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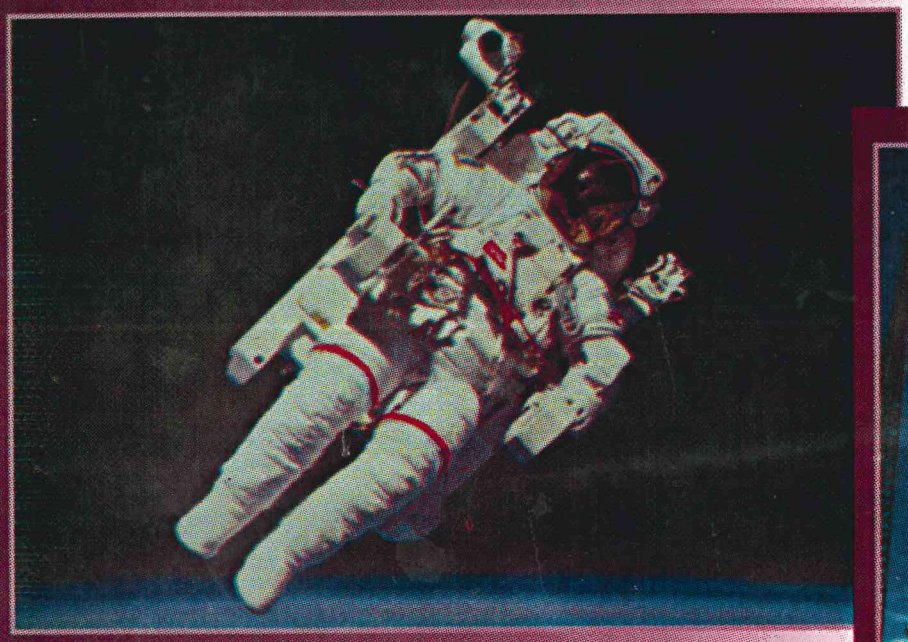

THEME:

THE ROLE OF SCIENCE, TECHNOLOGY AND MATHEMATICS IN THE ATTAINMENT OF MILLENNIUM DEVELOPMENT GOALS

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Evaluation Of The Serum Cholesterol, Cadmium And Urine Cadmium Levels In Smokers

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Abstract
The prevalent deaths from heart diseases resulting from smoking prompted this study to determine the serum cholesterol and cadmium in smokers. Higher levels of serum cholesterol (3.22 ±0.18 mmol/L) were obtained in smokers than in the non smokers (2.68 ±0.11 mmol/L). Also serum cadmium was higher in smokers (1.32± 0.11 mmol/L) and 0.13 ±0.04µl/L) for non smokers. Urine cadmium was 0.89±0.05mmol/L for smokers and 0.87 ±0.03 µl /L for non smokers. Duration and frequency of smoking also had an elevating effect on the serum levels of cholesterol, and cadmium as well as on the urine cadmium level. In all the samples analysed, the cadmium level did not exceed the WHO upper acceptable limit of 2µl/L.
Keywords: cholesterol, cadmium, smoking, serum, urine

