variety of oxidants and free radicals that has been shown to induce hypercoagulability and a hyper thrombotic state, also causing platelet aggregation and adhesiveness as a result of its nicotine content which is capable of initiating or promoting oxidative damage leading to various degenerative pulmonary and cardiovascular diseases as well as cancer<sup>4,5</sup>. Predictors such as age, duration and the average number of cigarette stick smoked per day are established factors for assessing the absolute risk of developing smoke-related complications in long-term smokers<sup>4</sup>.

## 2. Material and methods

A total of 200 apparently healthy male, within the age range of 18-40 years were randomly recruited for this study in Nnewi metropolis. One hundred (100) were cigarettes smokers (one to three sticks per day); while 100 were non cigarettes smokers within the same age range to serve as control. The ethical approval for this study was sought and obtained. Four milliliters of blood were collected from the antecubital vein of each participant (test and control samples), 2ml in Ethyenediamine tetra acetic acid (EDTA) bottles and 2ml in trisodium citrate anticoagulated tubes for the estimation of platelets and levels of plasma fibrinogen respectively. The samples collected were processed within 4 hours of blood collection. Fibrinogen was estimated according to the method described by von Clauss and result reported in milligram per deciliter (mg/dl). Platelets were estimated using Mythic 22 Automated Hematological Analyzers (USA) and the values obtained as platelets per micro liter. The data obtained was analysed using Statistical Package for Social Sciences (SPSS) (Version 23) software. This was presented as mean±SD and the results compared using Students paired sample t-test and Pearson r correlation at 95% CI (P<0.05).

## 3. Results

Mean fibrinogen values (mg/dl) of 365.90±66.79 was obtained for the smokers while the mean fibrinogen value for the non-smokers was 203.02±47.40. There was a significant difference between the mean plasma Fibrinogen level of cigarette smokers and non-cigarettes smokers (365.90±66.79 vs 203.02±47.40: p= 0.001) (Table 1)

There was no significant difference between the mean plasma fibrinogen level of those smoking one cigarette stick (219.11±10.34) and those smoking two sticks of cigarette (265.45±26.56) per day (Table 2). Significant association was observed in the mean plasma fibrinogen level between those men that smoked one stick of cigarette and three sticks of cigarette per day (219.11±10.34 vs 293.54±22.10: p=0.001) (Table 3)

**Table 1** Comparison between fibrinogen levels of smokers and non-smokers

Sample ID	N	Unit	Mean ±STD	t	P
Smokers	100	mg/dl	365.90±66.79	3.702	0.001**
Non smokers	100	mg/dl	203.02±47.40		

(P<0.05)

**Table 2** Comparison between fibrinogen levels of smoking one stick and smoking two sticks

Sample ID	N	Unit	Mean ±STD	t	P
<b>One stick</b>					
Smokers	34	mg/dl	219.11±10.34	5.102	0.501
<b>Two sticks</b>					
smokers	35	mg/dl	265.45±26.56		

(P<0.05)

The mean Platelet count (ul) for smokers was  $242.80 \pm 94.70$  and the control had a mean platelet count of  $204.80 \pm 76.31$ . There was no significant difference in the mean platelets count of cigarettes smokers when compared to the mean values of non-cigarettes smokers ( $242.80 \pm 94.70$  VS  $204.84 \pm 76.31$ ;  $p = 0.564$ ) (Table 4).

**Table 3** Comparison between fibrinogen levels of one stick smoker and three sticks smokers

Sample ID	N	Unit	Mean $\pm$ STD	t	P
<b>One stick</b>					
Smokers	34	mg/dl	$219.11 \pm 10.34$	3.222	0.001**
<b>Three sticks</b>					
smokers	31	mg/dl	$293.54 \pm 22.10$		

( $P < 0.05$ )

**Table 4** Comparison between platelet count of smokers and non- smokers

Sample ID	N	Unit	Mean $\pm$ STD	t	p
Smokers	100	$100 \times 10^9/l$	$242.80 \pm 94.70$	0.579	0.564
Non smokers	100	$100 \times 10^9/l$	$204.80 \pm 76.31$		

#### 4. Discussion

Smoking has been reported to exert a significant effect on almost all the hematological parameters, thereby affecting most organs of the body<sup>4, 5</sup>. In this study, plasma fibrinogen level and platelet count among cigarette smokers and nonsmokers were determined and there was a significant association between the mean plasma fibrinogen in the cigarette smokers and non-cigarette smokers ( $365.90 \pm 66.79$  VS  $203.02 \pm 47.40$ ;  $p = 0.001$ ). This implies that cigarettes elicit an increase in the plasma fibrinogen, which increase the risk of blood clots, which can, in turn, contribute to an increased risk of heart disease. High fibrinogen is associated with higher rates of heart disease, blood vessel dysfunction, and stroke. Similar studies documented also showed significant increase in plasma fibrinogen in smokers [6,7,8], where high platelet and fibrinogen levels were recorded in their findings.

The findings in this study indicates only a rise in the plasma fibrinogen level and no significant effect of cigarette smoking on mean platelets counts of smoker when compared with mean platelets counts of nonsmokers ( $242.80 \pm 94.70$  VS  $202.84 \pm 76.31$ ;  $p = 0.564$ ), despite the platelets count increase in smokers. Some authors [9,10], in their findings, reported no significant difference in platelets counts of smokers and nonsmokers. A study carried out by Ghahremanfard et al, [11] observed that, the mean platelet count was significantly higher in smokers than in non-smokers, in contrast to Metta S et al, [12] in 2015 that reported that, platelet count was significantly lower in chronic smokers. This study observed positive correlations among the cigarette's smokers in their plasma Fibrinogen and Platelets count, which implies that as plasma fibrinogen increases in cigarettes smokers, their platelets count also increased.

#### 5. Conclusion

The study showed that cigarettes smokers have increased fibrinogen level in their plasma than the non-cigarettes smokers. Since cigarette smoking is a strong risk factor for many diseases such as cancer, cardiovascular disease, renal disease, chronic obstructive pulmonary disease, atherosclerosis to mention but a few, the clinicians should take note of this when prescribing drugs and requesting for laboratory investigations for patients who are smokers of cigarette irrespective of number of cigarette sticks.

#### Compliance with ethical standards

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### *Disclosure of conflict of interest*

There are no conflicts of interest or financial support related to this study.

### *Ethical Approval*

The ethical approval for this study was sought and obtained from the Ethical Committee of Faculty of Health Sciences and Technology, Nnamdi Azikiwe University, Nnewi, Anambra State, Nigeria.

### *Statement of informed consent*

Informed consent was obtained from the subjects before they participated in the study.

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