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Effects of cigarette smoking on hematological parameters in male smokers in Al-Bayda city, Libya

Khaled S. Al Salhen[1*], Rhab D. Abdalslam[1]
[1] *Department of Chemistry, Science College, Omar Al-Mukhtar University, Libya,*
[*]*Email: Khaledk639@yahoo.co.uk*

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Abstract

Tobacco smoking use is widely spread throughout the world. Tobacco smoking has been claimed to cause a wide variety of health problems such as atherosclerosis, mutagenesis of exposed cells and cancer. The effects of smoking on human health are serious and in many cases, deadly. The present study was done to investigate the effect of cigarette smoking on some hematological parameters in male smokers. The study is carried out on thirty Libyan male smokers, who smoked at least 10 cigarettes per day for at least15 years. Their age ranged between 30-60 years. Control (non-smokers) group was collected with the same range of age for statistical comparison. The results of the study revealed a significant increase of hematological parameters (Hb: Hemoglobin; PCV: Packed cells volume; RBC: Red blood cell and WBC: White blood cell) in smoker group when compared with the control. No significant difference of the platelet count was recorded in smoker group when compared with control. Furthermore the level of malondialdehyde (MDA), which is an indicator of lipid peroxidation and oxidative stress significantly increased in cigarette smokers group when compared with control group. To conclude, cigarette smoking leads to oxidative stress by free radical generation (Reactive oxygen species-ROS) by the mechanism of lipid peroxidation. Smoking exerts negative influence on the hematological parameters and these are the contributing factors that lead to cardiovascular and atherosclerosis that usually occur in cigarette smokers and cause of death.

***Keywords*:** Cigarette smoking (CS), hematological parameters, malondialdehyde (MDA), blood cell count

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Introduction

Although tobacco has dangerous effect on human health, it still highly consumed throughout the world (Benowitz et al., 1988). Smoking is one of the most common addictions of modern times. It has been implicated as an etiological agent for various chronic diseases, including a variety of infection, cancers, heart diseases and respiratory illnesses (Mehta et al., 2008; Zhong et al., 2008). Cigarette smoke (CS) contains over 4000 compounds, including at least 200 toxicant, 80 known or suspected carcinogens, large quantities of oxidants and free radicals that induce oxidative stress (Abel et al., 2005; Carel and Eviatar, 1985; De Heens et al., 2008). Moreover, cigarette smoking generates many toxic and carcinogenic compounds harmful to the health, such as nicotine, nitrogen oxides, carbon monoxide, hydrogen cyanide and free radicals (Hoffmann et al., 2001).

Smoking has both acute and chronic effect on hematological parameters (Gitte, 2011). He found the mean plasma platelet count of smokers increased significantly compared with the non-smokers. During past decade, it was suggested that cigarette smoking affect the blood characteristics as well that leads to death (Asif et al., 2013; Aula and Qadir, 2013; Soldin et al., 2011). In number of studies, it has been found relationship between smoking and white and red blood cell counts (Asif et al., 2013; Mukherjee and Chatterjee, 2013; Wannamethee et al., 2005; Tiel et al., 2002; Friedman et al., 1996; Yarnell et al., 1991). Although in some studies suggested that increase in hemoglobin level in blood of smokers could be a compensatory mechanism (Asif et al., 2013; Dass et al., 2013; Mukherjee and Chatterjee, 2013). However, some were of view that smoking does not increase in hemoglobin level in all smokers and this relates to tolerance potential of individual to different kind of diseases (Asif et al., 2013; Tarazi et al., 2008). This study aimed at determining the effects of cigarette smoking on several hematological parameters in male population of Al-Bayda city in Libya.

Material and methods

Design of the study

This study was conducted among thirteen Libyan male voluntary cigarette smokers (test) and thirteen male non-smokers (control). Their age varied between 30-60 years. Two groups (smokers and non-smoker's) were collected with the same range of age for statistical comparison. The subjects are including smokers (n = 30) and non-

smokers as control group (n = 30). The smoker's were regularly consuming minimum of 10 cigarettes per day for at least 15 years. The smoker's were collected in El-Bieda city during the period (November - December 2013). The enrolled subjects did not have any serious health problem; no history of drug usage and none had donated or received blood in last 6 months. The clinical data, medical history and other relevant information were collected from subjects by personal interview.

Blood sampling

5 ml of whole blood samples were drawn by venipuncture from each member and placed in a heparinized tube and centrifuged at 3000 rpm for 15 minute. Then complete blood counts (CBC) were estimated within 1-2 hours of blood sampling on Selectra E fully automatic hematological analyzer from Hungary was calibrated by standardized commercially available calibrated kit. CBC counts (WBC, RBC, Hb, PLT and PCT) were measured in this study.

