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EFFECT OF EMITTED POLLUTANTS FROM CRUDE OIL REFINING ON BIOCHEMICAL BLOOD VARIABLES OF IN-SITE WORKERS

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Abstract

This study investigates the effect of emitted smokes from conventional crude-oil refining process on blood and lipids variables of in-site workers in Nineveh province, Iraq. The emitted smokes were classified into white and black smokes. The effect of smokes and exposure durations on blood and lipids variables were investigated. The investigated blood variables are haemoglobin (Hb), Packed Cell Volume (PCV), platelets, Red Blood Cells (RBCs), total proteins, uric acid, alanine transaminase (ALT), Aspartate Transaminase (AST), in addition to some blood lipids, namely cholesterol, triglycerides, Very Low-Density Lipoprotein (VLDL), Low-Density Lipoprotein (LDL) and High-Density Lipoproteins (HDL). Samples were directly collected inside the combustion area and tested at Ibn Sena hospital - Mosul. Data were analysed by one-way Analysis of Variance (ANOVA) using Student-Newman-Keuls and Duncan's multiple range test methods. Results show that exposure to both white and black smokes for 6 months has significantly increased the values of platelets, RBCs, AST, ALT and all blood lipids concentrations, while the other blood variables were significantly decreased compared to control. The Hb, PCV, total protein, AST and ALT were more sensitive to black smoke, whereas platelets, RBCs and uric acid were more sensitive to white smoke. The impact of 12 months of exposure duration on platelets, RBCs, ALT, AST, and all blood lipids values has significantly greater than the 6 months. In contrast, the exposure duration of 6 months showed a significant decrease in Hb and PCV as well as uric acid values. The different components of smokes lead to undesirable increment or decrement in blood variables that leads to many health problems and chronic diseases.

Keywords: Blood lipids, Blood variables, Crude oil spills, Oil combustion, Oil refining, Polluted air, Smoke toxicity.

1. Introduction

Environmental pollution can be identified as "the contamination of the physical and biological components of the earth/atmosphere system to such an extent that normal environmental processes are adversely affected" [1]. The polluted workplaces are very critical to the workers' health, and many diseases could occur mainly in polluting industries. In general, there are three types of acquired diseases: medical-personal diseases, work diseases and occupational diseases. These diseases can be generated as a reason of indiscernible process behaviours such as inefficient materials incineration and direct waste disposal to the environment. The high pollution levels of some industrial cities were negatively affected human strength and capacity and reduced his mental and physical abilities [2]. The released hydrocarbons (HC) from inefficient incineration of organic compounds as coal, crude oil, gases, and organic wastes have direct impacts on human health specifically on field-workers [3]. In Iraq, HC compounds are in every ecosystem due to wars, cars emissions, industrial gases, wastes, oil incineration, and many other sources.

During the years 2015 and 2016, the emitted smokes from crude-oil combustion areas of Nineveh province is an example of air pollution that negatively affected the health of on-site workers. The combustion areas were located in Badush, Muhallabiyah, and Hamam Al-Alil districts in Nineveh province, North of Iraq. The crude oil has been directly burned inside huge halls made on open lands to generate energy for crude oil refining process and block the visibility on satellites. The generated heat from oil combustion was used to heat a local-made oil-refining chamber for gasoline, diesel, and fuel oil production, as shown in Fig. 1. This critical incident has generated big smoke plumes and created huge clouds over the combustion areas for more than one year. The hazard sources on human health inside oil-refining areas are direct contact with oil spills and inhalation of emitted smokes. According to sites managers, many workers died because of high levels of pollutants, whereas the other workers were suffered from different kinds of blood and respiratory as well as skin diseases.

In this research, blood samples were randomly collected from two groups: the first group consists of in-site workers (workers-samples); while the second group belongs to unexposed people to emitted smokes (the control-samples). To investigate the effects of smokes on the workers-samples; specific blood variables were selected and measured for both groups.

Then, the results of the first group were compared to the results of the second group to investigate whether the workers-samples are higher, equal, or lower than the control samples. The selected blood variables are haemoglobin (Hb), Packed Cell Volume (PCV), platelets count, Red Blood Cells (RBCs), total proteins, uric acid, Alanine Transaminase (ALT), Aspartate Transaminase (AST). Further, specific blood lipids for workers-samples were also investigated and compared with control samples. The selected blood lipids are cholesterol, triglycerides, Very Low-Density Lipoprotein (VLDL), Low-Density Lipoprotein (LDL), High-Density Lipoproteins (HDL). The variations in blood variables between the exposed workers to white smoke with the exposed workers to black smoke were compared. The effect of exposure duration of 6 and 12 months have also been investigated on the selected blood variables.

1.1. Oil spills toxicity

As well known, crude oil contains organic and inorganic components as well as toxic constituents for humans, such as benzene, toluene, ethylbenzene, o-xylene, naphthalene, bentonite, anionic polyacrylamide, sodium carbonate, polyaromatic hydrocarbons and many more. In crude oil combustion areas, the in-site workers are facing the danger of the direct contact with oil spills especially if they leak to safety and protective clothing. The direct or indirect contact with toxic constituents can cause serious health problems, which increase the risk on workers side by side with the emitted smoke inhalation. Researchers found that oil spills could change blood-variables values, further; it can cause eyes and skin irritation, lung tissues, nervous system disorders, anaemia and cancer [2, 4].

