1. Introduction and literature review

1.1. Introduction

Smoking is a practice in which a substance is burned and the resulting smoke breathed in to be tasted and absorbed into the bloodstream". Cigarettes are primarily industrially manufactured but also can be hand-rolled from loose tobacco and rolling paper. Other smoking implements include pipes, cigars, bidis, hookahs, vaporizers, and bongs. It has been suggested that smoking-related disease kills one half of all long term smokers but these diseases may also be contracted by non-smokers. A 2007 report states that, each year, about 4.9 million people worldwide die as a result of smoking (west *et al*, 2007).

Cigarette smoking causes minerals disturbances which lead to serious consequences, Smoking leads to tissue hypoxia which leads to inadequate oxygenation of blood circulation that result in erythropoiesis and consequent increased production of erythropoietin (Elzayadi,2006), which enhances erythropoiesis and increases red cell mass above normal level (Balcerzek *et al*, 1975). This leads to increase in the number of destroyed red cells in the normal turnover process which subsequently increases iron overload which causes hepatocellular damage (Bacon *et al*, 1990). Chronic oxidative stress may modulate iron uptake and storage, leading to a self sustained and ever increasing spiral of cytotoxic and mutagenic events (Emrit *et al*, 2001).

Smoking causes magnesium deficiency due to decreased supply (lesser appetite) and reduced absorption caused by disturbances in the digestive system functions (Winiarczyk *et al*, 2008). Minerals disturbances may lead to sever and even life threatening metabolic abnormalities such as coronary heart disease, liver disease, lung infection, kidney failure, and disorders of endocrine system(John,2007).

There are many trace elements in the body that directly or indirectly participate in metabolism and play an essential role in it. More than 25% of the enzymes in the body require metals for activation and to function properly in metabolism (Shiffman *et al*, 2007).

Iron aids in the delivery of oxygen within the body through interaction with hemoglobin and myoglobin. Iron is also the main component of cytochrome C which is responsible for electron transfer within the mitochondria. Directly or indirectly, the iron status can have an effect on the ability of a cell to execute adenosine triphosphate (ATP) production and on the oxidation of glucose and other carbohydrates(Rude *et al*, 2006).Without iron, this oxidation is hindered, which leads to the production of inactive cells. When this happens to brain cells and the nervous system, cognitive function is affected (Bacon *et al*, 1990).

1.2. Literature review

1.2.1. Smoking

Reasons for smoking that many people smoke because of it is calming effect on the nervous system, but it should be discouraged of it is negative effect on the body. It damages the lung, blood vessels, and other organs, such as heart, but also harms that of others, there are many reasons why people take-up smoking, Many learn smoking from parents or their friends, Many people who spend their time with friends seem to catch the habit. Advertising plays a considerable role in encouraging individual to start smoking. People also look up to their elders such as teachers, doctors, and family leaders, they think that it is a prestigious to smoke (Bharg, 1976).

Smoking is the most common method of consuming tobacco. The agricultural product is often mixed with other additive and then pyrogatalyze, the resulting vapors are often inhaled and the active substance absorbed through the alveoli in the lung. The active substance triggers chemical reactions in nerve ending, which heightens heart rate, memory, alertness, and reaction time (Parrot *et al*, 1989). Dopamine and later endorphins are released, which are often associated with pleasure (Gilman *et al*, 2004). As of 2000 smoking is practiced by some1.22bilion people, men are more likely to smoke than women, though the gender gap decline with younger age (WHO, 2001).

1.2.1.1. Physical and biochemical properties of smoking

Conventionally, cigarette smoke is divided into two phases: a tar phase and a gas phase. The tar or particulate phase is defined as material that trapped when the smoke stream is passed through the Cambridge glass fiber filter

that retains 99.9% of all particulate material with a size>0.1µm(U.S. Department of Health and Human Services,2014).

The gas phase is the material that passes through the filter. The particulate (tar) phase of cigarette smoke contain>10^{17} free radicals/g, and the gas phase contain>10^{15} free radicals/puff. The radical associated with the tar phase are long-lived (hours to months), where as the radicals associated with gas phase have a shorter life span (Pyror*et al*, 1993).

Cigarette smoke that is drawn through the tobacco into an active smoker's mouth is known as mainstream smoke. Side stream cigarette smoke emitted from the burning end of cigarette. Mainstream cigarette smoke comprises 8%of tar and 92%of gaseous component (Taylor*et al 1992).* environmental tobacco smoke result from the combination of side stream smoke (85%) and small fraction of exhaled mainstream smoke (15%)from smokers9 (Glantz et *al, 1991*). side stream cigarette smoke contain a relatively higher concentration of the toxic gaseous component than main stream cigarette smoke (Powell *et al*, 1998). of all the known constituent, nicotine, a component of the tar phase, is the addictive substance of cigarette smoke.

Cigarette smoke affects all the organs of the body.The effect of cigarette in the body is related to: the age of person. How long they have smoked and how much smoke per day. Tobacco produce gases and many chemical compounds including pesticide residues artificial flavors, burning agents and poisonous substances such as nicotine, carbon dioxide, tar, acetone, ammonia methyl chloride, arsenic, nickel and others. There are many poisonous chemical in cigarette smoke, and some of these have been shown to be carcinogenic. The most poisonous of these are nicotine, carbon dioxide, and tar (Bharg , 1976) .