Introduction

Smoking is associated with an increased overall morbidity and mortality. Smoking of tobacco as cigarette is a global obsession and remains the number one cause of preventable deaths in the world today (Newcomb *et al*, 1992). Smoking is one of the major risk factors for cardiovascular disease, coronary artery disease cerebro-vascular disease, peripheral vascular disease and pulmonary or respiratory functions are also affected by smoking (Mc Bride, 1992 and Sherma, 1992); vision impairment through cataract and macular degeneration (Johnston and Gregory, 2001); reproduction (Dechanet *et al*, 2010). Others include multiple complications in pregnancy, peptic ulcer, osteoporosis and bone fracture. Long term cigarette smoking has an elevating effect on the cholesterol level. Cholesterol, a precursor for some hormones and also responsible for the flexibility of cell membrane increases the risk of arteriosclerosis with increased risk of coronary artery disease or stroke (Charaberlain *et al*, 1988). Seven out of every ten death due to heart disease are associated with high blood cholesterol, which may begin to stick to the walls of the blood vessels getting the blood vessels clogged overtime and slowing down the flow of blood which may result into heart attack and stroke. The lecithin acyltransferase (LCAT) as a key factor in the esterification of plasma cholesterol has a lower activity in smokers than in non smokers (Bielicki *et al*, 2001). Smoking also affects the high density lipoprotein (HDL) and the low density lipoprotein (LDL) esters levels (Imamura *et al*, 2001). Second hand smokers also share the same problems as direct smokers (Hej, 1999). Cadmium (Cd) an environmental toxicant is found in food and in tobacco *smoke*. Although the cadmium content of cigarette is much lower than that of food, cadmium in cigarette is however absorbed much more effectively by the lungs than the gut (Yadav, 2010). Smoking is a major source of exposure to cadmium in those who are not occupationally exposed to cadmium. Smoking creates an accumulation of cadmium in organs and severe anemia is associated with cadmium toxicity. Cadmium health implications includes coronary artery disease. High blood concentration of cadmium leads to an increase in the lipid peroxidation and changes in the lipid metabolism (Kleszczewska *et al,* 2005). Cd inhibits progesterone secretion in cultured human. This study was embarked upon to determine the cholesterol and cadmium levels in smokers and non smokers.

Material and Methods

Blood sample

Thirty two apparently healthy smokers and thirty two non smokers between the ages of 18- 36 years were used as test samples and control respectively. Using a disposable syringe 4ml of blood was drawn from the vein of the upper arms of each individual into an EDTA tube and serum separated into a cryo tube and preserved in an ice pack until required.

Urine Sample

Sterile polythene containers were given to each individual a day prior to collect the first morning urine sample. The samples were stored in the refrigerator until needed for testing.

Determination of Serum Cholesterol

Cholesterol kit with pack code 20050817 44c0121 product of DIALAB A-2351 Weiner Neulorf Austrailia was used for the serum cholesterol determination. The kit is made up of Good's Buffer pH 6.7, phenol, 4-amino antitipyrine, cholesterol esterase, cholesterol oxidase and peroxidise. To each test tube labelled blank, standard, and sample was added 100ul of reagent, while 10µl of distilled water was added to the blank and 10µl of standard to the standard tube and 10µl of serum to each test tube. These were mixed and incubated at 37°C for ten minutes. The absorbance of standard and the test samples were taken against a working reagent at 540 nm using the spectronic-20D spectrometer.

Determination of Serum Cadmium

The method of Shozo, (1982) was employed. 9ml of nitric acid, sulphuric acid and perchloric acid mixture (3, 1, 2) v/v/v to 1ml of plasma sample was heated until clear. The mixture was filtered through whatman No.1 filter paper and made up to the 50ml mark with distilled deionized water. The cadmium concentration was determined by the atomic absorption spectrophotometer.

Determination of Urine Cadmium

5ml of the urine sample was subjected to wet digestion with 20ml of concentrated nitric acid and heated for 2hours at 150°C, cooled and filtered using whatman No.1 filter paper and made up to 100ml mark. Cadmium standard was prepared from cadmium chloride trihydrate. The cadmium concentration was determined using the atomic absorption spectrophotometer as described by Shozo, 1982.

Results and Discussion

A slightly higher level of cholesterol was observed in the smokers compared to the non smokers (Table 1). This is in contrast to the finding of Cerami, (2000), who reported a decrease in the cholesterol level in smokers, this may be based on the knowledge that most smokers rarely feed well as smoking have a suppressing effect on the appetite for food. Also physical exercise have a lowering effect on the cholesterol level, in that the muscle cells can use up cholesterol as energy source in the muscle cells than store it in fat cells. Blood cholesterol is influenced by diet, heredity, metabolic diseases (diabetes mellitus), smoking etc (Kimokoti *et al*, 2010). The smokers in this study however being construction site workers are noted to be heavy feeders on cheap saturated fat diets, which allow for the blood cholesterol level to be slightly elevated, despite their high physical activity. John *et al*, (1993) and Burton and Kazantia (1988) suggested an increase in low density cholesterol (LDL) which leads to arterial disease and a decrease in high density cholesterol (HDL) among smokers. Smoking lowers the activity of the lipoprotein lipase enzyme, responsible for processing of chylomicrons and overall metabolism, leads to