Benzene is a toxic component to blood and bone marrow. It can cause several diseases such as aplastic anaemia and leukaemia and react with protein albumin in haemoglobin (Hb) in Red Blood Cells (RBCs) and forms protein adduct [5]. Further, benzene can affect blood variables by changing blood urea nitrogen, and White Blood Cells (WBCs), as well as platelets, count below the average values, leading to hepatic and blood-related diseases [6, 7].

Polycyclic Aromatic Hydrocarbons (PAHs) is another hazardous constituent that can also react with Hb and form PAH-hemoglobin adducts [8]. PAHs can be released from the combustion of crude oil and cause skin cancer [3, 9]. Carbon dioxide (CO) can bind with Hb to form Carboxyhemoglobin (COHb) which cannot carry O_2 around the body [10]. Crude oil contains heavy metals such as iron, copper, cadmium, nickel, arsenic and lead. These pollutants enter the body through ingestion, inhalation, skin, or consumption of polluted species.

Human blood is act as a pollutants carrier by transfer the contaminants through the body that could to be stored lately in the body tissues such as liver and kidneys [11]. The enzymes of the human body can be affected by oil spills pollutants in terms of the production levels and their enzymatic activities [12]. Prasad and Oberleas [13] illustrated the inhibition of human enzymatic activities by heavy metals and clarified how the immune and nervous systems could be affected. Arsenic is considered the most effective chemical on the skin. Although the low concentration in curd oil (about 0.84 mg/L); it can cause skin disorders and skin cancer [2, 14].

1.2. White and black smoke plums

The released smoke-plums from burning crude oil in Nineveh province were either white or black. Some sites had white smoke plums; others had black plums or both depending on oil chemical composition and availability of some constituents.

For example, black plume colour is referred to as the formation of soot, which is originally black [15]. About 20-25% of the black smoke mass is soot [16]. White plume colour is referred to the availability of water vapour and inorganic salts which is mostly sodium chloride [17-19]. In contrast to black smoke, white smoke shows almost no carbon in forms of CO, CH_4, NMHC, and black carbon [20]. Figure 1 illustrates smoke emissions from a traditional crude-oil refining chamber used in Nineveh Province.

There is no analysis for the white and black smokes for HC, suspended particulate matters or other substances from the sampling sites; and therefore, most of the

comparisons were based on values from the literature, which leads to under/overestimation of the smoke effects. Table 1 shows the different components of white and black smokes. Both types of smokes can cause chronic pulmonary diseases and change the levels of blood variables. Harte et al. [21] demonstrated the possibility of having chronic bronchitis and emphysema after a long exposure duration to respiratory pollutants. Another study found that black smoke, NO_2, CO, and SO_2 in polluted air could cause heart attack and other circulatory diseases [22].

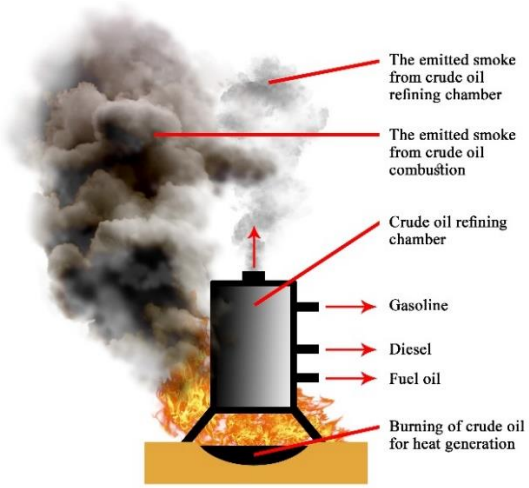

Fig. 1. Smoke emissions from a conventional crude oil refining chamber.

Table 1. Properties and components concentrations of white and black smoke emissions during direct combustion of crude oil*.

No.	Component or property	White smoke	Black smoke	Reference
1	Soot	0.5-0.8 g/kg, less than 40 g/kg**	Light and fluffy, 1.8 - 13.7 g/kg, 150-200 g/kg**, 200-250 g/kg**, 200-300 g/kg**	[15, 16, 20, 23, 24]
2	Formation of chain-aggregate soot	Fewer	Many	[16-18, 25]
3	Non-spherical particles	Fewer, small amounts	Many	[16-18]
4	Particles diameters	0.1 0.3 µm	Around 0.8 µm	[20]
5	TOC	0.0	1.5 - 10.8 g/kg	[24]
6	Organics	-	300 g/kg**	[26]
7	Water vapour	Many	Fewer	[18, 19]
8	Inorganic salts concentrations	800 g/kg** (mostly NaCl)	300 g/kg**, no more than 200 g/kg**	[16, 18, 20, 26-28]
9	Chloride	3220 µg/m^3,	15 - 182 µg/m^3	[18, 20]
10	Calcium	415 µg/m^3	6 - 84 µg/m^3	[18]
11	Sulfate	630 µg/m^3, 150 g/kg**	9 - 177 µg/m^3, 80 g/kg**	[18, 26, 27]
12	CO	3.2-3.8 g/kg,	0.9-12.9 g/kg,	[18, 24]

