BLOOD GLUCOSE, CHOLESTEROL AND BLOOD PRESSURE LEVELS IN CIGARETTE SMOKERS

By

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DEDICATION

To my dear family

Father, mother, brothers and sisters

To my dear friends and colleagues

With love and respect

Hafiz

Acknowledgement

Iam grateful to “Allah” for supporting me in all what concern in the life.

It give me pleasure to presenting my deep gratitude and thanks to my supervisor Dr. Barakat Elhussein for his continuous and precious advises and keen guidance through out this study.

Also I wish to express my gratitude to my co-supervisor Dr. Hayder Ahmed Giha for his inspiring guidance and creative ideas to build up this work.

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My thanks to every person who contributed directly or indirectly and whose not mentioned here.

ABSTRACT

This study was carried out in Khartoum State during April to June 2005. The blood pressure, blood glucose and blood cholesterol in smokers were measured.

Fourty healthy males, their ages in range of 18 – 35 years were included in this study all of them were smokers.

Two samples were taken from each smoker, one before the smoking as control and the other was taken 30 minutes after smoking of one cigarette to estimate the effect of smoking on the blood glucose and blood cholesterol.

For blood pressure, also it was measured before the smoking and 30 minutes after the smoking of one cigarette.

The results of the study showed that there was a significant increase in systolic blood pressure and blood glucose after the smoking but there was no significant increase in diastolic blood pressure and blood cholesterol.

بسم الله الرحمن الرحيم

ملخص الأطروحة

أجريت هذه الدراسة بولاية الخرطوم في الفترة ما بين شهري أبريل وحتى يونيو 2005م. في هذه الدراسة تم قياس ضغط الدم وسكر الدم والكوليسترول في الدم.

أستخدم في هذه الدراسة 40 رجل تتراوح أعمارهم بين 18 – 35 سنة وجميعهم مدخنين.

تم إجراء الدراسة بأخذ عينتين من الدم من كل مدخن، العينة الأولى أخذت قبل التدخين للتحكم والثانية أخذت بعد 30 دقيقة من تدخين سيجارة واحدة لقياس السكر والكوليستيرول. أيضاً تم قياس ضغط الدم قبل التدخين وبعد 30 دقيقة من تدخين سيجارة واحدة.

نتائج الدراسة أظهرت زيادة واضحة في ضغط الدم الإنقباضي وسكر الدم لدى المدخنين بينما لم تظهر النتائج زيادة واضحة في ضغط الدم الإنبساطي ومستوى الكوليستيرول في الدم.

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INTRODUCTION

Smoking is one of the most ancient and widespread people activities over the world (Croosland, 1980).

Approximately 5.5 trillion cigarettes are produced globally each year by the Tobacco industry, smoked by over 1.1billion people (World Health Organization, 2000).

Cigarette manufacture began at the end of the 19th century, and now cigarettes account for more than 90% of tobacco consumption (Rang *et al*., 1999).

The use of tobacco in cigarette form becoming increasingly popular after the Crimean War, this was enhanced by the development of certain types of tobaccos that are suitable for cigarettes. During the second half of the 20th century, the adverse effects of cigarettes started to become widely known (World Health Organization, 2000).

People smoke in order to stimulate mental activity and to reduce nervous tension, and smokers reported that smoking wakes them up when they are drowsy and calms them down when they are tense (Rang *et al*., 1999).

Socio-cultural factors also play an important role in determining who smokes, under what circumstances and for what reasons (Helman, 2000).

The power of some psychoactive drugs to produce valuable effect lies in their ability to influence certain centres at the base of the brain followed by the release of catecholamines. It is known that nicotine in cigarette in doses equivalent to those obtained by smoking releases catecholamines in these areas (Royal College of Physicians, 1978).

Smoking is widely spread in the latter years (World Health Organization, 2000) and it has many risks on our life (Rang *et al*., 1999; Edward *et al*., 1995 and Jean *et al*., 1991) some of them are cancer, cardiovascular diseases beside its role on the metabolism, therefore, the objective of this study is to estimate the effect of smoking on the following parameters:

1. blood pressure
2. blood glucose
3. blood cholesterol

CHAPTER ONE

LITERATURE REVIEW

1.1. Tobacco:

Within 150 years of Columbus finding in the new world, tobacco was being used around the world (Judith and Machael, 2002). It is chewed, smoked and a applied in a variety of ways all over the world and there is a large spectrum of tobacco products which are manufactured and marketed for human consumption, and the number and variety of these products are very large (World Health Organization, 1999).The use of tobacco in any form has been found to be seriously damaging the human health and it should be emphasized that inclusion of tobacco in any product meant for human consumption is unacceptable, and the most common tobacco product all over the world is the cigarette(World Health Organization, 1999).

1.2. Cigarette:

1.2.1. Definition and characteristics of cigarette:

A cigarette is a small paper wrapped cylinder (generally less than 120 mm in length and 10 mm in diameter) of cured and shredded or cut tobacco leaves.

The cigarette is ignited at one end and allowed to smoulder for the purpose of inhalation of its smoke from the filter end, inserted in the mouth, and the term, as commonly used typically refers to a tobacco cigarette, but can apply to similar devices containing other herbs, such as cannabis (World Health Organization, 2000).