Determination of plasma malondialdehyde (MDA)

The assessment of the lipid peroxidation process is achieved via determining the end product MDA. The level of plasma MDA was determined spectrophotometrically with a thiobarbituric acid (TBA) solution. In brief, to 150 µl plasma sample the following were added: 1ml (17.5%) trichloroacetic acid (TCA) and 1ml of 0.66% TBA, mixed well by vortex, incubated in boiling water for 15 minutes, and then allowed to cool. One ml of 70% TCA was added and the mixture allowed to stand at room temperature for 20 minutes, centrifuged at 2000rpm for 15 minutes, the supernatant was taken out for spectrophotometer assay at 532nm (Figure 1) (Muslih et al., 2002).

The concentration of MDA calculated as follow:

$$MDA\ (\mu mol/l)\ = \frac{Absorbance\ at\ 532\ nm}{L\ \times E_0}\ \times D\ \times\ 10^6$$

L: light path (1cm).

E_0: Extinction coefficient $1.56\times10^5\ M^{-1}.\ cm^{-1}$

D: Dilution factor.

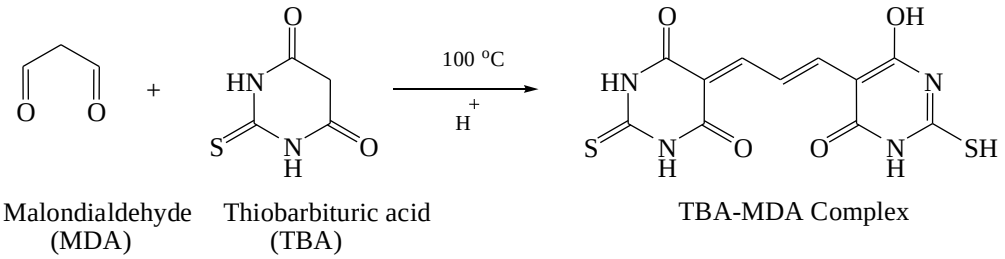

Figure 1. Thiobarbituric acid reaction (Gerard-Monnier et al., 1997).

Statistical analysis

Mean and standard deviation (SD) were calculated for all the hematologic and biochemical parameters. Unpaired t test were applied to test the significance of variance ($p<0.05$) of the parameters under study between control and smoker group.

Results and Discussion

Table (1) demonstrates general characteristics of Libyan male smokers and non-smokers groups. The mean age of male smokers and non-smokers were 49.11 ± 10.53 and 47.97 ± 9.83 years, respectively. The non-smoker subject was having mean weight of 73.13 ± 13.16 and smokers were having 71.82 ± 11.20 kg.

Table 1. General characteristics of smoker and non-smoker group

Characteristics	Control (Non-smoker) (n=30)	Smokers (n=30)
Age (years)	47.97 ± 9.83	49.11 ± 10.53
Weight (kg)	73.13 ± 13.16	71.82 ± 11.20

Data are expressed as mean ± SD; (n) the number of subjects.

Table (2) presents the level of MDA in smokers group significantly increased with mean value (3.856 µmol/l ± 0.213) when compared with its control group (2.637 µmol/l ± 0.253). This result is similar to Aula and Qadir (2013), Durak et al. (2002) and Schmid et al. (1996) published reports. CS is known to contain a large number of oxidants; it has been hypothesized that many of the adverse effects of smoking may

result from oxidative damage to critical biologic substances (Skurnik and Shoenfeld, 1998). Two major phases were identified in CS: a tar phase and a gas phase; both phases are rich in oxygen-centered, carbon-centered and nitrogen-centered free radicals as well as non-radical oxidants. From the analysis of each phase, it was estimated that a single cigarette puff contains approximately 1014 free radicals in the tar phase, and 1015 radicals in the gas phase. These include various compounds, which are capable of causing an increase in the generation of various ROS like superoxide ($O_2^{\bullet-}$) hydrogen peroxide (H_2O_2), hydroxyl ($OH^{\bullet}$) and peroxyl ($ROO^{\bullet}$) radicals. These ROS, in turn, are capable of initiating and promoting oxidative damage in the form of lipid peroxidation (Pasupathi et al., 2009). Durak et al. (2002) suggested that smoking creates a significant oxidant load in the erythrocytes. As a result, toxic free radicals and other oxidant substances in CS damages unsaturated fatty acids and some other oxidation- sensitive structures in the erythrocytes leading to increase MDA level.