		0.25-0.58 ppmv	0.58-3.35 ppmv	
13	CO_2	837-843 g/kg, 377-442 ppmv	776-845 g/kg, 375-446 ppmv	[18, 24]
14	NO_x	0.65-0.71 g/kg, 0.32 ppmv	0.34-0.59 g/kg, 0.32 ppmv	[18, 24]
15	CH_4	0.3-1.1 g/kg, 1.78-1.88 ppmv	0.6-1.6 g/kg, 1.79-1.88 ppmv	[18, 20, 24]
16	Non-methane hydrocarbons NMHC	2.8-8.0 g/kg, 0.89-1.69 ppmv	3.1-51 g/kg, 0.57-2.93 ppmv	[18, 24]

[*] Most of the smokes data (the values of the components) belong to the previous studies of Kuwait oil fires in year 1991. These data are assumed similar to the smokes data of Nineveh-province oil fires because the source of oil belongs to the same geographical zone (Kuwait and Basra oil fields).
[**] The units were converted from (% by mass) to (g/kg) to be comparable with other numbers that have the same unit formula (g/kg) for the same component).

2. Materials and Methods

2.1. Blood sampling

The combustion areas of Badush district was selected in this study, which located 15 km from Mosul in the northwest. Blood samples were directly collected from workers inside the areas of crude-oil combustion during two consecutive days. According to smokes colour, there were two types of combustion areas: areas with white smokes emissions and others with black smokes emissions. For every kind of combustion areas, ten blood samples were randomly collected from the workers for blood variables tests. Further, another ten blood samples were collected from unexposed people to the smokes (far away from the burning areas), and their samples were set as the control-samples for the comparison with the workers-samples. For this comparison, the randomly selected workers were men, between 18 to 40 years old, non-smokers, free of any chronic diseases, and worked inside the combustion areas for 6 months.

To investigate the effects of exposure duration on the selected blood variables, the workers were divided into two groups: the first group included workers under 6 months of exposure duration, while the second group included workers under 12 months of exposure duration. The obtained results from the first group were compared to the results of the second group, and both groups were compared with control-samples results. The randomly selected workers were men, between 18 to 40 years old, non-smokers, free of any chronic diseases, and worked inside the areas of white smoke emissions. Similar procedures were applied for all workers and control people during blood sampling. A blood sample of 8-10 ml was collected from the hand vein of each person. From this sample, 2 ml were located inside a storing anticoagulant tube (EDTA tube) for the blood test while 6 ml of blood were located inside the regular glass tube to obtain blood serum for biochemical tests [29].

2.2. Blood variables testing

The haemoglobin test (Hb), Packed Cell Volume (PCV), platelets count, and Red Blood Cells (RBCs) count were analysed using Sysmex coulter counter at the blood bank of Ibn Sena hospital - Mosul. The total protein concentrations in serum were estimated using Biuret protein assay and BIOLABO analytical set [30]. The concentration of total serum cholesterol was determined through enzymatic activities using BIOLABO analytical set [31]. ALT and AST were determined using the end-point colourimetric method by Randox analytical set [32]. Due to the difficulties of measuring the effect of oil spills on

blood variables side by side with smoke effect, it is considered that all workers were exposed to oil spills at the same levels. Oil spills are estimated between 1-3 barrels (220-660 L) for the burning area of 1000 m^3 per day. The range of oil spills was evaluated according to working hours (no less than 8 hrs per day), at least ten in-site workers and the produced petroleum derivatives, which are near from 30000 L per day.

2.3. Statistical analysis

The data were analysed by one-way analysis of variance (ANOVA) using Student-Newman-Keuls (SNK) and Duncan's multiple range test methods with a probability value of p-value ≤ 0.05. Statistical analysis was carried out using IBM® SPSS Statistics Version 25.0.

3. Results and Discussions

3.1. Effect of smoke type on biochemical blood variables

The impacts of white and black smokes on biochemical blood variables are presented in Table 2. In general, it is clear that the exposure to emitted smokes has negative effects on health through changing blood variables significantly (p <0.05) from the average control level.

Table 2. Effect of smoke type (white, black) on biochemical blood variables.

Blood variable	Unit	Control[A]	WS-6[B]	BS-6[C]	R^2	F-value[*]
Hb	g/dL	14.20±0.16^a	12.69±0.11^b	12.15±0.04^c	0.971	450.219
PCV	%	42.55±0.51^a	40.04±0.59^b	38.83±0.26^c	0.858	81.897
Platelets	× 10^9/L	216.40±4.81^b	248.29±12.26^a	223.75±9.36^b	0.548	16.392
RBCs	× 10^6/mcL	5.29±0.03^b	5.60±0.05^a	5.42±0.25^{ab}	0.295	5.649
Total protein	g/dL	7.77±0.14^a	5.57±0.53^b	5.85±0.07^b	0.844	73.028
Uric acid	mg/dL	5.13±0.49^a	1.86±0.14^c	3.57±0.36^b	0.888	106.814
AST	U/L	11.56±1.24^b	16.36±1.02^a	18.40±2.45^a	0.619	21.974
ALT	U/L	14.74±1.75^c	17.98±1.91^b	20.69±2.18^a	0.468	11.880

Means for the determined values in the same column followed by a different superscript letter (a, b, c) are significantly different from each other ($p \leqslant 0.05$). The means of blood variables were obtained from *A* = unexposed people to pollution (control), *B* = workers exposed to white smoke for 6 months, *C* = workers exposed to black smoke for 6 months. [*] ANOVA shows all biochemical blood variables are significant at ($\alpha < 0.01$).