A cigarette is distinguished from a cigar by its smaller size, use of processed leaf, and paper wrapping; cigars are typically composed entirely of whole leaf tobacco. Cigarette were largely unknown in English speaking world before the Crimean War, when British soldiers began emulating their Ottoman Turkish comrades, who resorted to rolling their tobacco with newsprint. Cigarettes despite their dwindling popularity, have accumulated a variety of nick names such as “smokes”, “butts”, “square”, “cig” and “fag” (World Health Organization, 2000).

1.2.2. Constituents of cigarette:

The leaves of tobacco plant are first dried to make cigarettes and then treated with a variety of chemicals and many additional ingredients are added and the contents of tobacco vary according to the hybrid of tobacco plant, the position of the leaf on the plant, and the region where it is grown (Smith, 2001).

Tobacco smoke contains more than 4000 chemicals, many of which are toxic, mutagenic and carcinogenic some of these including: acetone, aluminum, amonia, arsenic, benzene, butane, camium, caffeine, carbon monoxide, carbon dioxide, chloroform, copper, cyanide, DDT/Dieldrin, ethanol, formaldehyde, hydrogen cyanide, lead, magnesium, methan, methanol, mercury, nicotine, polonoum, tar, vinly choride (World Health Organization, 2000).

The amount of these ingredients can vary widely from one brand or type of cigarette to the other and the main components in tobacco smoke are nicotine, carbon monoxide, and tar but nicotine is the most important and the most harmful and pharmacologically active one (Simth, 2001).

1.2.2.1. Main components of tobacco in cigarette smoke:

1.2.2.1.1. Nicotine:

Nicotine is a chemical substance found in tobacco leaves and it is addictives as heroin or cocaine that keeps you coming back for more (Malson and Pickworth, 2002).

An average cigarette contains about 9-17 mg nicotine, of which about 10% is normally absorbed by the smoker, and this fraction varies greatly with the habits of the smoker and the type of cigarette (Rang *et al*., 1999)

1.2.2.1.1.1. Physical and chemical properties of nicotine:

Nicotine is highly lipid soluble and is one of the few natural liquid alkaloids. It is colorless, volatile base, that turns brown and acquires the odor of tobacco on exposure to air. In its formula nicotine a tertiary base is a combination of pyridine and methyl pyrrolidine as it's shown in fig. (1) (Croosland, 1980).

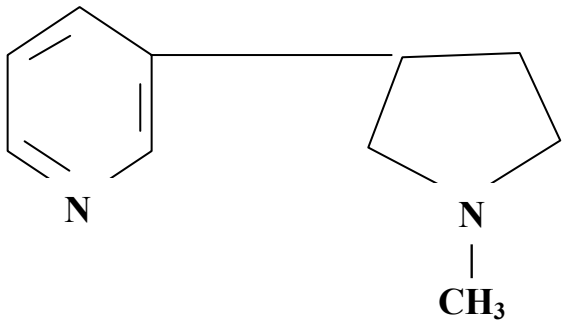


Fig. (1) Nicotine structure

1.1.2.1.1.2. Nicotine absorption and distribution:

Inhalation of the tobacco smoke is an efficient way of transferning nicotine into the blood stream.

Nicotine is absorbed from the lungs but poorly from the mouth and nasopharynx. The amount of nicotine absorbed from an average cigarette is

about 1.5mg which causes the plasma nicotine concentration to reach 130-200nmol/L, this value depends greatly on the type of cigarette and on the extent of inhalation of the smoke (Rang *et al*., 1999 and Spohr *et al.*, 1980).

The very low nicotine cigarettes are not popular, they simply do not give enough nicotine, therefore, would be necessary to smoke and inhale at least five of them at once to get the same level of nicotine in the blood which is obtained by smoking a more popular brand (Royal College of Physicians, 1978).

1.2.2.1.1.3. Mechanism of nicotine action :

Nicotine can affect every organ in the body, small doses stimulate and large doses paralyze all nerves which are activated by the acetylcholine a particular chemical transmitter of nervous impulses (Royal College of Physicians, 1978)

Nicotine reaches the brain rapidly, being present within a minute of the first pull of a cigarette, the action of it is extremely complex, it can produce stimulation and arousal on the one hand and relaxation on the other, these effects depend not only on the dose and individual constitution but also on mood and situation, thus if we are angry or fearful its effect is to calm and sedate, but if we are bored or fatigued it will arouse and stimulate

(Royal College of Physicians, 1978 and Ivery, 1978). The major action of nicotine consists initially of a transient stimulation and subsequently of a more persistent depression of all autonomic ganglia, small doses of nicotine stimulate the ganglion cells directly and facilitate the transmission of impulses but in large doses, nicotine initially causes stimulation then followed by a blockage of transmission (Ivery, 1978). Peripherally nicotine stimulates adrenal medulla to discharge catecholami-nes and also it increases blood pressure and heart rate (Myceky *et al.,* 2000)

1.2.2.1.1.4. Effects of nicotine:

1.2.2.1.1.4.1. Effect of nicotine on metabolism:

Nicotine in cigarette smoke stimulates certain ductless glands to secrete hormones. For example, it stimulates the adrenal gland to release adrenaline and noradrenaline which bring about a transient increase in concentration of blood glucose and elevation or fluctuation of plasma free fatty acids level (Royal College of Physicians, 1978). Elevation of plasma free fatty acids lead to increasing VLDL secretion by the liver, involving extratriacyl glycerol and cholesterol out put into the circulation. Factors leading to higher or fluctuating levels of free fatty acids include emotional

stress, nicotine from cigarette smoking, coffee drinking and taking of a few large meals rather than more continuing feeding (Murray *et al*., 1996).