Nicotine is the substance that cause addiction, swallowing one drop of nicotine can kill person, is smaller and more frequent dose, it can cause addiction among smoker. It also stimulate the release of epinephrine and other substance in the body. These increase heart and blood pressure and narrow the blood vessels.

Carbon monoxide is a poisonous gas produced by the incomplete burning of cigarette. in the lungs, it combine with the hemoglobin from carrying adequate amount of oxygen through the circulatory system (Bharg , 1976) .

The tar in cigarette smoke is cancer- producing substance that contains number of carcinogens.

The effect of cigarette are considerable: the heart rate goes up, the blood pressure increases, and the skin temperature drops, smoking lead to shortness of breath, and coughing; it reduce fitness, causes yellow teeth and fingers, decrease the sense of taste and smell, it lead to impotence, severe period pain, irregular period, underweight birth, premature babies, lung cancer, stomach cancer, and many other un pleasant and potentially deadly thing.Within second of a person smoking cigarette, the heart rate become faster and the blood pressure increase. In a short time, nicotine reaches the brain and stimulates the central nervous system. The effect of smoking is dependent on the amount of tobacco smoked (Bharg , 1976) .

Compared with a non smokers, smoking is estimated to increase the risk of coronary heart disease by 2 to 4 times, stroke by 2 to 4 time, men developing lung cancer by 23 times, women developing lung cancer by 13 times, and dying from chronic obstructive lung disease (such as chronic bronchitis and emphysema) by 12 to 13 times(Zayadi *et al,2006)* .

The liver is an important organ that has many tasks. Among other things, the liver is responsible for processing drugs, alcohol and other toxins to remove

them from the body. Heavy smoking yields toxins which induce necroinflammation and increase the severity of hepatic lesions (fibrosis and activity scores) when associated with hepatitis C virus (HCV) or hepatitis B virus (HBV) infection. Cigarette smoking increases the risk of developing HCC among chronic liver disease (CLD) patients.Independentlyof liver status. Association of smoking with hepatocellular carcinoma (HCC) irrespective of HBV status has been reported. Smoking induces direct or indirect effects on the liver.

-Direct toxic effect: Smoking yields chemical substances with cytotoxic potentials. These chemicals created by smoking induce oxidative stress associated with lipid per oxidation .Which leads to activation of stellate cells and development of fibrosis. In addition, smoking increases the production of pro-inflammatory cytokines (IL-1, IL-6 and TNF-α) involved in liver cell injury. It has been reported that smoking increases fibrosis score and histological activity index in chronic hepatitis C (CHC) patients, and contributes to progression of HBV-related cirrhosis.

-Indirect toxic effects (concomitant polycythemia) Heavy smoking is associated with increased carboxyhaemoglobin and decreased oxygen carrying capacity of red blood cells (RBCs) leading to tissue hypoxia. Hypoxia stimulates erythropoietin production which induces hyperplasia of the bone marrow. The latter contributes to the development of secondary polycythemia and in turn to increased red cell mass and turnover. This increases catabolic iron derived from both senescent red blood cells and iron derived from increased destruction of red cells associated with polycythemia. Furthermore, erythropoietin stimulates absorption of iron from the intestine. Both excess catabolic iron and increased iron absorption ultimately lead to its accumulation in macrophages and subsequently in hepatocytes over time, promoting oxidative stress of hepatocytes.

Accordingly, smoking might be a contributing factor to secondary iron overload disease in addition to other factors such as transfusional haemosidrosis, alcoholic cirrhosis, thalassemia, sideroplasticanemia and porphyria cutanea tarda. In the meantime, increased red cells mass and turnover are associated with increased purine catabolism which promotes excessive production of uric acid. Eventually uric acid is deposited in tissues and joints as manifested clinically by prickling sensation, purities and arthralgia (Zayadi *et al,2006)* .

