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Cadmium Status and Activities of Cardiac Marker Enzymes in Smokers: Using Experimental Animals Model

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ABSTRACT

This study was designed to determine blood cadmium concentration and the activities of selected cardiac marker enzymes in two different groups of smokers, using experimental animal model. Twelve wiener rabbits were randomly distributed into 3 groups of 4 animals each. Groups I and II animals were exposed to smoke from two different brands of cigarette commonly consume in Nigeria for a period of three weeks. Each animal in the test groups was treated with six sticks of cigarette per day in an inhalation chamber. Group III animals served as the controls were exposed to normal air. At the end of the test period, the animals were sacrificed. The blood samples were collected and blood Cadmium concentration (bCd) were determined by Atomic Absorption Spectrophotometry, activity of Lactate Dehydrogenase (LDH) and Creatinine Kinase (CPK) were analysed in the plasma by standard colorimetric technique. The lungs of the animal were also excised for histological studies. The results obtained revealed an increase in the mean activity of LDH and CPK in the test groups when compared with the control. There is significant increase in the mean concentrations of LDH and CPK between Group II and controls. ($P \leq 0.05$), but the increase is not significant between group I and controls. Also, the histological pictures of the test animals revealed evidence of lungs pathology. This results suggesting that cigarette smoking has associated health hazards, and thus added to the avalanche of overwhelming evidences against cigarette smoking. However this is not clearly manifested in the activities of the enzymes associated with cardiac functions.

Key words: Cigarette, Cardiac enzymes, Smokers and Cadmium.

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INTRODUCTION

Cigarette smoking is the largest preventable risk factor for morbidity and mortality in developed countries (Peto et al., 1992). From the late 15th century until about 1860, tobacco was widely thought to be a panacea for some medical problems as it was accepted widely as having medical therapeutic value. During this period, some people believed that it was literarily possible to breathe life into another, as long as that breath carried tobacco smoke. For this reason, African natives would trade land, livestock and slaves for tobacco (Blum, 1984). However, today, the story has changed as there are now overwhelming scientific evidence that Cigarette smoking, hereafter also referred to as “smoking,” is the largest single risk factor for premature death in developed countries (Peto et al., 1992). According to WHO statistics, every eight seconds now, someone somewhere in the world dies from preventable tobacco-related diseases (Akinbode, 2003). Tobacco epidermics now claims over 5 million lives every year, yet, the figure is rising (Akinbode, 2003). Tobacco in cigarettes, cigars and pipe mixtures produce smoke that contains an array of physiologically active chemicals.

The smoke can be described on the basis of two phases, or components (Hahn, 1957). The particulate and gaseous phases. The particulate phase includes waistline, nicotine, water and a variety of powerful chemical compounds collectively known as tar which includes phenol, cresols, pyrene, DDT, benzo(a)pyrene etc. (Hahn, 1997, Denissenko et al, 1996). The gaseous on the other phase is composed of CO, CO_2, NH_3, HCN, isopyrene, acetaldehyde and acetone. Most of these have been found to be carcinogenic (Hahn, 1997). Carbon monoxide is one of the most harmful components of tobacco smoke (Hahn, 1997). It is produced as a result of incomplete oxidation in burning tobacco. CO possesses a very strong physiological attraction for haemoglobin to form carboxyhaemoglobin. In this form, haemoglobin is unable to transport oxygen to the tissues and cells where it is needed (Kapier, 1996). High levels of carboxyhaemoglobin in heavy smokers are also associated with significant rises in the incidence of myocardial infarction (Hahn, 1997). And myocardial infarction has been implicated as the leading cause of death from cardiovascular diseases (Kannel, 1981; American Heart Association, 1997; Peto et al., 1992 Centers for Disease Control and Prevention, 2005). Also, Nicotine, a major constituent of cigarette smoke has been linked to the development of angina pectoris (Kapier, 1996), sudden heart attack (Kapier, 1996; AHA, 1997).

