Studying the levels of some Biochemical variables in blood serum for smoking of Tikrit University students

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Abstract. This study included (75) samples of the males students at the Tikrit University. The samples were divided into three groups; the first group: (25) blood samples pulled from healthy non-smoking students (males) as Control sample. The second group: (25 males) samples of diabetic mellitus (smoker students). Third group: (25 males) samples of hypertension (smoking students). The results of the study indicated a significant increase in the concentration of the levels Cholesterol, Triglyceride, Malondialdehyde (MDA), Lactate dehydrogenase (LDH) at level (P <0.005) in the samples of smoker students when compared to the healthy group. And showed decrease in the concentration level of HDL, Glutathione Peroxidase (GPx) at level (P <0.005) when compared to the healthy group (non-smokers).

Keywords. Biochemical variables, Blood serum, Smoking.

1. Introduction

The Antioxidants are a class of chemical substances naturally found in our food which can prevent or reduce the oxidative stress of the physiological system. The body is constantly producing free radicals due to regular use of oxygen. These free radicals are responsible for the cell damage in the body and contribute to various kinds of health problems, such as heart disease, diabetes, macular degeneration, and cancer. Antioxidants being fantastic free radical scavengers help in preventing and repairing the cell damage caused by these radicals [1]. Plants and animals are the abundant source of naturally producing antioxidants. Alternately, antioxidants can also be synthesized by chemical process as well as from the different kinds of agro-related wastes using biological process. Based on their solubility, antioxidants are broadly categorized into two groups: water soluble and lipid soluble. [2] In general, water-soluble antioxidants, such as ascorbic acid, glutathione, and uric acid, have functions in the cell cytosol and the blood plasma. Ascorbic acid is a redox catalyst which reduces and neutralizes the reactive oxygen species (ROS), glutathione has antioxidant properties as reducing agent and can be reversibly oxidized and reduced, while α-tocopherol, carotenoid, and ubiquinol are the examples of lipid-soluble antioxidants and protect the cell membranes from lipid peroxidation [3]. Enzymes are types of antioxidants that come from the protein and minerals we eat as part of our daily diets. These enzymes are synthesized in the human body, and include superoxide dismutase (SOD), glutathione...
peroxidase, glutathione reductase, and catalases. The human body does not produce antioxidant vitamins naturally, so it is essential to include dietary sources of them in our daily intake of food, be it through foods or supplements. Common antioxidant vitamins include vitamins A, C, E, folic acid, and beta-carotene [4]. Free radicals are highly reactive and unstable molecules, usually oxygen molecules, but not always. Their unstable nature is caused by having an unpaired electron. As a result of this unpaired electron, free radicals seek out and take electrons from other molecules, which oftentimes causes damage to the second molecule. When a free radical molecule does this, it is called “oxidation.” A molecule that has had its electron “stolen” from a free radical has been “oxidized.” Molecules that have been oxidized are now transformed into free radicals themselves and will seek to interact with another healthy molecule, thereby creating a vicious chain reaction of electron stealing in the body. When the body has undergone excessive oxidation, or more oxidation than can be combatted, it is said be undergoing “oxidative stress.”[5]. Like many things that occur in nature, free radicals are not only impossible to avoid, but necessary for life. Free radicals help us fight infection, begin the inflammation process that helps repair tissue injury, and short-term oxidative stress may inhibit aging. At the same time, excessive amounts are harmful to humans[6]. Free radicals can form in the body in a number of ways; Environmental exposure, Carcinogens like radiation from the sun, cigarette smoke, air and water pollution, pesticides and herbicides in the food we eat, asbestos and other nasties can cause free radical formation in our bodies. Exercise, while consistent moderate exercise has many positive benefits and can reduce the risk of breast cancer, excessive exercise or inconsistent but vigorous exercise uses a high amount of the body’s oxygen store and as a result, generates excessive free radicals. Stress, the chemicals cortisone and catecholamines created by mental stress can create free radicals. Our bodies, Free radical molecules are a natural byproduct of cell metabolism. Alcohol, consumption of alcohol of any kind of any amount produces free radicals in the body. Fat polyunsaturated fat like that found in vegetables oils is easily oxidized in the body and can create free radicals. Substitute polyunsaturated fats with monounsaturated fat [7]. Some conditions caused by free radicals include; deterioration of the eye lens, which contributes to vision loss, inflammation of the joints (arthritis), damage to nerve cells in the brain, which contributes to conditions (such as Parkinson’s or Alzheimer’s disease), acceleration of the ageing process, increased risk of coronary heart disease, since free radicals encourage low-density lipoprotein (LDL) cholesterol to stick to artery walls, certain cancers triggered by damaged cell DNA [8]. Smoking considers being wide public health problem, which reached today to the level of global epidemical. It is a risk factor for variety of disease (cardiovascular disease, stroke, chronic pulmonary disease, Alzheimer's disease, Parkinson's disease) [9]. About more than 5 million people die from smoking related illnesses, as the report of World Health Organization [10], this number will be doubled by 2025. The main addictive component of smoke are Nicotine, Hydrogen cyanide, Methanol, Butan and about more than 400 other chemicals. These chemicals induced the rate of Reactive Oxygen Species (ROS), which is a part of free radicals. Free radicals are highly unstable and capable of undergoing complex interaction in biological system, make oxidative stress, which occur when there are not enough antioxidant molecules to counteract their side effects [11]. Antioxidant are natural molecules in the biological system that scavenging free radicals or protecting from its effects. They can be synthesized endogenously in the body or determined by food intake [12]. Smoking is a rich source of oxidants. It has been considering the main cause of increase production of (ROS) which may exceed the capacity of antioxidants defense system [13].