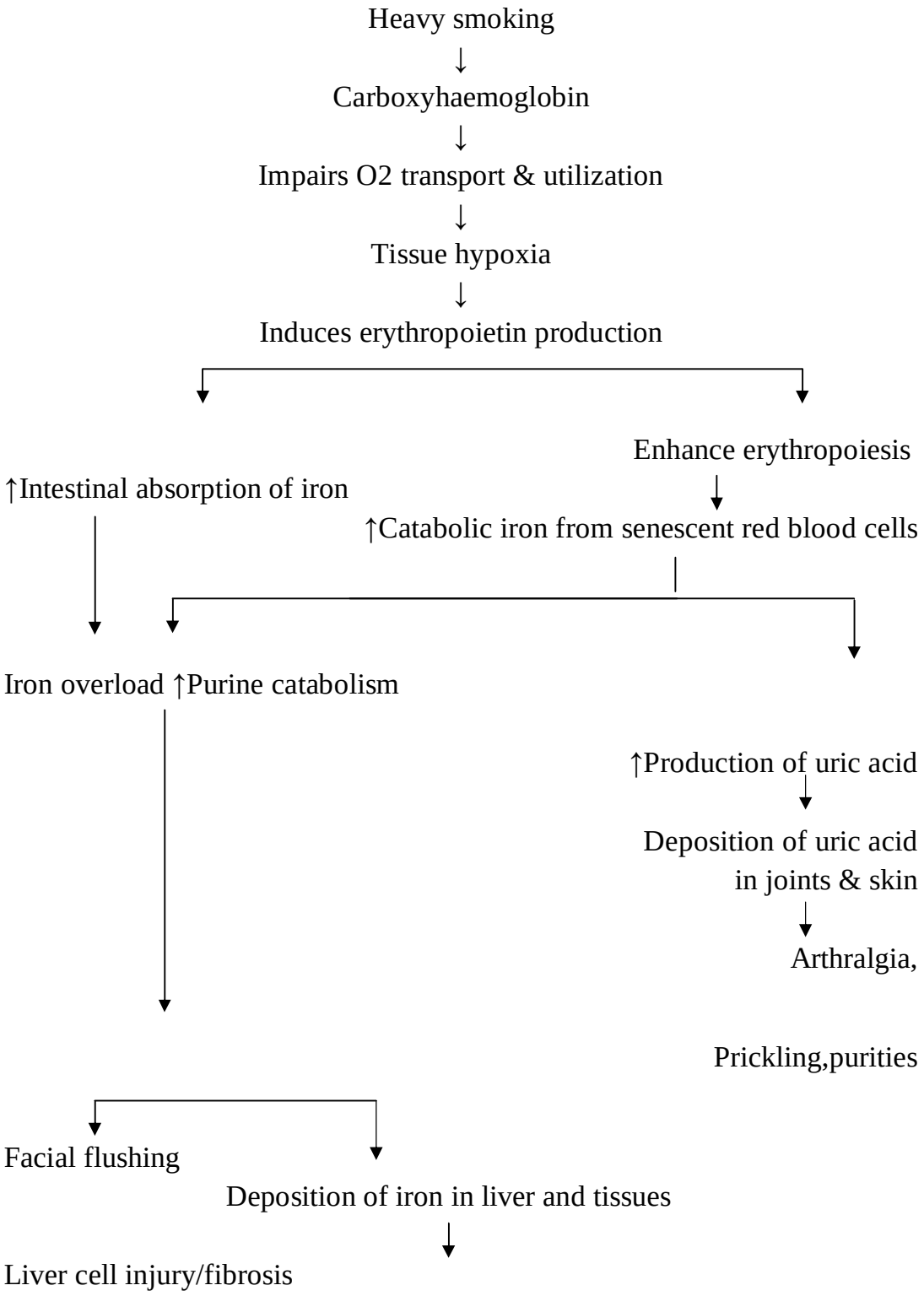

Figure 1.1 Development of smoker's polythycemia and its adverse effects (E l -Zayadi *et al*, 2006).

Smoking and respiratory disease; Smoking causes lung cancer, lung disease (e.g emphysema bronchitis, chronic air way obstruction) by damaging the air ways and alveoli of the lung (U.S. Department of Health and Human Services, 2014)

1.2.2. Iron

Normally very small quantities of iron are present in most cells of the body, in plasma, and in other extracellular fluid. Physiologically the body rigorously conserves its iron supply, so that less 0.1% of body iron content is lost daily, mostly in desquamated cells (tietz, 2008).

1.2.2.1. Chemistry and compound

Iron forms compounds mainly in the +2 and +3 oxidation states. Traditionally, iron (II) compounds are called ferrous, and iron(III) compounds ferric. Iron also occurs in higher oxidation states, an example being the purple potassium ferrate (K_2FeO_4) which contains iron in its +6 oxidation state. Iron (IV) is a common intermediate in many biochemical oxidation reactions.(Wonwoo, 2007).Numerous organometallic compounds contain formal oxidation states of +1, 0, −1, or even −2. The oxidation states and other bonding properties are often assessed using the technique of Mossbauer spectroscopy.(Reiff, *et al* ,1984). There are also many mixed valence compounds that contain both iron(II) and iron(III) centers, such as magnetite and Prussian blue ($Fe_4(Fe[CN]_6)_3$) .(Holleman, et al ,1985) . The latter is used as the traditional "blue" in blueprints.(War et al (1999). Hydrated iron(III) chloride, also known as ferric chloride. The iron compounds produced on the largest scale in industry are iron(II) sulfate ($FeSO_4 \cdot 7H_2O$) and iron(III) chloride ($FeCl_3$). The former is one of the most readily available sources of iron (II), but is less stable to aerial oxidation than Mohr's salt ($(NH_4)_2Fe\ (SO_4)_2 \cdot 6H_2O$). Iron (II) compounds tend to be

oxidized to iron (III) compounds in the air (Holleman, et al, 1985). Unlike many other metals, iron does not form amalgams with mercury. As a result, mercury is traded in standardized 76 pound flasks (34 kg) made of iron (Gmelin, Leopold,1852)

1.2.2.2.Biological roleof iron

Iron is a necessary trace element found in nearly all living organisms. Iron-containing enzymes and proteins, often containing hemeprosthetic groups, participate in many biological oxidations and in transport. Examples of proteins found in higher organisms include hemoglobin, cytochrome and catalase(Lippard *et al*, 1994).