Bornemisza and Suciu (1980) reported that nicotine inhaled from cigarette smoke causes increase in blood glucose level and they attributed that to the mobilization of catecholamines from adrenal gland under the effect of nicotine itself, and they mentioned that the releasing of catecholamines depend upon the extent of nicotine inhalation, and there was no elevation in blood glucose level after smoking when the smoking is not inhaled.

Gutierez *et al.* (1992) found that there was a significant increase in blood glucose level in smokers and they reported that nicotine contained in smoke produces an elevation of plasma catecholamines which in turn may be the cause of the increase in blood glucose level by several mechanisms. Also physical and emotional stress causes increased production of epinephrine and immediate increase production of blood glucose for energy requirement and as it is known smoking acts as stress factor (Norbert, 1982). Also Cheesbrough (1998) reported that the stress bring about by smoking might brought a transient increase in blood glucose level under the

effect of releasing catecholamine hormones by the action of nicotine in smoke.

1.2.2.1.1.4.2. Effect of nicotine on blood pressure:

The effect of nicotine on the cardiovascular system resultant of a number of actions, some mediated by the nervous system and some due to a direct action of nicotine. Nicotine stimulates the sympathetic ganglia causes a wide-spread vasoconstriction which is augmented by adrenaline release and the direct action of the nicotine on the blood vessels and the vasomotor centre, then all these influence together cause the blood pressure to rise sharply (Croosland, 1980).

The blood pressure rises temporarily each time a cigarette is smoked, primary because of the nicotine effect on catecholamines and if some one smoked 20 or more cigarette per day, the current elevation of blood pressure may be harmful (Doll *et al*., 1994).

1.2.2.1.1.4.3. Effect of nicotine on central nervous system:

The central effects of nicotine are complex and can not be summed up over all simply in terms of stimulation or inhibition (Rang *et al.,* 1999).

At cellular level, nicotine acts on nicotinic acetylcholine receptors, opening cations channel and causing neuronal excitation, it also causes

desensitization, at the spinal level nicotine inhibits spinal reflexes causing skeletal muscle relaxation. The peripheral effects of nicotine result from stimulation of autonomic ganglia of peripheral sensory receptors mainly in the heart and lungs and stimulation of these receptors elicits various autonomic reflex responses causing tachycardia, increasing cardiac out put and increasing arterial pressure and when the people smoke for the first time some times vomit, probably because of stimulation of sensory receptors in the stomach. (Rang *et al*., 1999).

1.2.2.1.1.5. Nicotine excretion:

Nicotine is oxidized and hydroxylated by microsomal oxidases, which yield less effective metabolic products (Casarett and Doll, 1975).

This occurs mainly in the liver within 1-2 hours (half life) and the inactive metabolite cotinine has a long plasma half-life, and can be used as a measure of smoking habits.

Both nicotine and its metabolites are rapidly eliminated by the kidney, and the rate of urinary excretion depends on the pH of the urine, thus excretion diminished when the urine is alkaline, also nicotine is excreted in the milk of lactating women who smoke, the milk of heavy

smokers may contain 0.5 mg per liter (Joel *et al*., 1996 and Russel and Feyerabend, 1978).

1.2.2.1.2. Carbon monoxide:

Carbon monoxide is a poisonous gas without smell and colour and it arises while the cigarette paper is burning. The quantity of carbon monoxide depends on how quick the cigarette is burning and on porosity of cigarettes paper, and usually its quantity increases at the end of cigarette (Smith, 2001).The carbon monoxide inhaled from the cigarette is diluted with air in the lung and reaches a final concentration of about 400 parts per million, although it is low, this concentration is significant because carbon monoxide has a high affinity for the oxygen-carrying red pigment of the blood, hemoglobin, about 200 times higher than oxygen itself (Royal College of Physicians, 1978).

1.2.2.1.2.1. Effects of carbon monoxide:

Carbon monoxide binds to the haemoglobin, which normally carries oxygen from the lungs via the blood stream, and therefore, reduces the amount of oxygen reaching body tissues (Lee and Sim, 2003). The elevation of blood pressure and heart rate due to the effect of nicotine raises the body's demand for oxygen but when this combined with the effect of

carbon monoxide this was creates an imbalance between cell's increased demand for oxygen and the reduced amount of oxygen that the blood can supply, then this makes the heart work even harder to meet the body's need, which in time can lead to ischaemia and may be myocardial dysfunction (American heart.org., 2004).

In the blood of cigarette smokers an average content of carboxy haemoglobin which is resulted from the combination of carbon monoxide with haemoglobin has been estimated at about 2.5% compared with 0.4% for non smokers (Rang *et al.,* 1999).