lower HDL (Harley and Smith, 1993). This is in agreement with the comparison in figure 1 on the level of cholesterol in relation to age and duration of smoking. Higher cholesterol was observed in the age range between 18 and 24, while lower cholesterol was observed for the 25- 31 and 32 -38 age ranges. Most of the older smokers have been smoking for long time duration. Sherma *et al*, 1997 demonstrated plasma cholesterol in the early stage followed by a fall at the later stage. This could be due to the effect of nicotine which increases the metabolic rate in smokers. Smoking does not appear to permanently affect cholesterol levels, once smoking is stopped, the effect of smoking on cholesterol stops within a few weeks or months (Michael, *et al*, 2004).

The plasma cadmium level in Table 1 showed values that are in the normal range. An increase in the plasma cadmium was observed for the different age groups, higher level of plasma cadmium was observed for the smokers within the 32-38 age group and the least in the 18-24 age group. This agrees with the findings of Shima et al, 1996 who reported an increase in plasma cadmium level in older smokers; hence duration of smoking is likely to increase the cadmium level in plasma (Elinder *et al*, 1983 and Jarup *et al*, 1998). *Smoking* is associated with the accumulation of *cadmium* in tissues. Lungs and prostate cancer in
Smokers (Anetor et al, 2010). The studied parameters in smokers did not differ significantly from non-smokers.

Table 1: Serum Cholesterol, Serum Cadmium and Urine cadmium

Parameter	Serum Cholesterol (mmol/L)	Serum Cadmium (ug/L)	Urine Cadmium
Smokers	3.22 ±0.18	1.32± 0.11	0.89±0.05
Non Smokers	2.68 ±0.11	0.125 ±0.04	0.87 ±0.03

Table 2: Serum Cadmium and occurrence frequency in tobacco smokers.

Cadmium in Serum (µg/l)	Cadmium in Urine (µg/L)	Frequency	Duration of Smoking (Years)
0.1±0.01	0.4±0.03	7	2
0.30±0.02	0.62±0.03	5	4
0.50±0.02	0.9±0.04	6	6
0.70±0.03	0.96±0.04	4	8
0.80±0.03	1.12±0.05	3	10
1.00±0.01	1.10±0.05	2	12
1.1±0.01	0.52±0.08	2	14
1.5±0.02	1.55±0.09	1	20

Reference

Anetor, JI., Ajose, F., Anetor, GO., Iyanda, AA., Babalola, OO. And Adeniyi FA. (2010). High cadmium and Zinc ratio in cigarette: Potential implications as a biomarker of risk prosrate cancer. Nigerian Journal of Physiological Science, 23: 41-49.

Bielicki, JK., Knoff, LJ. Tribble, DL and Forte, TM. (2001). Relative sensitivities of plasma lecithin: cholesterol acyltransferase, platelet-activating factor acetylhydrolase, and paraoxonase to in vitro gas-phase cigarette smoke. *Journal of Arteriosclerosis* **155**, Issue 1, Pages 71-78

Burton BG. and Kazantzis (1988). Prostrate cancer and Chronic respiratory and renal disease in British Cadmium workers: A case control study. *British Journal of Industrial Medicine* 42; 540-545.

Cerami A (2000). Tobacco smoke as a source of toxic reaction glycation products' proceedings of national academy of Sciences, USA (PNAS). **94 (25)**: 3915-20.

Chamberlain WJ, Schlotzhaner WS and Chortyk Ot, (1988). Chemical composition of non- smoking tobacco products. *Journal on Agricultural Food chemistry* **36**: 48-50.