1.2.2.3. Human iron metabolism

Human iron metabolismis the set of chemical reactions maintaining human homeostasis of iron. The control of this necessary but potentially toxic substance is an important part of many aspects of human health and disease. Hematologists have been especially interested in the system of iron metabolism because iron is essential for red blood cells, where most of the human body's iron is contained. Understanding this system is also important for understanding diseases of iron overload, like hemochromatosis, and iron deficiency, like iron deficiency anemia (Ganz , 2003).

1.2.2.4. Important of iron regulation

Iron is an absolute requirement for most forms of life, including humans and most bacterial species. Plants and animals all use iron; hence, iron can be found in a wide variety of food sources. Iron is essential to life due to its unusual flexibility to serve as both an electron donor and acceptor. Iron can also be potentially toxic. Its ability to donate and accept electrons means that if iron is free within the cell, it can catalyze the conversion of hydrogen

peroxide into free radicals. Free radicals can cause damage to a wide variety of cellular structures, and ultimately kill the cell. To prevent that kind of damage, all life forms that use iron bind the iron atoms to proteins. This binding allows cells to benefit from iron while also limiting its ability to do harm (Andrews , 1999).

1.2.2.5. Function of iron

The most important group of iron-binding proteins contains the heme molecules, all of which contain iron at their centers. Humans and most bacteria use variants of to carry out redox reactions and electron transport processes. These reactions and processes are required for oxidative phosphorylation. That process is the principal source of energy for human cells; without it, most types of cells would die. The iron-sulfur proteins are another important group of iron-containing proteins.

Some of these proteins are also essential parts of oxidative phosphorylation. Humans also use iron in the hemoglobin of red blood cells, in order to transport oxygen from the lungs to the tissues. Iron is also an essential component of myoglobin to store and diffuse oxygen in muscle cells. The human body needs iron for oxygen transport. That oxygen is required for the production and survival of almost all cells in our bodies (mature erythrocytes being one exception)(Pigott*et al* ,1999).

1.2.2.6. Iron deficiency

Iron deficiency is a condition resulting from too little iron in the body. Iron deficiency is the most common nutritional deficiency and the leading cause of anemia in the United States (Centers for Disease Control and Prevention. Iron deficiency – United States, 1999).

The terms anemia, iron deficiency, and iron deficiency anemia often are used interchangeably but equivalent. Iron deficiency ranges from depleted iron stores without functional or health impairment to iron deficiency with anemia, which affects the functioning of several organ systems (Akman,et al, 2004).

Iron deficiency is a concern because it can:

- Iron deficiency can delay normal infant motor function (normal activity and movement) or mental function (normal thinking and processing skills) (Friel, *et al* , 2003)
- Iron deficiency anemia during pregnancy can increase risk for small or early (preterm) babies (. Scholl, et al, 1992). Small or early babies are more likely to have health problems or die in the first year of life than infants who are born full term and are not small.
- Iron deficiency can cause fatigue that impairs the ability to do physical work in adults, Iron deficiency may also affect memory or other mental function in teens (National Library of Medicine, NIH).

1.2.2.7. Iron overload

The body is able to substantially reduce the amount of iron it absorbs across the mucosa. It does not seem to be able to entirely shut down the iron transport process. Also, in situations where excess iron damages the intestinal lining itself (for instance, when children eat a large quantity of iron tablets produced for adult consumption), even more iron can enter the bloodstream and cause a potentially deadly syndrome of iron overload. Large amounts of free iron in the circulation will cause damage to critical cells in the liver, the heart and other metabolically active organs (Schrier, *et al* ,2011).

Iron toxicity results when the amount of circulating iron exceeds the amount of transferrin available to bind it, but the body is able to vigorously regulate its iron uptake. Thus, iron toxicity from ingestion is usually the result of extraordinary circumstances like iron tablet over-consumption(Andrews, 1999). Rather than variations in diet. The type of acute toxicity from iron ingestion causes severe mucosal damage in the gastrointestinal tract.

Chronic iron toxicity is usually the result of more chronic iron overload syndromes associated with genetic diseases, repeated transfusions or other causes. In such cases the iron stores of an adult may reach 50 grams (10 times normal total body iron) or more. Classic examples of genetic iron overload includes hereditary hemochromatosis (HH) and the more severe disease juvenile hemochromatosis (JH) caused by mutations in either the gene RGMc gene, a member of a three gene repulsive guidance molecule family, (also called hemojuvelin (HJV), and HFE2), Hemojuvelin, or the HAMP gene that encodes (an iron regulatory peptide)(Severyn et al , 2009).

1.2.3. Total iron-binding capacity

Is a medical laboratory test that measures the blood's capacity to bind iron with transferrin. It is performed by drawing blood and measuring the maximum amount of iron that it can carry, which indirectly measures transferrin(Yamanishi et al, 2003).Since transferrin is the most dynamic carrier. TIBC is less expensive than a direct measurement of transferring (Kasvosve et al, 2002).

The TIBC should not be confused with the UIBC, or "unsaturated iron binding capacity". The UIBC is calculated by subtracting the serum iron from the TIBC (Bryce *et al*, 2007).