As shown in Fig. 2, the highest increment ratio to control is 59.2% and 41.5% for AST-BS-6 and AST-WS-6, followed by 40.4% and 22% for ALT-BS-6 and ALT-WS-6, respectively. According to Duncan's multiple range test, the difference between AST-WS-6 of 16.35 U/L and AST-BS-6 of 18.40 U/L is not significant; means that white and black smokes have the same efficiency on AST enzyme. Further, both AST-WS-6 and AST-BS-6 values are significantly (p <0.05) higher than the control (11.56 U/L) with a coefficient of determination (R^2) of 0.619 and F-value of 21.974. The ALT-BS-6 value of 20.69 U/L is significantly (p <0.05) higher than ALT-WS-6 value of 17.98 U/L, it can be deduced that ALT enzyme is more sensitive to black smoke than the white smoke. Same as AST, both ALT-BS-6 and ALT-WS-

6 values are significantly ($p < 0.05$) higher than the control with an R^2 value of 0.468 and F-value of 11.88.

In addition to ALT and AST, white smoke has significantly increased the values of RBCs and platelets above to control values, contrary to black smoke that did not show any significant change, as shown in Table 2. The increment ratio to control was 14.7% for platelets-WS-6 and 5.9% for RBCs-WS-6, compare to 3.4% for platelets-BS-6 and 2.5% for RBCs-BS-6. The exposure to oil-refining smokes has decreased the values of Hb, PCV, total protein and uric acid below the reference values (control samples), as shown in Fig. 3. The decrements in these variables can cause several health problems. The highest decrease in ratio to control-samples was 36.7% for uric acid-WS-6 followed by 30.4% for uric acid-BS-6. The uric acid-WS-6 value of 1.86 mg/dL is significantly different from 3.57 mg/dL for uric acid-BS-6 and 5.13 for control. The analytical method for uric acid shows a high R^2 value of 0.888 and F-value of 106.814.

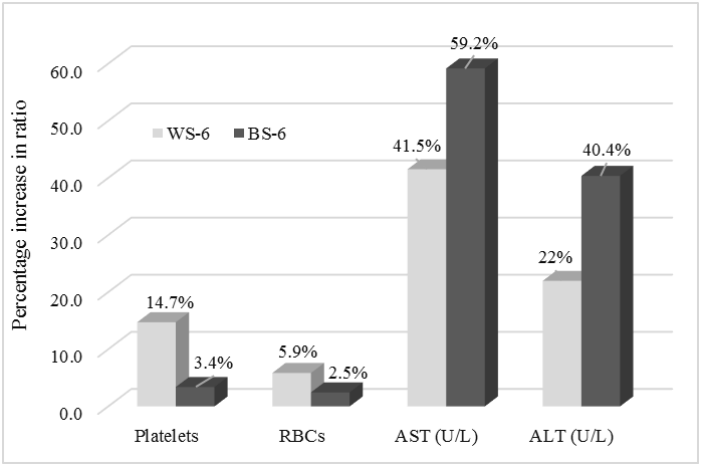

Fig. 2. Percentage increase in ratio of workers to control blood-variables values for white and black smoke exposure.

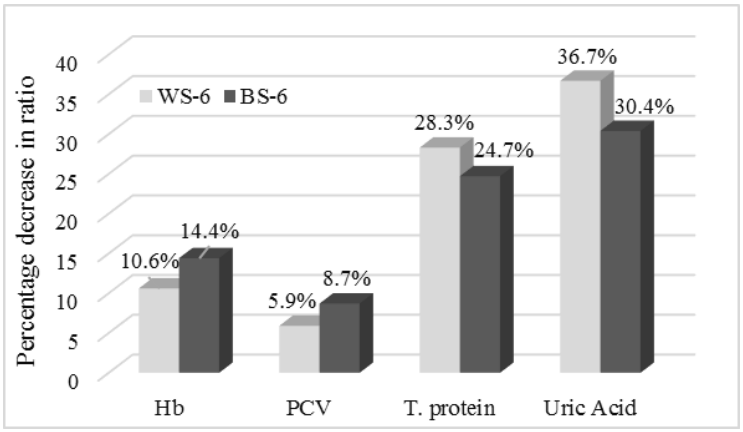

Fig. 3. Percentage decrease in ratio of workers to control blood-variables values for white and black smoke exposure.

Regarding total protein, Duncan's multiple range test shows that the decrement of 28.3% for total protein-WS-6 is not significantly different from 24.7% for total protein-BS-6, but both values are significantly lower than the control. The various components of both smoke types seem to have a similar effect on total protein through decrease its values to the same levels. The analytical method for total protein shows a high R^2 value of 0.844 and F-value of 73.028. The values of Hb-BS-6 and PCV-BS-6 are 14.4% and 8.7% lower than the control, respectively. The black smoke was significantly ($p < 0.05$) decreased the values of Hb and PCV comparing to white smoke. In addition, white smoke has significantly reduced the amounts of Hb and PCV by 10.6% and 5.9%, respectively, from the control values. The analytical method for Hb shows the highest R^2 value of 0.971 and F-value of 450.219.