Diricam P and Micheal T (1998): Cigarrete smoking in individual with schizophrenia,

Medscope sychiatria aand mental health **15(12)**: 873-879.

Elinder CG, Lind B and Kjellstrom T (1983). Cadmium in kidney cortex, liver and pancreas from Swedish autopsies: estimation of biological half time in kidney cortex considering calorie intake and smoking habits. *Achives Environmental Health* **31 (6)**: 292-302.

Harley KJ and Smith TJ (1993). Cadmium inhalation exposure estimates; their significance with respect to kidney and liver cadmium burden. *Journal of Toxicol. Environmental health* **15:** 173- 187.

Hej R (1999): Opoids induce whike nicotine suppress apoptosis in human lung cancer cells,

cell growth and differentiation. *The Molecular Biology Journal of the American association of cancer research* **5(10)**: 1033-40.

Immamura R, Grisso J and Hirshinger N (2001): Cocaine and tobacco use and risk of spontaneous Abortion. *New England Journal of Medicine* **340**:333-339.

Jarup L , Berglund M, Elinder CG, Nordberg G and vahter M (1998) Health Effects of cadmium exposure a review of Literature and a risk estimate. *Environmental Health* **24** (suppl,) 1; 1-51.

John E, Jusy AA, Julian RT, Menon CR and Muir DCF (1993). Biological monitoring of Ontario cadmium workers: Determination of whole blood and urinary cadmium levels. International Meetings on Molecular Mechanism of metal Toxicity and carcinogenicity Collegio Universitario Urbino. Italy. Pp 19-22

Johnston, M and Gregory, A (2001). **Smoking linked to blindness . The New Zealand Herald's Report Oct. 26.**

Kimokoti, RW, Newby, PK Gona, P, L , **Gona. P,** Zhu **L, Jasuja, GK. Pencina, MJ, McKeon-O'Malley, C, Fox, CS . D'Agostino, RB and. Millen, BE** (2010). Diet Quality, Physical Activity, Smoking Status, and Weight Fluctuation Are Associated with Weight Change in Women and Men. *Journal of Nutrition*, **140,** No. 7, 1287-1293.

Kleszczewska E, Lisowski P, Kleszczewski T, Adamczuk A, Trzciński R (2005). Evaluation test of cadmium concentration influence on selected lipid metabolism parameters in smokers with stable coronary artery disease scheduled for coronary artery bypass grafting (CABG) Przegl Lek .62(10):1051-3

Mcbride J, Amery A, Bernard a Bruanx P, Bulpitt CJ, Claeys F De Plaen P Ducoffre G, Fagard R,nLauwerys RR, Lijncen P, Nick I, saint Remy A, Roels H, Rondia D,

Sartor F and Thijs I (1991): Blood pressure, the prevalence of cardiovascular diseases and exposure to cadmium: a population study. *American Journal of Epidimology* **134**:257-267.

Micheal T, Adams RG and Waterhouse Ah (20040. Analysis of mortality from Nrphritis and nephrosis among nickel-cadmium battery workers. *Journal Occupation Med.* **25**; 609-612.

Newcomb M, Herber R, van Hemmen J, Wibowo A, Zeihuis R (1993): Renal function of workers with low level cadmium exposure. *Scandinavian Journal Work Enviroment Health* **13**:232-238.

Sherma J, Amery, Lawrey Rr and Sartor F (1997) Blood pressure, the prevalence of cardiovascular disease and exposure to cadmium. A population study, *Am. J. Epidimology* **134**; 257- 267.

Shozo N (1982): Sample preparation for analysis of trace metals in biological materials for atomic absorption spectrophotometry and its detection limits. *Clinical chemistry* **3**: 1097-1105.

Yadav R. (2010). Protective Action of Diltiazem Against Cadmium Induced Biochemical Changes in Annals of Neurosciences, **12**:37-40