We usedserum iron and percent transferrin saturation clinicians usually perform this test when they are concerned about anemia, iron deficiency or iron deficiency anemia. However, because the liver produces transferrin, alterations in function (such as cirrhosis, hepatitis, or liver failure) must be considered when performing this test. It can also be an indirect test of liver function, but is rarely used for this purpose. The percent transferrin saturation (i.e., the result of the formula of serum iron/TIBC x 100) can also be a useful indicator, TIBC isincreased in iron-deficiency, use of oral contraceptives and in pregnancyand decreased in hypoproteinemia due to many causes, and in a number of inflammatory states(Finch, *et al* 1982)

1.3. Rationale

Every hundreds of thousands around world die from disease caused by smoking cigarette. Number of researches indicated that smoking has numerous immediate health effects on the liver, respiratory, cardiovascular, gastrointestinal, immune and metabolic system, lung cancer,other cancer, heart disease, and mineral disturbance(Elzayadi, 2006).

Only a single study was conducted in Sudanese smokers to evaluate serum iron (Abdalla, *et al* 2013) but no studies were found in evaluation of Sudanese smokers'total iron binding capacity, that’s why we attempted to evaluate the levels of serum iron and total iron binding capacity in Sudanese smokers.

1.4. Objectives

General objective:

To study the influence of cigarette smoking on serum iron and total iron binding capacity level among Sudanese male smokers.

Specific objectives:

1. To measure serum iron and total iron binding capacity concentrations in cigarette smokers in comparison to non smokers healthy individuals.
2. To correlate between the level of serum iron and total iron binding capacity with number of cigarettes per day and duration of the smoking per year.

2. Materials and Methods

2.1. Materials

2.1.1. Study design: This is a descriptive analytical case control study.

2.1.2. Study area: The study was conducted in cigarette smokers in Khartoum state**.**

2.1.3. Study population: This study included 70 cigarette smokers and 50 apparently healthy non smokers as control during March to June 2015.

2.1.4. Inclusion Criteria: Male cigarette smokers were included.

2.1.5. Exclusion criteria: Individuals without kidney diseases, anemia and iron overload were excluded.

2.1.6.Samples: About 5ml of venous blood were collected from each patient at the plane containers after clotting centrifuged for 3 minutes at 3000 RPM to obtain serum, and analyzed.

2.1.7. Ethical consideration: individuals who voluntarily accepted to participate in the study were included.

2.1.8. Equipments:

- Cabas integra400
- Centrifuge
- Sterile plane containers
- Disposable syringes
- 70% alcohol
- Tourniquets

- Cotton
- Micropipettes (automatic pipettes)
- Graduated pipettes

2.1.9. Data analysis: Data was analyzed by using the SPSS computer program

2.2. Methodology

2.2.1 Estimation of serum iron concentration method: (Appendix II)

Principle of method:

Guanidine/FerroZine method, Fe(III) is released from transferrin by guanidine hydrochloride and reduced to Fe(II) by ascorbate and hydroxylamine. Bivalent iron ions form a red-colored chelate complex with FerroZine. To prevent copper interference, cupric ions are bound to thiourea.

$$\text{Transferrin-Fe(III)} \xrightarrow{\textit{Guanidine-HCl}} \text{apotransferrin} + \text{Fe(III)}$$

$$\text{Fe(III)} \qquad \text{Fe(II)} \xrightarrow{\textit{Reducing agents}}$$

$$\text{Fe(II)} + 3\ \text{FerroZine} \longrightarrow \text{Fe(II)-(FerroZine)3}$$

The color intensity is directly proportional to the iron, concentration. It is determined by measuring the increase in absorbance at 552 nm.

2.2.2 Estimation Unsaturated iron binding capacity method: (Appendix III)

Principle of method:

Direct determination with FerroZine.

$$\text{Fe(II)} + \text{transferrin} \xrightarrow{\textit{Alkaline Buffer}} \text{transferrin-Fe(III)} + \text{Fe(II) (excess)}$$

$$\text{Fe(II) (excess)} + 3\ \text{FerroZine} \longrightarrow \text{Fe(II)-(FerroZine)3}$$

The color intensity is directly proportional to the unbound excess iron concentration and indirectly proportional to the unsaturated iron-binding capacity. It is determined by measuring the increase in absorbance at 552 nm.

2.2.3. Quality control

The control materials (normal and abnormal) used in this study and the values obtained fall within the defined limits.

3.Results

Seventy smokers were enrolled in this study to assess the influence of cigarette smoking on serum iron and total iron binding capacity and fifty non smokers were served as control group.

Cobas Integra 400 auto analyzer was used for estimation of serum iron and total iron binding capacity levels.

Statistical analysis was done by using SPSS computer program and the results were as follow:

Table 3.1 shows the comparison of means of serum iron and total iron binding capacity in smokers and non smokers groups.

Figure 3.1 a scatter plot shows a significant positive correlation between serum iron and number of cigarettes per day (p. value=0.000, r=0.604).

Figure 3.2 a scatter plot shows a significant positive correlation between serum iron and duration of smoking per year (p. value=0.000, r=0.797).

Figure 3.3 a scatter plot shows a significant negative correlation between total iron binding capacity and number of cigarettes per day (p. value=0.000, r= -0.499).

Figure 3.4 a scatter plot shows a significant negative correlation between total iron binding capacity and duration of smoking per year (p. value=0.000, r= -0.410).

Table (3-1):Comparison between means of serum iron and total iron binding capacity levelsin smokers and non smokers.