Researchers stated that the increase of ALT and AST levels in blood serum could occur due to the hepatic failure of exposed workers to oil spills. As a result, ALT and AST enzymes release to the bloodstream in high quantities [7]. Moreover, the ALT enzyme is made in the liver. Consequently, any damage in the liver's cells will spill this enzyme into the circulatory system; hence, ALT levels are used as an indicator for liver's health [33-35].

The black smoke emission has decreased the value of Hb below the reference value. This reduction could occur due to high levels of CO in black smoke that could reach up to 12.9 g per 1 kg of the smoke weight [24]. During inhalation, CO binds with Hb and forms carboxyhemoglobin (COHb), which leads to the loss the Hb in the blood [10]. Moreover, Hb can react with benzene and PAHs inside the human body to produce Hb adduct. Hence, the levels of Hb reduce in blood [5, 8]. In agreement with the previous study by Das and Chatterjee [36], the PCV values of workers were lower than the average levels. The researchers found that the exposure to polluted air with the suspended particulate matter, respirable particulate matter, SO_2 and NO_x could decrease the level of PCV. Davidson and Penney [37] illustrated that blood volume could be increased during the exposure to high amounts of CO; hence, PCV value is decrease compared to the total blood volume.

The RBCs values for in-site workers were higher than the reference level (control). According to Davidson and Penney [37], due to inhalation of CO at high levels, the RBCs values increased above the reference level. This increment occurs due to the high levels of CO in the emitted smoke, as shown in Table 1. In contrast to the RBCs, results show that total protein values are lower than the control. This decrement is in agreement with the previous study by Majd et al. [38] that indicate the high amount of CO, SO_2, NO_2, and HC could be the reason of low total protein levels in the blood. The inhalation of smokes and the direct contact with oil spills in the burning areas have lower the level of uric acid below the average value, as shown in Table 2. This reduction occurs when heavy metals stored inside the liver and damage its functionality. Subsequently, the liver produces small quantities of amino acids and enzymes responsible for proteins formation. Thus, uric acid produces from proteins metabolism in low levels [39].

3.2. Effect of smoke type on blood lipids concentrations

The exposure to white and black smokes has increased all blood lipids concentrations above the reference levels (control), as shown in Table 3. The increment ratios of blood lipids are different from each other. As shown in Fig. 4, the highest increment ratio is 224% for VLDL-WS-6 followed by 179.5% for triglycerides-WS-6.

Table 3. Effect of smoke type (white, black) on blood lipids concentrations (mmol/L).

Blood lipid*	Control[A]	WS-6[B]	BS-6[C]	R^2	F-value*
Cholesterol	4.49±0.28[c]	6.41±0.15[a]	5.78±0.17[b]	0.892	111.59
Triglycerides	1.27±0.10[c]	3.55±0.32[a]	2.52±0.29[b]	0.883	101.684
VLDL	0.25±0.04[c]	0.81±0.14[a]	0.63±0.06[b]	0.782	48.360
LDL	2.92±0.14[c]	4.33±0.25[a]	3.16±0.17[b]	0.855	79.774
HDL	1.13±0.06[b]	2.14±0.12[a]	1.24±0.13[b]	0.905	128.543

Means for the determined values in the same column followed by a different superscript letter (a, b, c) are significantly different from each other ($p \leq 0.05$). The means of blood lipids variables were obtained from *A* = unexposed people to pollution (control), *B* = workers exposed to white smoke for 6 months, *C* = workers exposed to black smoke for 6 months. * ANOVA shows all blood lipids variables are significant at ($\alpha < 0.01$).

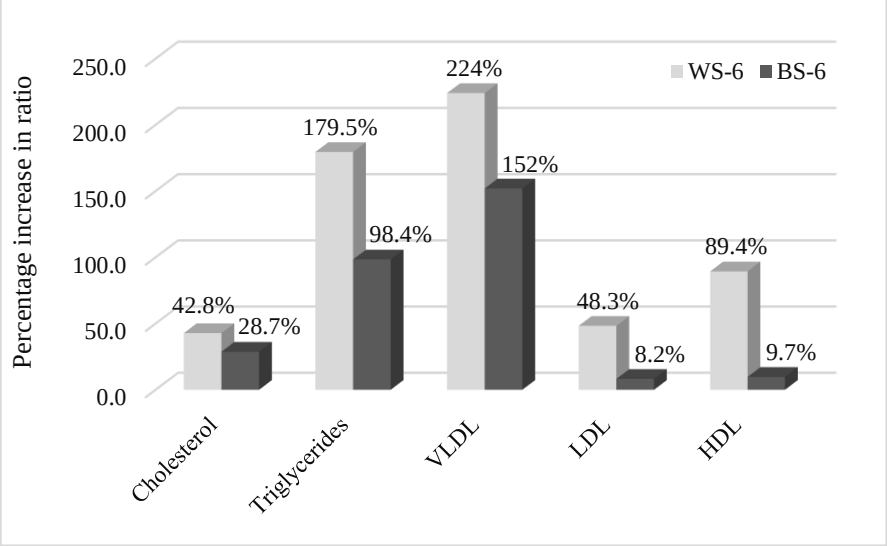

Fig. 4. Percentage increase in ratio of workers to control blood-lipids concentrations for white and black smoke exposure.