In a recent study, increased MDA levels have been reported in blood samples from chronic smokers compared to non- smokers (Aula and Qadir, 2013). In some other studies it has been established that smokers need more antioxidant vitamins, suggesting that antioxidant supplementation is vital for smokers and scavenging potentially harmful free radicals produced by CS (Brown et al., 1994; Diplock, 1987).

Table 2. Comparison effect of malondialdehyde (MDA) in smokers and non-smokers group

Parameters	Control (Non-smokers) (n=30)	Smokers (n=30)	P value
MDA (μmol/l)	2.637 ± 0.253	3.856* ± 0.213	0.000

Data are expressed as mean ± SD; (n) the number of subjects.*represents significant at $P<0.05$.

Table (3) shows the changes in hematological parameters of smokers and non-smokers: WBC, RBC, Hb and PCT were significantly high; PLT did not show any significant difference. Smokers had significantly higher level of WBC count than non-smokers control group (11.74 $x10^9l^{-1}$ ± 0.65 vs. 6.95 $x10^9l^{-1}$ ± 0.65). Some of the adverse effects of smoking include: initiation of endothelial injury (Pittilo, 2000). Detecting endothelial damage may be the most useful step in the early diagnosis of

atherosclerosis. Although the endothelium releases many molecules into the circulation and arterial wall, not all of them are specific to the endothelium and are therefore of limited research or diagnostic potential (Blann and Lip, 1998). WBC count is perhaps the most useful, inexpensive and simple biomarker for endothelial damage. In fact several studies have shown that WBC count is an independent predictor of atherosclerosis and cardiovascular disease (Loimaala et al., 2006; Madjid et al., 2004). In this study, the high WBC count in male smokers is consistent with other published reports (Asif et al., 2013; Aula and Qadir, 2013; Al-Awadhi et al., 2008; Rajasekhar et al., 2007; Gregory et al., 2005; Wannamethee et al., 2005; Smith et al., 2003; Blann et al., 1998; Freedman et al., 1996; Schwartz and Weiss, 1994; Noble and Penny, 1975; Burney and Bonus, 1972; Corre et al., 1971). Smoking in its own right increase inflammation and oxidative stress (Tarazi et al., 2008). Our study indicates that CS is associated with changes in inflammatory biomarker level, such WBC count, and these may be due to CS containing many toxic and carcinogenic compounds harmful to healthy (Asif et al., 2013; Aula and Qadir, 2013; Frohlicha et al., 2003; Hoffmann et al., 2001; Hansen et al., 1990) that can induce inflammatory processes. CS has been shown to be associated with an elevated peripheral blood leucocyte count (Schwartz and Weiss, 1994) and has a powerful influence on WBC count (Frohlicha et al., 2003), which may be a biomarker of exposure to oxidants (Crowell and Sarnet, 1995). One of the possible mechanisms of increasing of total WBC may be due to the glycoprotein from the tobacco leaf, which can stimulate lymphocyte proliferation, and differentiation by interacting with a specific membrane component, as occur in antigenic response (Freedman et al., 1996). The mechanism for smoking-induced increase in WBC count is not clear. It has been suggested that inflammatory stimulation of the bronchial tract induces an increase in inflammatory markers in the blood but it has also been suggested that nicotine may induce an increase in blood lymphocyte counts (Calapai et al., 2009; Geffken et al., 2001). While leukocytosis may simply be a marker of smoking-induced tissue damage, the high a count can promote cardiovascular diseases through multiple pathologic mechanisms that mediate inflammation, plug the microvasculature, induce hypercoagulability and promote infarct expansion (Loimaala et al., 2006; Madjid et al., 2004). In fact several studies have shown that WBC count is an independent predictor of atherosclerosis and cardiovascular disease (Loimaala et al., 2006; Madjid et al., 2004). The high WBC count ($P<0.05$) in our male smoking subjects may also suggest that they might be at greater risk for developing cardiovascular diseases than non- smokers.

In this study, we found increased MDA level of smokers compared to non-smokers (Table 2). These results suggest that smoking creates a significant oxidant loading to increase in the leukocytes counts may indicate to an activation of the human's defense mechanism and immune system. Therefore, the smokers group also has a higher white cell count than non-smokers have suggested that chronic tissue damage is a possible mechanism.

The Red blood cell (RBC) count also showed significant increase in smokers than non-smokers ($6.91\ x10^{12}l^{-1} \pm 0.65$ vs. $3.86\ x10^{12}l^{-1} \pm 0.41$) (Table 3). RBC values were significantly high in smokers than those of non-smokers ($P<0.05$) and these results agree with the previous studies on male smokers (Kume et al., 2009; Ho, 2004; Bain et al., 1992; Levenson et al., 1987).