Variable	Smokers N=(70)	Non smokers N=(50)	p.value
Serum iron(µg/dl)	162.2±54.6	I08.4±36.8	0.000
Total iron binding capacity(µg/dl)	254.3±53.9	329.2±66.3	0.000

Independent sample T test was used for comparison, value conceder significant at level ≤ 0.05

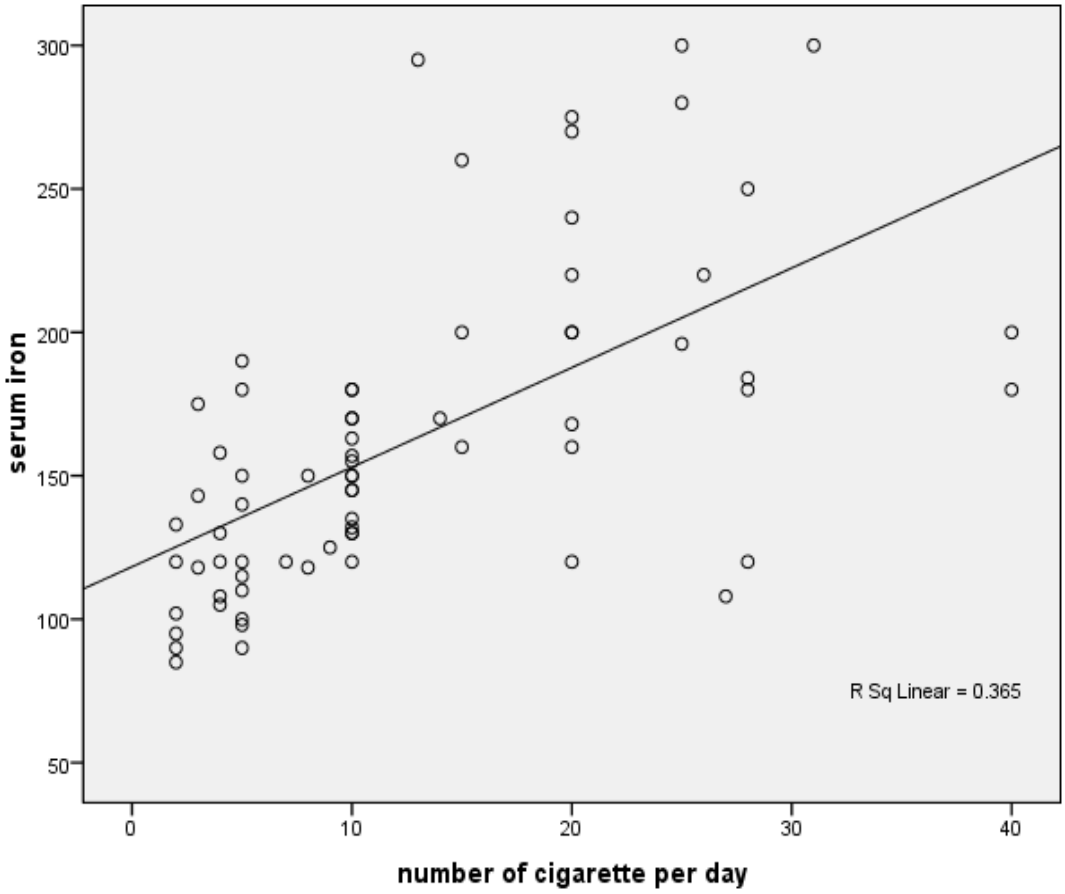

Figure 3.1: Correlation between serum iron and number of cigarette per day among Sudanese smokers.

(p. value=0.000, r=0.604).