The VLDL-WS-6 value of 0.81 mmol/L is significantly ($p<0.05$) higher than VLDL-BS-6 of 0.63 mmol/L and the control value of 0.25 mmol/L. Similar to VLDL, the LDL-WS-6 of 4.33 mmol/L is significantly ($p<0.05$) higher than LDL-BS-6 of 3.16 mmol/L and control value of 2.92 mmol/L. Hence, it leads to the fact that VLDL and LDL are more sensitive to black smoke than the white. As for HDL, there is no significant difference between the effect of white and black smokes on HDL levels, but both smokes are significantly increased HDL values compared to control. The analytical method for HDL shows a high R^2 value of 0.905 and F-value of 128.543.

The white smoke emissions were found to be more effective than the black smoke for all lipids. The white smoke has increased the values of cholesterol and triglycerides by 42.8% and 179.5%, respectively, from control values. On the other hand, the increment of black smoke emission was 28.7% for cholesterol and 98.4%

for triglycerides. The effectiveness of black smoke on blood lipids could be related to the high content of organic and inorganic carbons.

3.3. Effect of exposure duration on biochemical blood variables

The effects of 6 and 12 months of exposure duration on biochemical blood variables are presented in Table 4. In general, the negative impacts on human health increase with the increase of exposure duration to the emitted smokes. The levels of blood variables of 12 months exposure are higher to the undesired levels than the 6 months.

Table 4. Effect of exposure duration (6 or 12 months) on biochemical blood variables.

Blood variables	Unit	Control[A]	WS-6[B]	WS-12[C]	R^2	F-value[*]
Hb	g/dL	14.20 ± 0.16^{a}	12.69 ± 0.11^{c}	13.02 ± 0.09^{b}	0.941	215.854
PCV	%	42.55 ± 0.51^{a}	40.04 ± 0.59^{b}	42.19 ± 0.77^{a}	0.636	23.597
Platelets	$\times 10^9$/L	216.40 ± 4.81^{c}	248.29 ± 12.26^{b}	343.78 ± 19.62^{a}	0.899	120.709
RBCs	$\times 10^6$/mcL	5.29 ± 0.03^{c}	5.60 ± 0.05^{b}	5.81 ± 0.05^{a}	0.930	179.259
Total protein	g/dL	7.77 ± 0.14^{a}	5.57 ± 0.53^{b}	5.79 ± 0.13^{b}	0.842	71.882
Uric acid	mg/dL	5.13 ± 0.49^{a}	1.86 ± 0.14^{c}	4.11 ± 0.50^{b}	0.862	84.375
AST	U/L	11.56 ± 1.24^{c}	16.36 ± 1.02^{b}	17.50 ± 1.79^{b}	0.660	26.227
ALT	U/L	14.74 ± 1.75^{c}	17.98 ± 1.91^{b}	19.18 ± 1.52^{b}	0.398	8.936

Means for the determined values in the same column followed by a different superscript letter (a, b, c) are significantly different from each other ($p \leqslant 0.05$). The means of blood variables were obtained from *A* = unexposed people to pollution (control), *B* = workers exposed to white smoke for 6 months, *C* = workers exposed to white smoke for 12 months. [*]ANOVA shows all biochemical blood variables are significant at ($\alpha < 0.01$).

The increment of platelets, RBCs, AST and ALT values continued from 6 months to 12 months exposure duration. The highest increase in the ratio of workers to control blood-variables is for platelets, which reached 58.9% on 12 months compared to 14.7% on 6 months exposure, as shown in Fig. 5. ANOVA shows a statistically significant difference between platelets-WS-12 of 343.78×10^9/L, platelets-WS-6 of 248.29×10^9/L and control of 216.4×10^9/L. The analytical method for platelets shows a high R^2 value of 0.899 and F-value of 120.709. Similar to platelets, RBCs shows a statistically significant difference between RBCs-WS-12 of 5.81×10^6/mcL, RBCs-WS-6 of 5.6×10^6/mcL and control of 5.29×10^6/mcL. The increment ratio of the obtained RBCs values to control was 9.8% for RBCs-WS-12 and 5.9% for RBCs-WS-6.

There was no significant difference between 6 and 12 months of exposure duration for workers-variables of AST and ALT. The increase in the ratio of AST-WS-6 to control is 41.5% while it is 51.4% for AST-WS-12. Further, the increment to control was 22% for ALT-WS-6, which is statically closed to 30.1% for ALT-WS-12. On the other hand, the decrement of Hb, PCV, total protein and uric acid of 6 months exposure were more than the 12 months. The workers-variables of Hb, PCV and uric acid on the 6 months exposure are significantly lower than the 12 months, while the values of total protein are not significantly changed between the two exposure durations.

There is no significant change in ALT and AST values between the exposure to 6 and 12 months, but both variables are significantly higher than the control.

Researchers assumed that the increase of ALT and AST levels in blood serum could occur due to hepatic failure that releases these enzymes to bloodstream [7]. Moreover, the ALT enzyme is made in the liver. Consequently, any damage in the liver's cells will spill this enzyme into the circulatory system; hence, ALT levels are used as an indicator for liver's health [33-35]. The RBCs is proportional to the exposure duration, is in agreement with the previous studies. Researchers found that the long exposure duration to smoke could negatively affect the lungs functioning by increasing the value of RBCs due to the lack of oxygen in the lungs [33, 40-42].