1.2.2.1.3. Tar:

The word "tar" describes the particulate matter which generated by burning tobacco, forms a component of cigarette smoke, and each particle composed of a large variety of chemicals consisting mainly of nitrogen, oxygen, hydrogen, carbon dioxide, carbon monoxide and a wide range of volatile compounds (Malson and Pickworth, 2002).

If smoker smoked about 10 cigarettes a day for ten years he would put on his trachea and bronchi about 1kg of tobacco tar, the bigger part of this tar is eliminated with cough and the smaller part stays in the lungs (Rang *et al.,* 1999).

1.2.2.3.1. Effects of tar:

The smaller part of tar which is not eliminated and remained in the lungs increases the possibility for lungs cancer and also irritants in tar can damage the lungs by causing narrowing of the bronchioles and damage of the small hairs which protect the lung from the dirt and infection (Rang *et al*., 1999).

Beside the effects of the main components of the cigarette smoke (nicotine, carbon monoxide and tar) which are mentioned previously for each one, it was found that the cigarette smoke by all its constituents collectively were associated with the cancer particularly of the lung, mouth, oesophagus, respiratory tract and urinary tract, also the cigarette smoke slightly reduces the smoker's weight because it increases the metabolic rate and its effective suppressant that former smokers often develop junk food habits (Royal College of Physicians, 1978).

CHAPTER TWO

MATERIALS AND METHODS

2.1. Study area and population

This study was performed in Khartoum state during April to June 2005, and the population of study were healthy males, their ages in range between 18 – 35 years.

2.2. Experimental design:

Fourty healthy males, their ages in range of 18–35 years were randomly selected from Khartoum state.

Two fasting blood samples were taken from each smoker, one before the smoking as control, and the other was taken 30 minutes after the smoking of one filtered cigarette (BRINGI) to estimate the effect of smoking on the level of blood glucose and blood cholesterol.

For blood pressure, it was also measured before the smoking and 30 minutes after the smoking of one regular filtered cigarette.

2.3. Blood collection:

After an over-night fast 5ml of venous blood were collected, and each sample was divided into two anticoagulant containing containers. One container for the estimation of glucose and the other for the estimation of

cholesterol. Then each smoker smokes one regular filtered cigarette (BRINGI), 30 minutes after the smoking other 5ml of venous blood were collected again from the smokers. Then the blood samples were centrifuged at 5000 r.p.m for 10 minutes for plasma separation . Plasma was stored at (-20° C) till analyzed.

2.4. Instruments:

1. Centrifuge.
2. Colorimeter.
3. Sphygmomanometer.

2.5. Biochemical analysis:

2.5.1. Glucose estimation:

Principle:

The oxidation of glucose is catalyzed by glucose oxidase (GOD), the resultant hydrogen peroxide (H_2O_2) is oxdatively coupled with 4-amino phenazone and phenol in the presence of peroxidase (POD) to yield a red quinineimine dye, the concentration of which at 546 nm is proportional to the concentration of glucose:

$$\alpha\text{-D. glucose} \xrightarrow{\text{Mutarotase}} \beta\text{-D. glucose)}$$

$$\beta\text{- D-glucose} + H_2O + O_2 \xrightarrow{\text{GOD}} \text{D-gluconic acid} + H_2O_2$$

H_2O_2 + 4-aminophenazone + phenol $\xrightarrow{POD}$ quinone imine + $4H_2O$

Reagents:

1. Phosphate butter (pH 7.5)
2. Amino phenazone
3. Phenol
4. Glucose oxidase
5. Peroxidase
6. Mutarotase

Procedure:

1. Three tubes were labeled as blank, standard and sample.
2. 0.01ml of distilled water was pipetted into the blank tube and 0.01ml of standard was pipetted into the standard tube also 0.01ml of sample was pipetted into the sample tube.
3. 1ml of glucose oxidase reagent was pipetted in each tube.
4. The tubes were mixed and incubated at room temperature for 10 minutes or at 37°C for 5 minutes.
5. The optical density (O. D) of the sample and standard was red using colorimeter against a reagent blank at a wavelength of 500 nm.

Calculations:

Glucose concentration (mg/dl) =

$$\frac{\text{O. D of sample}}{\text{O. D of standard}} \times \text{standard concentration}$$

2.5.2. Cholesterol estimation

Principle:

Cholesterol is determined after enzymatic hydrolysis and oxidation. The indicater quinonemine is formed from hydrogen peroxide and 4- amino phenazone in the presence of the phenol and peroxides.

$$\text{Cholesterol ester} + H_2O \xrightarrow{CHE} \text{cholesterol} + \text{fatty acid}$$

$$\text{Cholesterol ester} + O_2 \xrightarrow{CHO} \text{cholestene} - 3 - \text{one} + H_2O_2$$

$$2H_2O_2 + 4 - \text{a min ophenazone} + \text{phenol} \xrightarrow{POD} \text{quinonei min e} + 4H_2O$$

Reagents:

1. Phosphate buffer (pH 6.5)
2. 4- amino phenazone
3. Phenol
4. Peroxidase
5. Cholesterol esterase
6. Cholesterol oxidase

7. Sodium azide

Procedure:

1. Three tubes were labeled as blank, standard and sample.
2. 10µl of distilled water were pipetted into the blank tube and 10µl of standard were pipetted into the standard tube also 10µl of the sample were pipetted into the sample tube.
3. 1000µl of cholesterol oxidase reagent were added to each tube.
4. The tubes were mixed and incubated at room temperature for 10 minutes or at 37º C for 5 minutes.
5. The optical density of the sample and standard was measured using a colorimeter against a reagent blank at a wavelength of 500 nm.