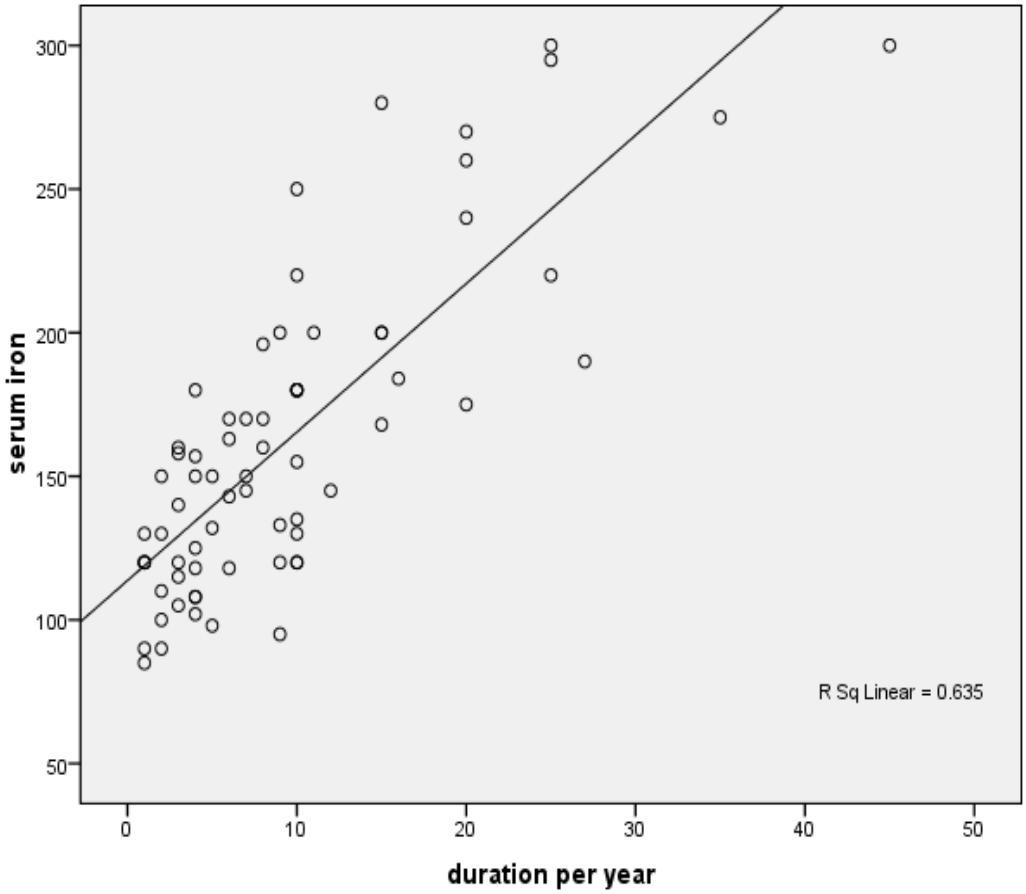

Figure 3.2: Correlation between serum iron and duration per year among Sudanese smokers.

(p. value=0.000, r=0.797).

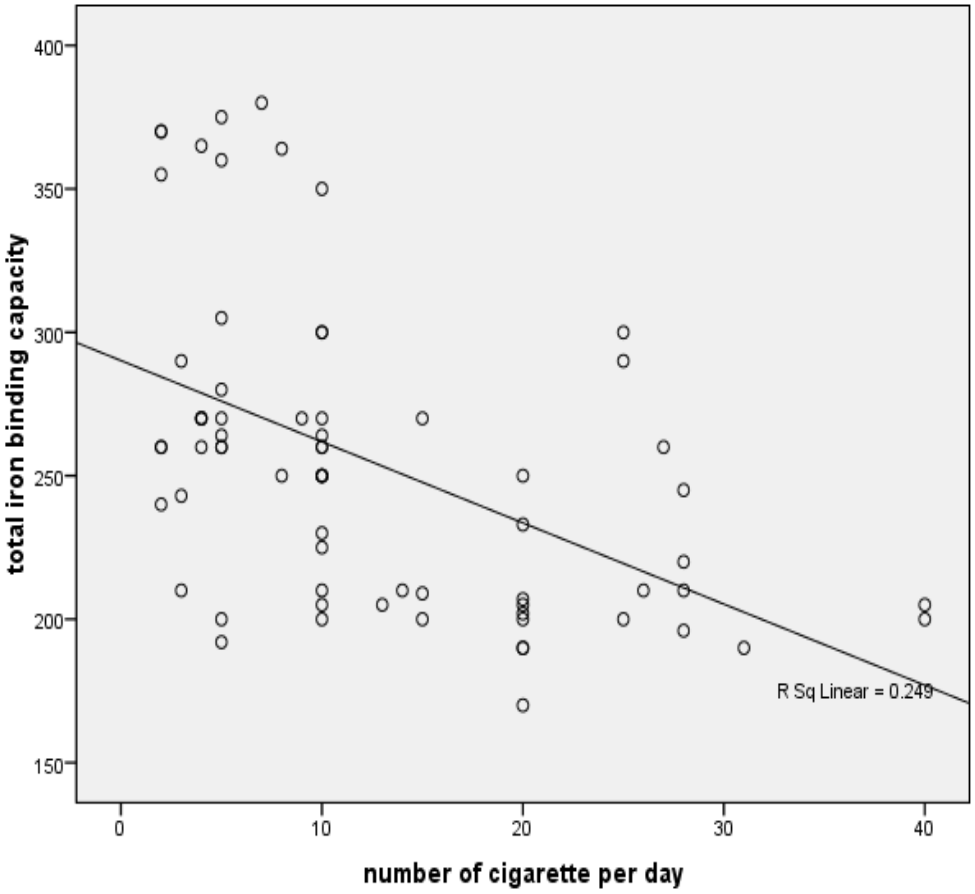

Figure 3.3: Correlation between total iron binding capacity and number of cigarette per day among Sudanese smokers.
(p. value=0.000, r= - 0.499).