2. Materials and Methods

This study has carried out in College of Science (November 2019) to (February 2020) at Tikrit University. The study included (50) samples of the smoker students their ages (21-23) years old. As well as choosing randomly group included (30) sample of (non-smoker students) of age (21-23) years old.
2.1. The samples divided into three groups

First Group, included (25) non-smoker students. Second Group, included (25) diabetic mellitus (smoker students). Third Group, included (25) hypertension (smoker students).

2.2. Method Of Collection The Sample

The blood sample was collected from (75) samples of the students healthy (non-smoker) as well as smoker students of age (21-23) years old. The blood samples were taken from the vein. 10 mL from each student, the blood was put in disposable test tubes. The tubes are empty of (EDTA) in order make Biochemical tests. After that, The blood was left at room temperature for 20 minutes, The blood was separated by using a centrifuge at speed of (3000) rpm for 10 minutes. The serum was extracted using Micropipette, in order to make Biochemical tests which included (Cholesterol, Malondialdehyde, Lactate dehydrogenase, HDL-Cholesterol, Alanine aminotransferase (ALT), Aspartate aminotransferase (AST), Glutathione Peroxidase, Triglyceride, Uric acid, Creatinine.

2.2.1. Measurement of Cholesterol concentration

To evaluate and estimate the Cholesterol in the blood serum, was dependent and using the (Kit) which its equipped (Biolabo) Company made in France with reference number 02160 [14].

2.2.2. Measurement of Triglycerides concentration

It have been used the (Kit) of Triglycerides equipped by (Biomaghreb) Company made in France with number 20133[15] to estimate and evaluate the Triglycerides enzyme in the blood serum.

2.2.3. Measurement of HDL-Cholesterol concentration

It have been used the (Kit) of HDL-Ch., equipped by (Biolabo) Company mad in France with number 02160 [14] to evaluate and estimate the HDL-Ch in the blood serum.

2.2.4. Measurement of Lactate dehydrogenase

It have been measured (LDH) in the blood serum by the following the steps of attached with inspection (Kit) provided by( Biolabo) Company mad in France [16].