Calculations:

Cholesterol concentration (mg/dl) =

$$\frac{\text{O.D of sample}}{\text{O.D of standard}} \times \text{standard concentration}$$

2.6. Blood pressure measurement:

The blood pressure was measured by using sphygmomanometer.

Procedure:

1. The rubber bag of sphygmomanometer was wrapped around the upper arm as cuff.
2. Astethoscope was placed on to the antecubital fossa.
3. The cuff was inflated by the hand-pump to a pressure above than in the brachial artery at where the blood flow to the forearm was stopped and no sound of pulse could be heard.
4. The cuff was deflated slowly until the pulse sound was heard, and the reading at the point was taken as the systolic blood pressure.
5. Further deflation was allowed for the cuff, until the sound of pulse was disappeared, and the reading at this point was taken as diastolic blood pressure.
6. The blood pressure was taken as $\frac{\text{Systolic blood pressure}}{\text{diastolic blood pressue}}(\text{mmHg})$

2.7. Statistical analysis:

Data was analyzed by t-test. In the analysis, the values of blood pressure, blood glucose and blood cholesterol which were obtained before the smoking were compared with their values obtained after the smoking by using t-test.

CHAPTER THREE

RESULT AND DISCUSSION

As it is observed in the latter years there was a wide spread of cigarette smoking among the Sudanese people mainly among the young (World Health Organization, 2000). Beside its role in causing cancer, cardiovascular diseases and many others, cigarette smoking also has a role on the metabolism and blood circulation (Myceky *et al.*, 2000). Therefore, the aim of this study is to investigate the effect of cigarette smoking on the blood pressure, blood glucose and blood cholesterol levels in healthy subjects.

3.1. Blood pressure in smokers:

3.1.1. Systolic blood pressure in smokers:

Table (1) and Fig. (2) presented the systolic blood pressure in smokers. In comparsion between smoker's systolic blood pressure before the smoking and smoker's systolic blood pressure after the smoking, there was a significant increase in smoker's systolic blood pressure after the smoking.

3.1.2 Diastolic blood pressure in smokers:

Diastolic blood pressure in smokers is presented in Table (1) and Fig. (3). There was no significant increase in smoker's diastolic blood pressure after the smoking when it was compared with its value before the smoking.

Table (1): Levels of blood pressure, blood glucose and blood cholesterol in smokers.

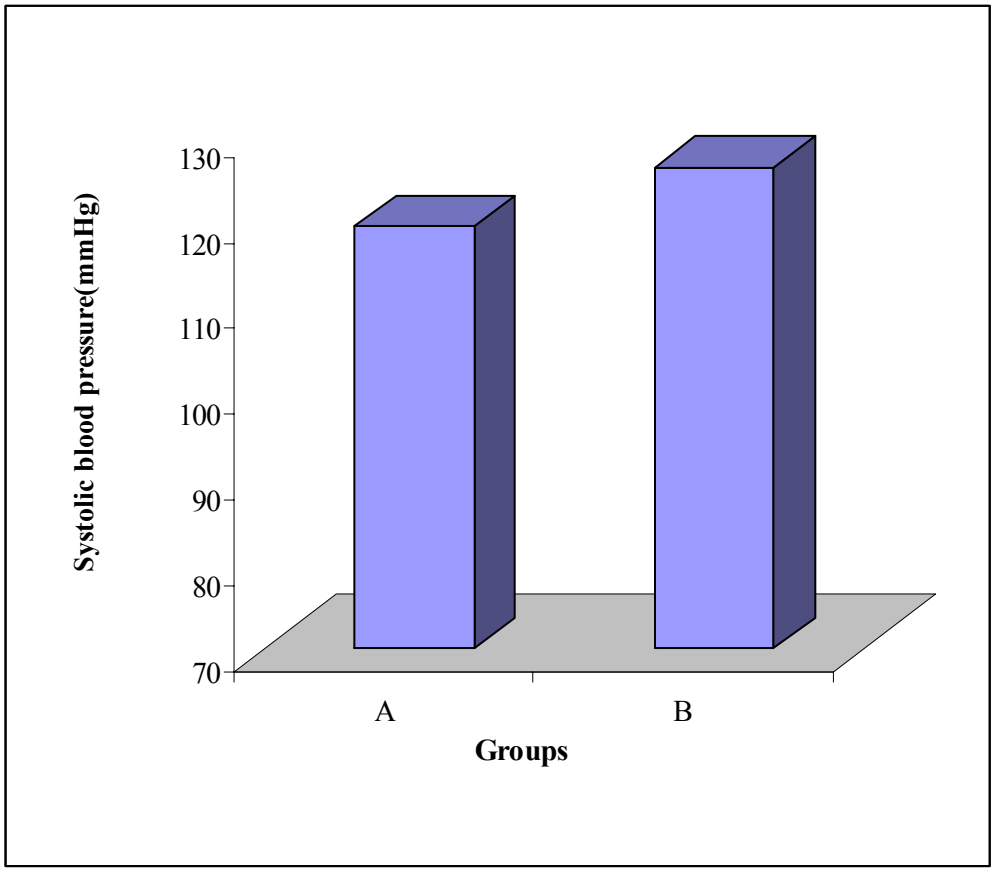
Parameters / Groups	Systolic blood pressure	Diastolic blood pressure	Blood glucose (mg/ dl)	Blood cholesterol (mg/dl)
A	119.25 ± 6.05[a]	73.13 ± 5.39[a]	85.85 ± 9.89[a]	143.75 ± 25.88[a]
B	126.13 ± 7.47[b]	75.00 ± 5.19[a]	92.08 ± 13.66[b]	147.48 ± 26.00[a]