There is also irrefutable evidence linking cigarette smoking to cancer of various sites. Approximately one fifth of the deaths in the United States are attributable to smoking, and 28% of the smoking-attributable deaths involve lung cancer, 37% involve vascular disease, and 26% involve other respiratory diseases (Peto et al., 1992; Travis, 1995). The National Cancer Institute's ,1996 also described over 43 carcinogens in cigarette smoke to include Polyaromatic Hydrocarbons, heterocyclic hydrocarbons, N-nitosamines , aromatic amines , aldehydes, inorganic compounds and radioactive elements. Furthermore, studies have shown that smoking is the primary risk factor for accelerated decline in respiratory functions (Crowley, 1996). Also in all its dimensions, the reproductive process has been found to be impaired by the use of tobacco; problems can be found in association with infertility (Kapier, 1996), ectopic pregnancy (Kahn, 1994), breast feeding (Kapier, 1996), health of the fetus (Eliopoulos, 1994), health of the neonate (Eissen, 1993; Kapier, 1996), and the early onset of menopause (Kapier, 1996). Hatcher, 1992 has also found that women who both smoke and use oral contraceptives are four times more likely to die from myocardial infarction than women who only smoke. Tobacco smoking has also been implicated as the most important source of cadmium exposure in the general population. The absorption of Cd from the lungs has been found to be much more effective than that from the gut, and as much as 50% of the Cd inhaled via cigarette smoke may be absorbed (United State Department of Health and Human Services, 1986; Day, 1993; Travis, 1995). The risks of involuntary (passive smoking) have also been documented. Current scientific opinions suggest that smokers and no smokers are exposed to very much the same smoke when tobacco is used within a common airspace. In the opinion of Glantz and Parmely (1991), it is likely that for each pack of cigarettes smoked by a smoker, non-smokers who must share a common air supply with them will involuntarily smoke the equivalent of 5 cigarettes per day. Scientific studies have suggested that non-smokers who are married to smokers are 3 times more likely to experience heart attack than non-smoking spouses of non-smokers (Overpeck and Morris, 1988), and they have 30% greater risk of lung cancer (Glantz and Parmely, 1991). Furthermore, the children of smokers are twice more likely to experience bronchitis or pneumonia, wheezing, coughing and sputum production than children of non-smokers (Overpeck et al., 1988). Against this backdrop of avalanche of evidences on the morbidity of Cigarette smoking, this study, therefore is to assess the level of Cd- which is the major heavy metal found in cigarette smoke; the activities of Cardiac enzymes (that is, Lactate dehydrogenase and Creatinine kinase) which are pointers to incidence of myocardial infarction; and possibility of lungs pathology in smokers using experimental animals model.

MATERIALS AND METHODS

Twelve (12) weaner rabbits of New Zealand breed weighing between 1050 to 1350 g were used for the study. They were made to acclimatise for 6 weeks, after which they were randomly grouped into 3 groups of four rabbits each. They were fed with fresh forage (Tridax procumbes) and compounded feeds. Group I animals were induced with smoke from Brand 1cigarette while group II animals were induced with Brand 2 cigarette in an inhalation chamber. The group III animals were left as controls without any contact with cigarette smoke. The test animals (groups I and II) were transferred one at a time into the inhalation chamber, subjecting them to smoke from 2 sticks of cigarette each time. This was repeated thrice per day for each test animal, thus subjecting each animal to 6 sticks of cigarette per day for 3 weeks. After the test period, the animals were sacrificed. About 5 ml of whole blood samples were obtained from each animal, using pyrogen-free needles and syringes (Becton-Dickson, Dublin Ireland). The blood samples were then dispensed separately into plain containers tubes containing EDTA (Sterilin Manufacturing Company, England). They were kept frozen at -70^0 C prior to analyses. The lungs were also removed and transferred into containers containing 10% formalin prior to histological examination.

Table 1. Cd, CPK and LDH values for Groups I, II and control.

Parameter	Control	Group I	Group I I
Cd (mg/mL)	0.06± 0.08	0.04 ± 0.02	0.06 ± 0.03
LDH (U/mL)	8.45 ± 3.69	10.55 ± 3.50	15.93 ± 4.9^{S}
CPK (U/mL)	1.43 ± 0.28	1.83 ± 0.34	2.23 ± 0.33^{S}

Values represent mean ± SD $^{S}P < 0.05$ significant, Cd = Cadmium concentration, LDH = Lactate Dehydrogenase, CPK = Creatinine kinase.