Interestingly, the values of Hb, PCV and uric acid increased at 12 months toward the reference values after the decrement in values at 6 months of the exposure duration, as shown in Fig. 6. Contrary to the findings of Honda et al. [43], which concluded that Hb is sensitive to the exposure duration. The researchers found that Hb values were decreased with the increase of exposure to polluted air with particulate matter and NO_2, and the values continued to decline with the rise of exposure duration up to 5 years. Because of the relation between Hb, PCV and RBCs, the increase in Hb and PCV values toward the control values could be related to the continuous increment in RBCs levels that reached up to 5.81×10^6/mcL at 12 months of the exposure duration, as shown in Table 4.

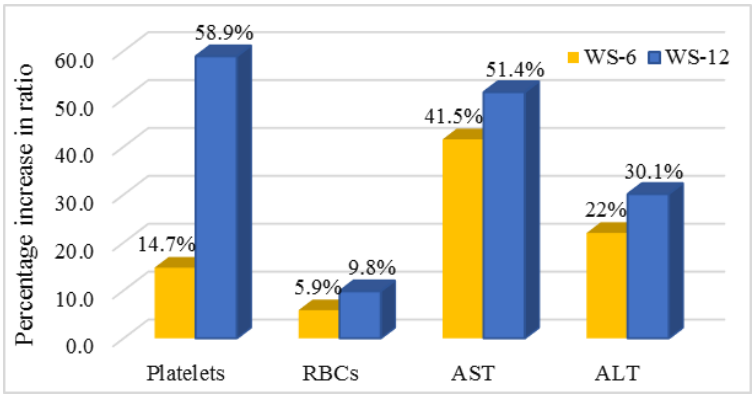

Fig. 5. Percentage increase in ratio of workers to control blood-variables values for 6 and 12 months exposure durations.

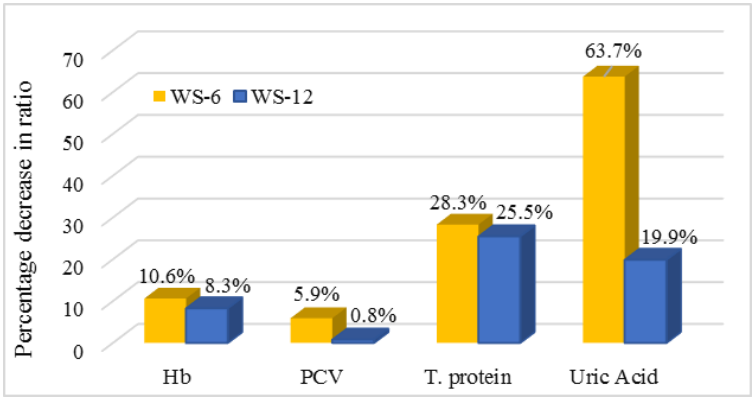

Fig. 6. Percentage decrease in ratio of workers to control blood-variables values for 6 and 12 months exposure durations.

3.4. Effect of exposure duration on blood lipids concentrations

All blood lipids were found to be sensitive to the exposure duration. The values of 12 months of exposure were significantly ($p < 0.05$) higher than the exposure duration of 6 months and the control, as shown in Table 5.

Table 5. Effect of exposure duration (6 or 12 months) on blood lipids concentrations (mmol/L).

Blood lipid*	Control[A]	WS-6[B]	WS-12[C]	R^2	F-value*
Cholesterol	4.49±0.28^{c}	6.41±0.15^{b}	6.91±0.24^{a}	0.922	159.546
Triglycerides	1.27±0.10^{c}	3.55±0.32^{b}	4.69±0.39^{a}	0.928	173.890
VLDL	0.25±0.04^{c}	0.81±0.14^{b}	1.10±0.11^{a}	0.857	80.940
LDL	2.92±0.14^{c}	4.33±0.25^{b}	5.12±0.19^{a}	0.922	160.028
HDL	1.13±0.06^{c}	2.14±0.12^{b}	2.40±0.22^{a}	0.882	101.308

Means for the determined values in the same column followed by a different superscript letter (a, b, c) are significantly different from each other ($p \leqslant 0.05$). The means of blood lipids variables were obtained from *A* = unexposed people to pollution (control), *B* = workers exposed to white smoke for 6 months, *C* = workers exposed to white smoke for 12 months. *ANOVA shows all blood lipids variables are significant at ($\alpha < 0.01$).

The highest increase in ratio to control is 340% for VLDL-WS-12, as shown in Fig. 7. Longer exposure time means more exposure to pollutant; hence, more negative effects on workers' health. The levels of cholesterol in blood were higher by 42.76% for cholesterol-WS-6 and 53.9% for cholesterol-WS-12 comparing to control. Researchers found that heavy metals such as Cd, Pb, and Hg could increase cholesterol levels in blood serum of mice and interact with lipids structures, lipids metabolism, and responsible enzymes for lipids metabolisms [44]. Previous studies stated that the exposure to heavy metals is effective on blood lipids values and it can change the blood lipids levels according to exposure kind and duration as well as the original lipids levels [45].