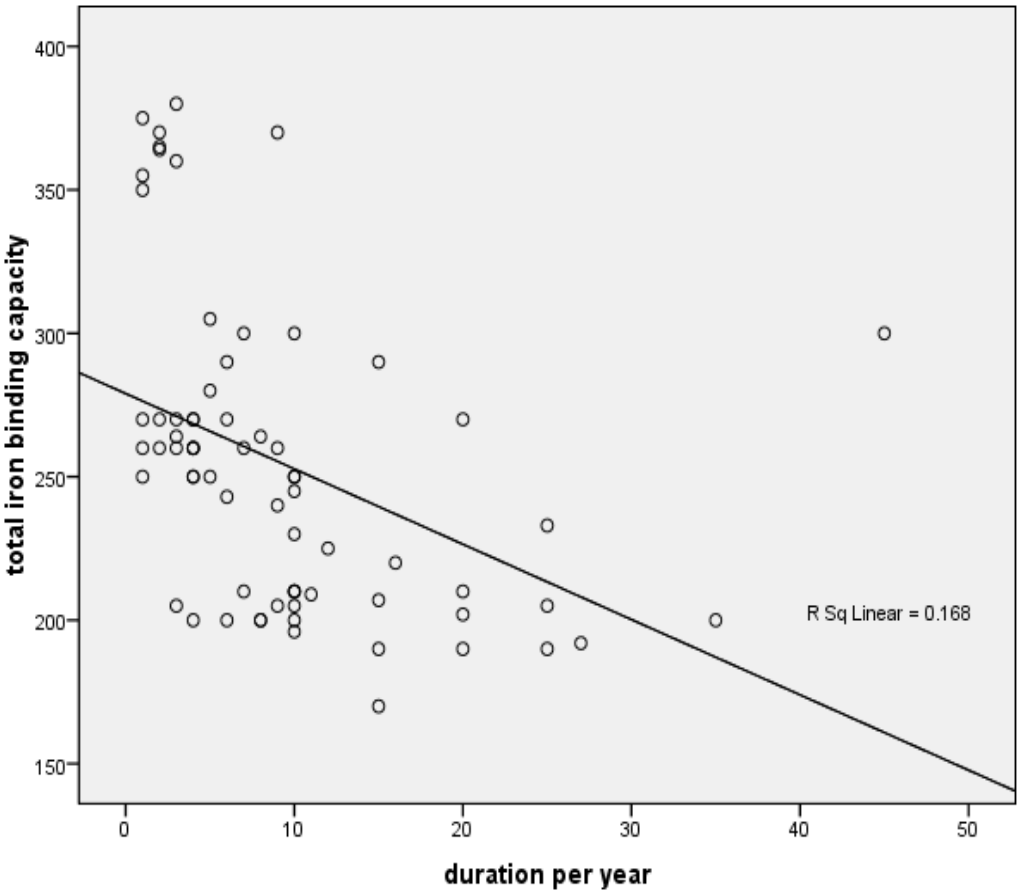

Figure 3.4 :Correlation between total iron binding capacity and duration per year among Sudanese smokers.

(p. value=0.000, r= - 0.410).

4. Discussion, conclusion and recommendations

4.1. Discussion

Cigarette smoking causes minerals disturbances which lead to serious consequences, Smoking leads to tissue hypoxia which leads to inadequate oxygenation of blood circulation that result in erythropoiesis and consequent increased production of erythropoietin (Elzayadi,2006), which enhances erythropoiesis and increases red cell mass above normal level (Balcerzek *et al*, 1975). This leads to increase in the number of destroyed red cells in the normal turnover process which subsequently increases iron overload which causes hepatocellular damage (Bacon *et al*, 1990).

This is a case control study aimed to study the effect of smoking on serum iron and total iron binding capacity. One hundred and twenty Sudanese male (70 smokers and 50 non smokers) were enrolled in this study to study the effect of smoking on serum iron and total iron binding capacity. After evaluation of serum iron and total iron binding capacity by auto analyzer, the statistical analysis was done by using SPSS computer program and the results showed thatserum ironlevel was significantly higher in smoker group when compared to non smoker group and serum total iron binding capacity level was significantly lower than in smoker group when compared to non smoker group (table 3-1). This result agreed with results of study done in India by Pannuru and his team , to show influence of Chronic Cigarette Smoking on Serum Biochemical Profile In Male Human Volunteers, showed that the smokers hadsignificantly higher than the non smokers inserum iron (p.value ≤0.05) and mean ±SD (118.26±5.95)VS(90.83±5.37) (Pannuru, et al ,2009) .

Also the results showed that total iron binding capacity level was significantly low in smoker group when compared to non smoker group(table 3-1) , and this is disagreed with study done in Turkey byBesime and his team conducted in Effects

of smoking on healthy youngmen's hematologic parameters, showed that is dose not significant difference in total iron binding capacitylevel compared to non smoker ($p<0.670$), and mean± SD (330.91±57.19) VS (334.61±54.76)(Besime .*et al*,2014).

Results of this study revealed that increases in serum iron is proportional with duration of smoking per years and number of cigarettes smoked per day and revealed that decrease in serum total iron binding capacity is inversely relation with duration of smoking per years and number of cigarettes smoked per day .

4.2 Conclusion

The study results concluded that:

1. The level of serum iron is significantly increased in cigarette smokers compared to non smokers andlevel of serum total iron binding capacity is significantly decreased in cigarette smokers compared to non smokers.
2. The level of serum iron is positively correlated with number of cigarette per day and duration of smoking per year and level of serum total iron binding capacity isnegatively correlated with number of cigarette per day and duration of smoking per year.

4.3 Recommendations

1. Cigarette smoking is a bigsocial health problem, so that increasing education about serious effect of it is very important.
2. Smokers should estimate iron profile regularly as alarm of tissue hypoxia.

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Appendix1

Sudan University of Science and Technology

College of Graduate Studies

Influence of Smoking on Serum iron and total iron binding capacity among Sudanese Male Smokers

Khartoum state 2015

Questionnaire

Name: ..... No of sample ( ).

Age: .....

Duration of smoking/ years: .....

Number of cigarettes / day: .....

History of other diseases: .....

Results:

Serum iron: ……µg/dl

Serum TIBC: …….… µg/dl