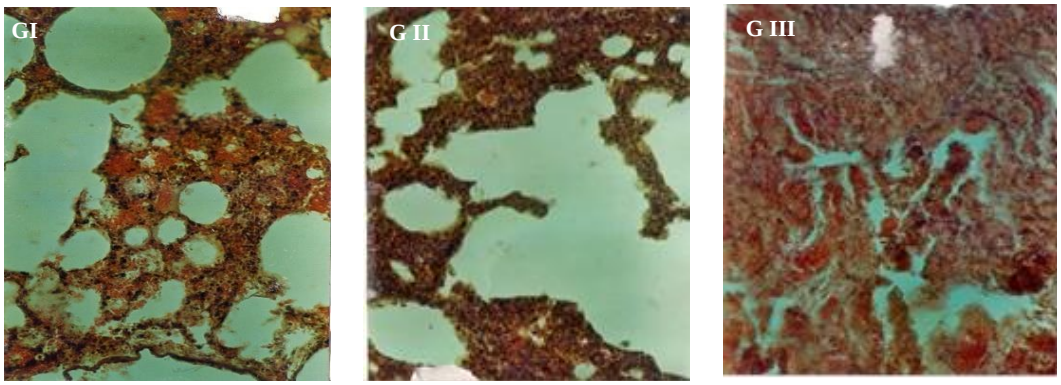

Figure 1. Histological Pictures of the lung of animals in the three groups.**G I**- Histological picture of the lungs of group I animals (Brand 1). **G II**- Histological picture of the lungs of group II animals (Brand 2).**G III**- Histological picture of the lungs of group III animals (Control).

Biochemical Analysis

The blood Cd concentration was determined by Atomic Absorption Spectrophotometric (AAS) method at 228 nm. The activities of CPK and LDH status determinations were carried out using Colorimetric method (Taussky, 1956).

Histological Examination

The histology of the lungs was examined through the aid of electron photomicroscopy of the cut samples which were previously stained with hematoxylin-eosin. This was carried out at the Pathology Department of the University College Hospital (UCH), Ibadan.

Statistical Analysis

Values of biochemical parameters were expressed as Mean ± SD. The statistical analysis was carried out using Student t test. Comparisons were made between different Groups, values < 0.05 were considered as significant.

RESULTS AND DISCUSSION

The results obtained in the study are as presented in Table 1 and Figure 1. Table 1 shows the results of cadmium concentration and the activities of the lactate dehydrogenase and creatinine kinase in the control and the two test groups. There is no significant difference between these values in all the three groups in respect of the cadmium concentration, there is also no significant difference between the activity of lactate dehydrogenase and creatinine kinase between the control and Group I but there is a significant different for these parameters between the control and Group II.

CPK and LDH assays in plasma are widely used in the investigation of myocardial infarction. In general, the higher and the longer the rise in plasma enzymes activities, the greater the extent of myocardial damage, and usually the poorer the prognosis (Hahn, 1997). The results of this study revealed that are increased activities of LDH and CPK between the control and the test groups however these increase is not significant the group induced with Brand 1 (Group I) and that of the control (Table 1), but revealed a significant difference between the LDH and CPK status of the group induced with Brand 2 (group II) to that of the control ($P < 0.05$) (Table 1).

There is no significance difference between the values of all the parameters (Cd, CPK and LDH) for groups I and II. But generally, when compared to the control, there were marked increments in the mean concentrations of the enzymes in the test groups, while that of Cd concentration did not appreciably change between the three groups (Table 1). The inference from the Cd status of the three groups is that it is probable that it takes much longer time before Cd could bioaccumulate in the

biological specimen (that is, blood) of smokers as reflected in this animal's model. The histological examination of the lungs (Figure 1) revealed very obvious morphological changes in the test groups as there were basophilic striping of the erythrocytes (pictures GI and GII), This change was more pronounced in G II animals, the contol groups however appears normal. This observation is in tandem with the results from the other parameters assayed. It is not however clear, the factors responsible for this observed higher damage in the lungs, as well as higher values of the parameters in group II animals as compared to group I, despite the fact that both brands of cigarette contain similar content (tar and nicotine) at same concentrations.

The answer probably lies in the variations in the concentrations of the over 1000 chemically active compounds that make up the tars. Conclusively from the results, it is evident that smoking is really a menace to health as it could inflict some pathological damage to the lungs, and increase the activities of cardiac enzymes in smokers. The pathological consequence of cigarette smoking on the heart is not clearly established in this study

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