2.2.5. Measurement of Glutathione Peroxidase

It have been measured (GPx) in the blood serum by the following the steps of attached with inspection (Kit) provided by( Biolabo) Company mad in France [17].

2.2.6. Measurement of Malondialdehyde

It have been measured (MDA) in the blood serum by the following steps of attached with inspection (Kit) provided by( Biolabo) Company mad in France [18].

2.3. Statistical analysis

The data of the samples study were collected and analysed statistically by using program (SAOOS) of windows. Including (Mean ±S.D). The significant differences at level (P <0.005)[19].
3. Results and Discussion

The results in (Figure 1) showed significant increase in the Cholesterol concentration under levels of \((P < 0.005)\) in the smoker students as compared with in other groups, and this agree with results study of [20]. This increase of the Cholesterol concentration might be because high activity Cholesterol transferase responsible about Cholesterol absorption intestine and then induce as a result of oxidative stress which infect B-cell in pancreatic by the effect of reactive oxygen species [21]. On the hand, this results was revealed to the change in the concentration of the cholesterol is caused by the smoking which increase Cholesterol levels in the blood serum. The Smoking increases inflammation in the body when occurs chemicals in cigarette smoke injure cells, causing swelling and interfering with proper cell function. Also, the Smoking causes oxidative stress, a condition that occurs as chemicals from cigarette smoke combine with oxygen in the body [22] and all these reasons causes ledes to damage of cells. Evidence strongly suggests that both inflammation and oxidative stress may be related to an increased risk of diabetes. The Smoking causes rise both of Hypertension and glucose level in the body, so the Smoker people are less response for the insulin and subsequently the glucose rises in their blood causing Hypertension [23].

Table 1. Arithmetic average for studied croups according to the Disease kind and smoking kind (Heavy smokers).

| The samples                        | NO. | Cholesterol mmol/L | Triglyceride mmol/L | HDL mmol/L  |
|------------------------------------|-----|--------------------|---------------------|-------------|
| Control group                      | 25  | 4.2±2.3            | 1.7±0.3             | 1.51±0.01   |
| group (diabetic mellitus)          | 25  | 6.8±3.5            | 5.6±3.2             | 1.02±0.03   |
| group Hypertension                 | 25  | 7.4±4.6            | 6.2±4.1             | 1.03±0.02   |

Figure 1. Illustrated the concentration of the cholesterol in the smoker students and compare with in other groups.

The results in (Figure 2) showed significant increase in the triglyceride concentration levels \((P < 0.005)\) in the smoker students as compared with in other groups, and this agree with results study of [24], [25] during his study on the association between the lipid profile and chronic smoking. Both research above also found very low levels of HDL, with an increase in the levels of TC, in smokers and these
Results were an agreement with the finding of our study. Previous studies have reported that smokers have higher serum triglyceride (TG) and blood glucose concentrations and lower high-density lipoprotein cholesterol (HDL-C) concentrations than non-smokers [26].

![Figure 2](image1.png)

Figure 2. Illustrated the concentration of the triglycerides in the smoking students and compare with in other groups.

The results in (Figure 3) showed significant decrease in the HDL-Cholesterol concentration levels (P <0.005) in the smoking students as compared with in other groups, smoking is associated with reduced HDL cholesterol levels. Cigarette smoking can alter the critical enzymes of lipid transport, lowering lecithin: cholesterol acyltransferase (LCAT) activity and altering cholesterol ester transfer protein (CETP) and hepatic lipase activity, which attributes to its impact on HDL metabolism and HDL subfractions distribution. In addition, HDL is susceptible to oxidative modifications by cigarette smoking, which makes HDL become dysfunctional and lose its other protective properties in smokers. Therefore, cigarette smoking has a negative impact on both HDL quantity and function, which can explain, in part, the increased risk of cardiovascular disease in smokers [27, 28].