Levenson et al. (1987) and Ho (2004) reported that increase the level RBC and Hematocrit are associated with blood viscosity and clotting in smokers. High level of RBC is termed as polycythemia and very high RBC mass slows blood velocity and increase the risk of intravascular clotting, coronary vascular resistance, decreased coronary blood flow, and a predisposition to thrombosis (Ravala and Paula, 2010). This elevation may lead to congenital heart disease, pulmonary fibrosis and elevated erythropoietin (Kume et al., 2009; Milman and Pedersen, 2009; Tarazi et al., 2008; Tiel et al., 2002). The mechanism by which polycythemia causes thrombosis is still under investigation, but smoking cigarettes creates a unique condition of combined polycythemia to hypoxia consequent chronic lung disease, leading to elevated red cell production due to an elevated carboxyhemoglobin level, with concomitant plasma volume reduction. Overall, thrombosis is a serious complication of polycythemia and can lead to death in up to 8.3% of patients (Ravala and Paula, 2010).

Similarly, smokers had significantly higher mean hematocrit (PCV) 44.52 ± 4.99 % than non-smoker's 34.17 ± 2.91 % and hemoglobin values were also significantly higher in smokers 16.14 ± 1.39 g/dl than non-smokers 13.84 ± 0.54 g/dl (Table 3).

The extremely higher value ($P<0.05$) observed for PCV in Libyan smokers in this study, agrees with Isabell and Hagerup (1971). This was explained by the increase in carbon monoxide level in the blood of smokers (Bashiru et al., 2006). Furthermore, hematocrit values were also significantly high in smokers than those of non-smokers and are in accordance with the results of Tarazi et al., (2008); Tiel et al. (2002) and Kume et al. (2009) in male smokers. Higher levels of hematocrit may cause

polycythemia vera (PV), a myeloproliferative disorder in which the RBCs are produced excessively by bone marrow, and also related to an increased risk of development of atherosclerosis and cardiovascular disease (Ferro et al., 2004).

Smoking is associated with an increase in hematocrit or red blood cell count, this was also noticed in this work and the increased may be attributable to increased level of carbon monoxide (CO) and carboxyhaemoglobin (Haustein et al., 2004). The results of Burtis and Ashwood also showed that differences observed might be due to the increased level of carboxyhaemoglobin in smokers, thereby creating continuous state of hypoxia (Burtis and Ashwood, 2001). Any condition that causes the quantity of oxygen transport to the tissue to decrease ordinarily increases the rate of red cell production by stimulating erythropoietin secretion (Oke et al., 2012). The increased number of red cells compensates for impaired ability of red cells to transport oxygen. The blood erythrocyte count is therefore increase in smokers (Burtis and Ashwood, 2001). In cigarette smoking, carbon monoxide (CO) is produced by the incomplete combustion of carbon-containing material. CO has a very high affinity for hemoglobin relative to that for oxygen (approximately 200-fold) (Carallo et al., 1998). Thus, CO displaces oxygen from hemoglobin in red cells to produce carboxyhemoglobin, which reduces the release of oxygen to tissues (Cronenberger et al., 2008). Higher levels of hematocrit and hemoglobin have been demonstrated in smokers, and these increases are likely to be compensatory for exposure to CO (Roethig et al., 2010). Increased hematocrit and hemoglobin concentrations observed in smokers that may contribute to a hypercoagulable state (Leroy et al., 2012; Cronenberger et al., 2008).

Results illustrated in table (3) showed significant increase ($P<0.05$) in hemoglobin concentration of smokers group. These finding are in agreement with several studies done in the past with volunteer smokers have proven that great percentage of the smokers have the elevated hemoglobin level (Asif et al., 2013; Mahsud et al., 2010; Kume et al., 2009; Bashiru et al., 2006; Bain et al., 1992). An elevated hemoglobin concentration is usually the result of increased red blood cell production as a compensatory mechanism when blood oxygen carrying capacity is compromised to meet the demand of tissue (Carallo et al., 1998).

Similar changes occur on acute exposure to CO (Ramsey, 1969), which may therefore be the mediator of this effect. CO may act by increasing capillary responsible for low plasma volume of smokers with relative polycythaemia is increased venous tone

(Velasquez et al., 1974), possibly attributable to catecholamine release induced by nicotine (Jackson and Spurr, 1978). Our study demonstrates that acute change in Hb, PCV and RBC attributable to change in plasma volume are seen in smokers in general.

On the other hand mean of platelets count statistically did not change ($P<0.05$) in smokers group with mean value (244.62 ± 50.78) when compared with the control groups (243.23 ± 52.64) (Table 3).

