



## **Inflammatory Markers in Workers Occupationally Exposed to Petrol and Petroleum Products**

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### **Authors' contributions**

*This work was carried out in collaboration among all authors. Author BES designed the study, performed the statistical analysis, author NEO wrote the protocol, while author AON wrote the first draft of the manuscript and managed the analyses and the literature searches. All authors read and approved the final manuscript.*

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### **ABSTRACT**

**Aim:** The aim of this study was to assess inflammatory markers in workers occupationally exposed to petrol and petroleum products

**Study Design:** This study is a cross-sectional study.

**Place and Duration of Study:** Sample: Abia State University Teaching Hospital, Aba, Abia State and Laboratory Department, JAROS Inspection Services Limited, Port Harcourt, Rivers State, between April 2018 and June 2018.

**Methodology:** A total of 204 samples comprising of 123 auto-mechanics and 81 non -auto-mechanics were assayed. Detailed information of the bio-data of the subjects including age, gender, medical history, health information and lifestyle were obtained from each participant. Blood samples were collected from for the analysis of inflammation markers, IL-6, TNF- $\alpha$  and CRP were determined using standard methods and techniques. The effect of age and duration of exposure on the inflammation parameters were considered. Statistical Analysis System (SAS), STAT 15.1, developed by SAS Institute, North Carolina State University, USA was used for statistical analysis. Data were presented as Mean  $\pm$  SEM, comparison of means of groups that are more than two was done using Analysis of Variance (ANOVA), and the Tukey test of multiple comparison was used to test for variance within and across groups.

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**Results:** There was significant increase in the means of IL-6, TNF- $\alpha$  and CRP in the exposed subjects ( $p < 0.05$ ) compared with the control subjects. There was no significant difference ( $p > 0.05$ ) in the means of IL-6, TNF- $\alpha$  and C-reactive protein (CRP) between the age groups of the exposed and the control subjects. Similarly, there was no significant difference between the groups, based on duration of exposure. This suggests that the toxic effect does not depend on the age or duration of exposure but on other factors for the automechanics in Aba.

**Conclusion:** This study shows that the exposure of automechanics may significantly increase the serum IL-6 TNF- $\alpha$  and Hs-CRP levels. Increase in the serum levels of the inflammation markers is predictive of the danger of future pathology in automechanics compared with non automechanics in Aba metropolis. Age and duration did not influence significant variation in the automechanics.

*Keywords: Automechanics; petroleum products; interleukins; c-reactive proteins; exposure.*

## 1. INTRODUCTION

Petrol and petroleum products are the major sources of pollution in the mechanic workshop. Pollution is due to the presence of toxic chemicals such as mono and polyaromatic hydrocarbons, benzene, toluene, flourene, benzo (a) pyrene and metals such as lead, mercury [1]. The presence of these chemicals are known to cause cellular damage by the production of free radicals referred to as reactive oxygen species (ROS) and inflammatory processes [2][3]. Association of inflammation with onset of biological disorders is a long standing phenomenon, [2][4][5]. Reports show that chronic inflammation is a root cause of chronic inflammatory diseases such as respiratory diseases, cardiovascular diseases (CVDs), diabetes mellitus, hepatic, renal and neurological disorders [2][3][6]. Chronic inflammation was found to increase atherosclerosis and cardiovascular disease morbidity and mortality compared to the control individuals [7].

Rheumatoid arthritis (RA) has been described as an inflammatory disease and a risk factor to CVDs with interactions that affect other systemic disorders. A population study showed that RA can reduce life expectancy between (5-10) years [7][8]. Chronic inflammation has also been described as an integral factor in aging process [9]. Approximately 3 out of 5 global deaths is caused by chronic inflammatory diseases. Physical activity and good nutrition status maintains good health by reduction of inflammatory mediators [9][10][3]. Harmful effects of exposure have been described both at the local and at the systemic levels [11][12]. Ingestion of petrol is associated with discomfort from the mouth to the stomach and systemic disorders may follow via biotransformation of petrol components [13]. Occupational studies on petrol attendant showed positive correlation with

oxidative stress and inflammation [9][14][9]. Auto-mechanics are professionals who maintain and repair automobiles. They are generously exposed to petrol and petroleum products via inhalation of fumes or particulate matter, ingestion and contact with skin. These automechanics, clean components of the engine, of the car with petrol, without protective device. Siphoning of petrol with their mouth is another observed common practice in this occupation. This practice exposes the automechanic to accidental ingestion of petrol. However, there is pausing of literature on the impact of these occupational practices on the levels of inflammatory markers in automechanics in Nigeria, thus, the essence of the present study.

## 2. MATERIALS AND METHODS

### 2.1 Area of Study

The area of study is Aba and its metropolis, located in the South Eastern zone of Nigeria. It lies on the geographical coordinates of 5<sup>0</sup>07N, 7<sup>0</sup>22<sup>0</sup>E. The city is known for heavy traffic flow and poorly unmaintained road network. Automobiles are imported as 'fairly used' vehicles. Thus with the poorly maintained roads, many vehicles are frequently damaged and the automechanics are often engaged in automobile repairs.