Means (±SD) within the same column having different superscript letters are significantly different at $P < 0.05$ based on t-test.

Group A = Smokers before the smoking as control group

Group B = Smokers after the smoking

Fig. (2): Systolic blood pressure in smokers



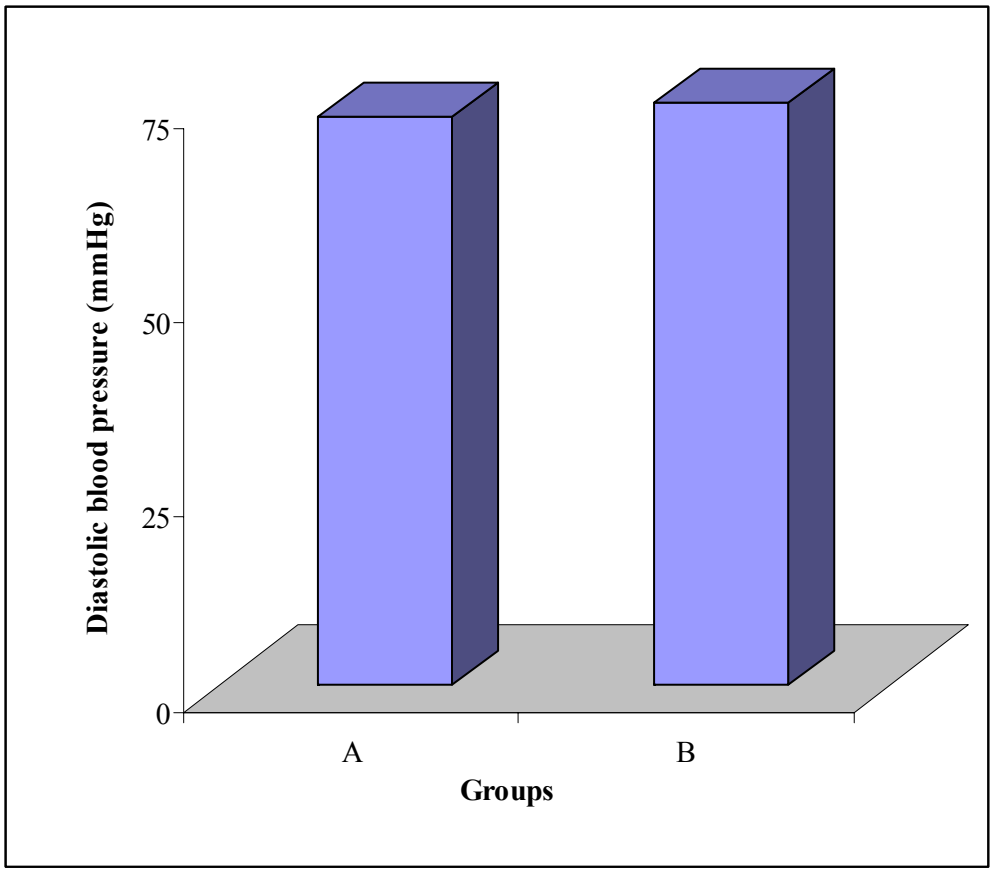
Group A = Smokers before the smoking as control group
Group B = Smokers after the smoking

Croosland (1980) reported that nicotine in cigarette smoke stimulates the sympathetic ganglia which causes a wide–spread vasoconstriction which is augmented by adrenaline release and the direct action of the nicotine on the blood vessels and the vasomotor centre, these influences together cause the blood pressure to rise sharply. Also Rang *et al.* (1999) mentioned that the peripheral effect of nicotine in cigarette smoke resulted from stimulation of autonomic ganglia at peripheral sensory receptors and the stimulation of these receptors elicits various autonomic reflex responses causing increasing cardiac out put and increasing blood pressure.

Doll *et al*. (1994) also reported that the blood pressure rises temporary each time a cigarette is smoked, primary because of the nicotine effect on catecholamines.

The significant increase in systolic blood pressure and the non-significant increase in diastolic blood pressure was often refers to the stress cases which are not remained for along time (personal communication, 2005). Joel *et al.* (1996) mentioned that the half- life of the nicotine which is the most harmful and pharmacologically active component in cigarette smoke is 1–2 hours in addition to its effects on the releasing the stress hormone epinephrine.