![Figure 3](image2.png)

Figure 3. Illustrated the Concentration of the HDL-cholesterol in the smoker students and compare with in other groups.
Table 2. Arithmetic average for studied croups according to the disease kind and smoking kind (Heavy smokers).

| The samples                      | NO. | Malondialdehyde (MDA) ug/l | Lactate dehydrogenase (LDH) u/l | Glutathione Peroxidase (GPx) u/l |
|----------------------------------|-----|----------------------------|---------------------------------|---------------------------------|
| Control Group                    | 25  | 2.65±1.33                  | 181.8±1.02                      | 1.53±0.24                       |
| group (diabetic mellitus)        | 25  | 5.67±3.2                   | 277.8±133.1                     | 0.191±0.180                     |
| group Hypertension               | 25  | 6.4±4.1                    | 302±214.2                       | 0.195±0.183                     |

The results in (Figure 4) In this study MDA levels were found to be significantly (p<0.005) raised in smokers. High serum levels of MDA in smokers could have amplified production of reactive oxygen species (ROS) which in turn might have accelerated lipid peroxidation manifested by increased levels of MDA, as well as the role of oxidative stress on glucose metabolism in a high-risk of diabete. ROS can activate specific pathways whose products interfere with insulin signalling, the activation of the redox-sensitive nuclear factor-kappa beta (NF-kB) leads to the expression of cytokines such as temporal necrosis factor α (TNF-α), and interleukins (ILs) such as IL-1α and IL-6 and all these products have a quenching effect on insulin signalling [29, 30, 31].

![Figure 4](image)

Figure 4. Illustrated the Concentration of the malondialdehyde in the smoker students and compare with in other groups.

While in the (Figure 5) findings showed significantly increasing in LDH level of smoker’s group. Lactate dehydrogenase enzyme is found in almost every tissue in the body and its level increase in blood in many tissues damage pathological disorders. We think that the reasons for elevated of LDH significantly due to the smoke chemicals compounds may affect the respiratory tract as well as all of the cells in respiratory system [32]. The increased of LDH considered as indicator of cell necrosis or tissues damage or may be attributed on the smoking induced cell damage anywhere in the body, which may leak cellular contents along with LDH into serum [33]. Elevated of LDH levels is usually considered to reflect cell damage, and this is agree with the bad effect of smoking in generated free radicals and oxidative stress in cell which lead to release more of LDH to the blood stream by cell injury [34].
The results in (Figure 6) showed Glutathione Peroxidase levels were found to be significantly (p<0.005) lowered in smokers than in non-smokers. The significantly differences of oxidative enzymes levels GPx may be due to nicotine increased generation of superoxide anion and hydrogen peroxide, which in turn results in generation of hydroxyl free radicals. Generation of these free radicals have been shown to participate in many toxic reaction and increased production of superoxide and hydrogen peroxide may cause deleterious injury to alveolar macrophages by causing release of proteolytic enzymes [35].

**Table 3.** Body Mass Index (Kg/m²) sample of the study.

| (BMI)          | mean±S.D  |
|---------------|-----------|
| Control Group | 23.62±0.52|
| smoking students | 26.23±0.83|
Figure (7) shows the relationship of Body Mass Index (BMI) among the studied samples, as there was a statistically significant (p < 0.05). The results rates increase between the BMI of smokers’ students compared with the control group. Smoking and obesity are major public health challenges and the prevalence of both is increasing globally. Smoking increases the risk of cancer, respiratory and cardiovascular diseases, and is the leading preventable cause of death in developed countries [36]. Obesity is the fifth leading cause of death, globally, and accounts for 44% of cases of diabetes and 23% of ischaemic heart disease [37].

4. Conclusion

The smoking effects on the levels of the Cholesterol, Triglyceride, Malondialdehyde (MDA), Lactate dehydrogenase (LDH) (increase) and decrease in the concentration level of (HDL-Cholesterol, Glutathione Peroxidase (GPx) in the smoker students, and smoking effect on the diabetic mellitus, hypertension and Body Mass Index.

5. References

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