This result is in agreement with previous results by Asif et al. (2013); Al-Awadhi et al. (2008); Butkiewicz et al. (2006) and Suwansaksri et al. (2004) who reported that there is no statistically significant difference in platelet count between male smokers and non-smokers. According to Blann et al. (1998), smoking two cigarettes a day by chronic smokers of both sexes do not affect the platelet count; Hawkins (1972) also appears to have substantiated the findings of the present study. She observed no significant difference between the platelet counts of non-smokers, light smokers, and heavy smokers.

Various reports have focused on the influence of smoking on platelets because of a possible association between smoking and alteration of blood platelets. Some of these results showed an increase of platelets turnover and a decrease of platelet survival in smokers; increased destruction of platelets, however, was not sufficient to reduce the number of circulating platelets (Fuster et al., 1981).

Table 3. Comparison of hematological parameters in control and smoker's group

Parameters	Control (Non-smoker) (n=30)	Smokers (n=30)
Hb (g/dl)	13.84 ± 0.54	16.14* ± 1.39
PCV (%)	34.17 ± 2.91	44.52* ± 4.99
RBC ($x10^{12}l^{-1}$)	3.86 ± 0.41	6.91* ± 0.91
WBC ($x10^{9}l^{-1}$)	6.95 ± 0.65	11.74* ± 2.55
PLT ($x10^{9}l^{-1}$)	244.62 ± 50.78	243.23 ± 52.64

Data are expressed as mean ± SD; (n) the number of subjects. *represents significant at $P<0.05$. Hb: Hemoglobin; PCV: Packed cells volume; RBC: Red blood cell; WBC: White blood cell; PLT: Platelets.

Conclusion

In conclusion, in the present study we found that male smokers have a higher plasma concentration of MDA compared with non-smokers. Smoking exerts negative influence on the hematological parameters (e.g. hemoglobin, WBC count, RBC count and PCV). In our result Hb, RBC, WBC counts and PCV are significantly higher in smokers. Too many blood cells or the high level RBC, WBC and PCV can make the smoker's blood more viscous so the blood does not flow efficiently and can contribute to the formation of clots. This can increase the risk of clotting complications, such as stroke, heart attack, deep vein thrombosis or pulmonary embolism.

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تأثير تدخين التبغ على مكونات الدم لدى الذكور المدخنين في مدينة البيضاء– ليبيا

خالد سالم الصالحين و رحاب ذاود عبد السلام

الملخص

ينتشر تدخين التبغ على نطاق واسع في العالم. يسبب التدخين مجموعة واسعة من المشاكل الصحية مثل تصلب الشرايين وطفرات الخلايا المسببة للسرطان. تأثير التدخين على صحة الإنسان خطير و يؤدي في كثير من الحالات الى الوفاة. أجريت هذه الدراسة من اجل تقييم تأثير التدخين على مكونات الدم لدى الذكور المدخنين بمدينة البيضاء في ليبيا. تضمنت الدراسة ثلاثين عينة من الليبيين الذكور المدخنين الذين يدخنون مالا يقل عن 10 سيجارة يوميا ولفترة تدخين لا تقل عن 15 عام. تضمنت مجموعة المدخنين الفئة العمرية من 30–60 عاما. وتم اختيار مجموعة السيطرة (غير المدخنين) من نفس الفئة العمرية وذلك من اجل المقارنة الاحصائية. أوضحت النتائج عن زيادة معنوية في مكونات الدم (هيموجلوبين و حجم الخلايا المعبأه و كريات الدم الحمراء والبيضاء) لدى المدخنيين مقارنة بمجموعة السيطرة. وكذلك اظهرت النتائج عدم وجود فروقات معنوية في الصفائح الدموية لدى المدخنين مقارنة بمجموعة السيطرة. بالاضافة الي ذلك توجد زيادة معنوية في المالوندايالديهايد (MDA) التي تعبر عن مؤشر حالة الاجهاد التأكسدي وبيروكسيد الدهون لدى المدخنين مقارنة بمجموعة السيطرة. مما سبق يمكن أن نستنتج أن التدخين يؤدي الي الاجهاد التأكسدي من خلال توليد الشقوق الحرة الضارة (أنواع ألاوكسجين الفعالة ROS) و ألية عمل بيروكسيد الدهون. يتضح من هذا الدراسة التاثير السلبي للتدخين علي مكونات الدم التي تؤدي الى مرض تصلب الشرايين وأمراض القلب الوعائية التي تحدث عادة لدى المدخنين وتسبب لهم الوفاة.

مفتاح الكلمات: تدخين السجائر، مكونات الدم، المالوندايالديهايد ، عدد خلايا الدم