Fig. (3): Diastolic blood pressure in smokers



Group A = Smokers before the smoking as control group
Group B = Smokers after the smoking

According to what has been mentioned previously the increase in blood pressure after the smoking could be due to the direct action of nicotine in vasomotor centre augmented by the release of adrenaline under the effect of nicotine itself and then these influences result in vasoconstriction followed by elevation of blood pressure.

3.2 Blood glucose in smokers:

Table (1) and Fig. (4) showed the effect of cigarette smoking on blood glucose. The smoker's blood glucose level after the smoking was significantly higher than that before the smoking.

Nicotine in cigarette smoke stimulates the adrenal gland to release adrenaline and noradrenaline which bring about a transient increase in concentration of blood glucose (Royal College of Physicians, 1978).

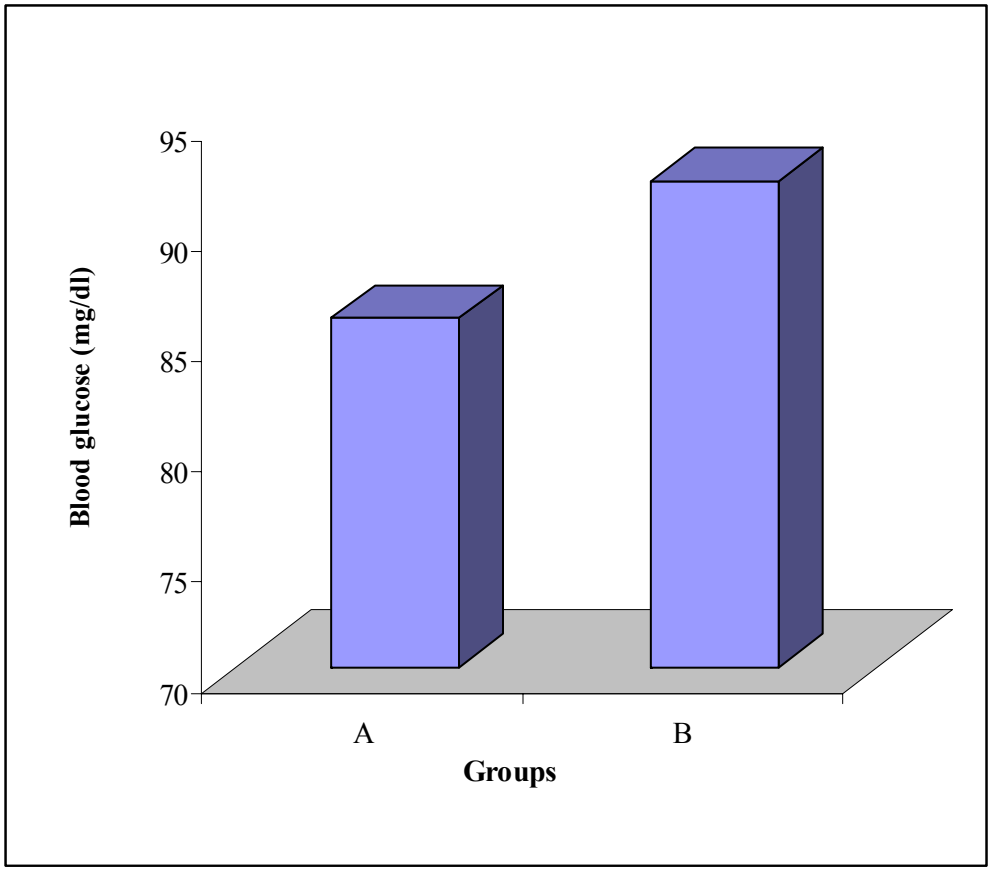
Gutierrez *et al.* (1992) reported that there was a significant increase in blood glucose level in smokers and they reported that nicotine contained in cigarette smoke produces an eleviations of plasma catecholamines which in turn may be the cause of increase in blood glucose level.

Bornemisza and Suciu, (1980) reported that nicotine inhaled from cigarette smoke causes increase in blood glucose level and they attributed

that to be due to the release of catecholamines from the adrenal gland under the effect of nicotine. Physical or emotional stress causes increased production of epinephrine and immediate increase production of blood glucose for energy requirement (Norbert, 1982). Also Cheesbrough (1998) mentioned that the stress bring about by smoking might brought a transient increase in blood glucose level under the effect of releasing catecholamine hormones by the action of nicotine in smoke.

From the reports mentioned above the increase in blood glucose level after smoking could be due to the release of the catecholamines hormones from the adrenal gland under the effect of nicotine contained in cigarette smoke, and as it is known catecholamines are mobilized in cases of stress and bring about a transient increase in blood glucose concentration.