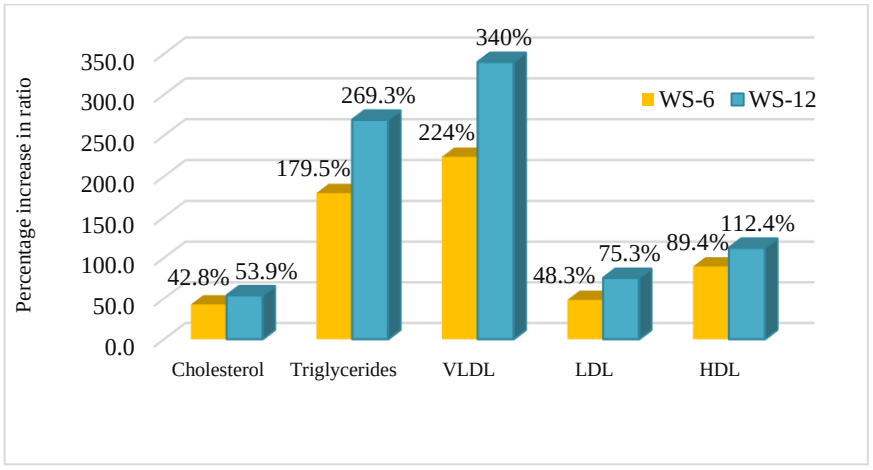

Fig. 7. Percentage increase in ratio of workers to control blood-lipids concentration for 6 and 12 months exposure durations.

4. Conclusions

Oil-refining smoke has different detrimental effects on human health. The impacts of white and black smokes on blood variables and lipids are different due to the difference in smoke composition. Generally, the emitted smokes change blood variables and lipids through increment or decrement the values from the normal average levels. The exposure to black smoke has increased the workers-values of AST and ALT and decreased Hb and PCV more than the white smoke. These variables are sensitive to carbon especially in the form of CO; thereby, the high CO levels in black smoke could be the main reason for the significant increment or decrement for the mentioned variables. On the other hand, the exposure to white smoke has increased the values of platelets and RBCs and decreased total protein and uric acid more than the black smoke. These variables are more sensitive to white smoke than the black due to the high levels of sulphate and NO_x contents in white smoke.

The exposure duration of 6 months has increased all blood lipids for more than the 6 months. Contrary to Hb, PCV, total protein and uric acid values which were more sensitive in the 6 months than the 12 months duration. This could be related to the continuous rise in RBCs levels that could reach up to 5.81×10^6/mcL for 12 months of the exposure duration. The effect of oil-spills has also increased or decreased the values of blood variables and lipids side by side with smoke. Under these circumstances, workers should take into consideration all the necessary procedures starting from the design of the emission chamber and ending with preventive health services. More studies on the effects of white and black smokes on blood variables side by side with oil spills should be achieved.

Abbreviations

ALT	Alanine Transaminase
ALT-BS-6	Effect of Black Smoke on Alanine Transaminase for 6 Months
ALT-WS-6	Effect of White Smoke on Alanine Transaminase for 6 Months
ALT-WS-12	Effect of White Smoke on Alanine Transaminase for 12 Months
ANOVA	Analysis of Variance
AST	Aspartate Transaminase
AST-BS-6	Effect of Black Smoke on Aspartate Transaminase for 6 Months
AST-WS-6	Effect of White Smoke on Aspartate Transaminase for 6 Months
AST-WS-12	Effect of White Smoke on Aspartate Transaminase for 12 Months
BS-6	Effect of Black Smoke for 6 Months
Cholesterol-WS-6	Effect of White Smoke on Cholesterol for 6 Months
Cholesterol-WS-12	Effect of White Smoke on Cholesterol for 12 Months
Hb	Haemoglobin
HC	Hydrocarbons
HDL	High-Density Lipoproteins

LDL	Low-Density Lipoprotein
LDL-BS-6	Effect of Black Smoke on Low-Density Lipoprotein for 6 Months
LDL-WS-6	Effect of White Smoke on Low-Density Lipoprotein for 6 Months
NMHC	Non-Methane Hydrocarbons
PCV	Packed Cell Volume
PCV-BS-6	Effect of Black Smoke on Packed Cell Volume for 6 Months
Platelets-BS-6	Effect of Black Smoke on Platelets for 6 Months
Platelets-WS-6	Effect of White Smoke on Platelets for 6 Months
Platelets-WS-12	Effect of White Smoke on Platelets for 12 Months
RBCs	Red Blood Cells
RBCs-BS-6	Effect of Black Smoke on Red Blood Cells for 6 Months
RBCs-WS-6	Effect of White Smoke on Red Blood Cells for 6 Months
RBCs-WS-12	Effect of White Smoke on Red Blood Cells for 12 Months
SNK	Student-Newman-Keuls
TOC	Total Organic Carbon
Total Protein-BS-6	Effect of Black Smoke on Total Protein for 6 Months
Total Protein-WS-6	Effect of White Smoke on Total Protein for 6 Months
Triglycerides-WS-6	Effect of White Smoke on Triglycerides for 6 Months
Uric Acid-BS-6	Effect of Black Smoke on Uric Acid for 6 Months
Uric Acid-WS-6	Effect of White Smoke on Uric Acid for 6 Months
VLDL	Very Low-Density Lipoprotein
VLDL-BS-6	Effect of Black Smoke on Very Low-Density Lipoprotein for 6 Months
VLDL-WS-6	Effect of White Smoke on Very Low-Density Lipoprotein for 6 Months
WS-12	Effect of White Smoke for 6 Months

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