Fig. (4): Blood glucose in smokers



Group A = Smokers before the smoking as control group
Group B = Smokers after the smoking

3.3 Blood cholesterol in smokers:

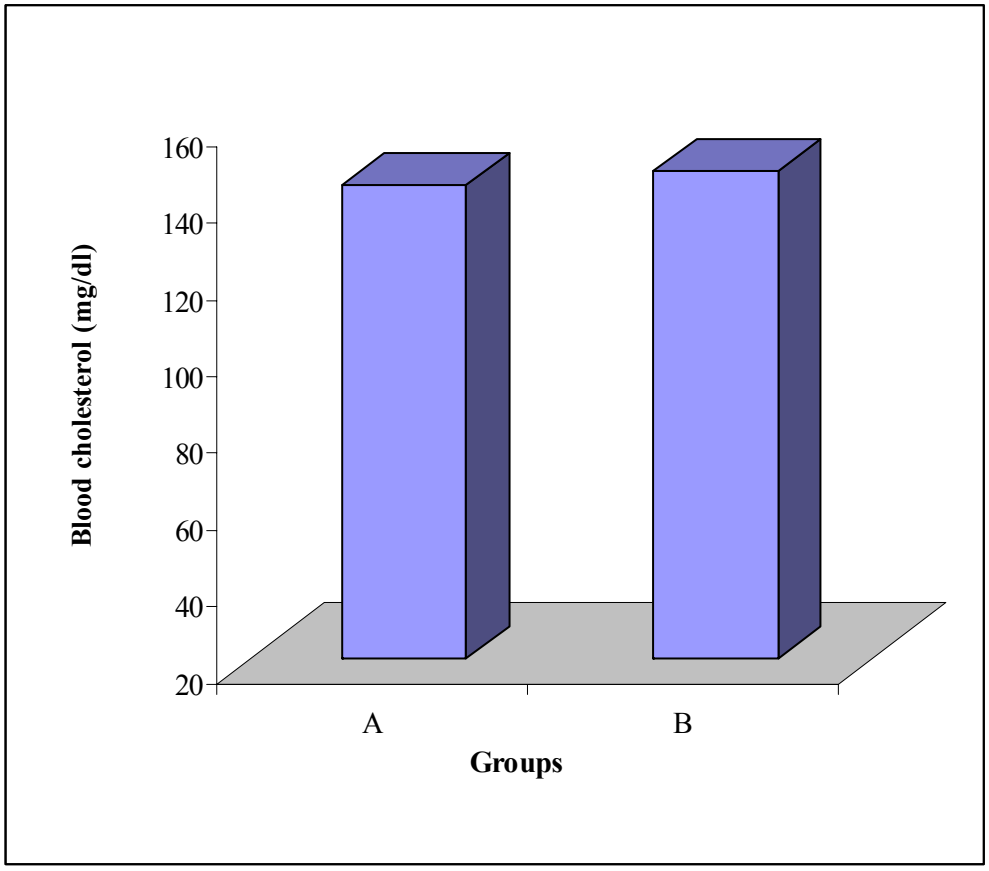
The effect of cigarette smoking on blood cholesterol was showed in Table (1) and Fig. (5). There was no significant increase in smoker's blood cholesterol after the cigarette smoking when compared with its value before the smoking.

Royal College of Physicians (1978) reported that nicotine in cigarette smoke stimulates the release of adrenaline and noradrenaline from adrenal gland which leads to elevation or fluctuation of plasma free fatty acids level. Elevation of plasma free fatty acids bring about increase in very low density lipoprotein (VLDL) by liver involving extratriacyl glycerol and cholesterol out put into the circulation (Murray *et al.*, 1996).

Also Murray *et al.* (1996) mentioned that one factor that leads to higher or fluctuating levels of free fatty acids include nicotine from cigarette Smoking.

The non-significant increase in blood cholesterol after smoking could be due to the action of nicotine in cigarette smoke on the adrenal gland to release the catecholamines which bring about fluctuation in level of free fatty acids which in turn leads to fluctuation in level of very low density liopprotien and then the level of cholesterol that released into the circulation.

Fig. (5): Blood cholesterol in smokers



Group A = Smokers before the smoking as control group
Group B = Smokers after the smoking

CONCLUSION

It is concluded from this study that the cigarette smoking had a significant effect on systolic blood pressure, blood glucose and had no significant effect on diastolic blood pressure and blood cholesterol in smokers during the half life of the effective components of smoke in the body.

RECOMMENDATIONS

Smoking is widely spread among the young peoples in last years. From this study which studied the effect of smoking on three parameters , it is recommended that more studies must be carried out on the effect of smoking on the metabolism by taking other parameters like free fatty acids and lipoproteins fractions so as to give full information in this regard.

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APPENDIX (1)

Questionnaire

Blood Pressure, Blood Glucose and Blood Cholesterol Levels In Smokers

- Serial No. .....
- Name: .....
- Age: .....
- Weight: .....
- Duration of smoking:

  < 1 year ( ) 1 – 5 years ( )

  6 – 10 years ( ) > 10 years ( )

- Number of cigarette smoked per day ?

  1 – 5 ( ) 6 – 10 ( ) > 10 ( )

- Rate of smoking (hours) during each:

  > 2 ( ) > 2 - < 3 ( )

  > 3 - < 4 ( ) > 4 ( )

- Are you suffering from stress after the smoking ?

  Yes ( ) No ( )

- If you are not smoked for a long time (e.g. one day), what is your complaint ?

  Headache ( ) Tension ( )

  Fatigue ( ) Other (Specify) ( )

.....

- Are you having snuff ?

  Yes ( ) No ( )

APPENDIX (2)

Smoking prevalence by gender

Region	Men (%)	Women (%)
Africa	29	4
America	35	22
Europe	46	26
South – East Asia	44	4
Western Pasific	46	8
Eastern Mediterranean	35	4

Source: World Health Organization (2000).