### 2.2 Study Design

This cross-sectional study was conducted between June and August, 2018. Detailed information of the bio-data of the subjects including age, gender, medical history, health information, duration of occupational practice and lifestyle were obtained from each participant using a questionnaire.

### 2.3 Inclusion and Exclusion Criteria

Blood samples were collected from automechanics who were resident in Aba Metropolis and had worked as automechanics in Aba for not less than two years and the control group or non-automechanics. These were apparently healthy subjects, with no known case of acute or chronic diseases such as liver disease, kidney disorder, cardiovascular diseases (CVDs), hypertension, diabetes mellitus, diarrhea, arthritis, HIV, blood related diseases such as anaemia, leukaemia, or under any medication within the period of this study. Also included were those who were not associated with alcoholism and smokers and those who willingly gave their consent to participate. However individuals associated with alcoholism, smokers and those with known illness, or undergoing any form of medication within the period of study and those who were not willing to give their consent were excluded.

### 2.4 Collection of Samples

Fasting blood samples, were collected into containers and carefully labeled to avoid loss of identity of samples during, sample handling. Five (5) mls of blood was manually collected using the method of Arkin et al. [15]. Each sample was centrifuged at 3000 revolutions per minute for 5 minutes. With sterile disposable rubber pipettes, serum samples were separated into sterile sample bottles and were stored in the refrigerator at 2-4°C until ready use.

### 2.5 Determination of Inflammatory Markers

The determination of Interleukin-6 (IL-6) was determined based on enzyme linked immunosorbent assay (ELISA) technique according to the method of Felding et al. [16], using, Melsin laboratory kit for, human IL-6. Reference range for IL-6 is 0-48pg/ml. The determination of tissue necrosis factor alpha (TNF- $\alpha$ ) was by ELISA technique according to the method of Herreweghe et al. [17] using, Melsin kit for, research on human TNF- $\alpha$  only from Melsin medical company limited, Changchun in China. Reference range of the diagnostic kit for TNF- $\alpha$  is 0-80pg/ml, while HS-CRP was by turbidometric assay according to the method of Freidman and Young [18], using high sensitivity (hs-CRP) kit from Biosystems

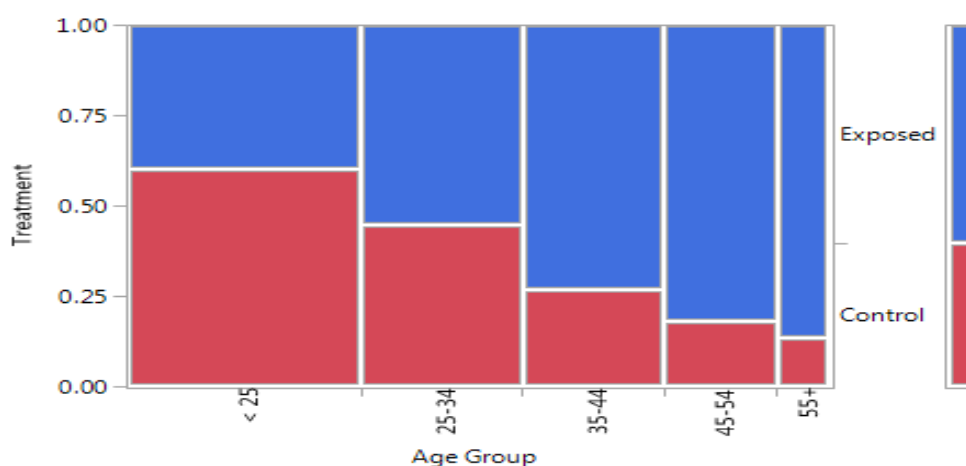
laboratories, Barcelona (Spain). Reference range for Hs-CRP for males: 2.13ng/l – 7.90ng/l.

### 2.6 Statistical Analysis

The statistical software used for analysis is statistical analysis system (SAS) Model, STAT 15.1. a product of SAS Institute, North Carolina State University, USA. Data was reported as Means $\pm$ SEM for all parameters. Student t-test analysis was applied for comparison of two means and for more than one group of two means, analysis of variance (ANOVA), and the Tukey test of multiple comparison were used. The Pearsons correlation was used to determine the correlations between parameters. Variation in the means of parameters was considered statistically significant at  $p < 0.05$ .

## 3. RESULTS AND DISCUSSION

Exposures to petrol and petroleum constituents have been reported in association with cellular damage. Inflammation is a necessary process for the repair or replacement of damaged cells [19]. However it has also been established that chronic inflammation forms the bedrock for most chronic diseases [13]. Earlier studies have reported variously the effect of these exposures. Significant increase in the mean CRP and IL-6 TNF- $\alpha$  has been reported in the study of traffic-related occupationally exposed individuals [4]. In this study, there was significant increase in the mean serum levels of inflammation markers CRP, and IL-6 TNF- $\alpha$  in the exposed individuals compared with the control indicating cellular damage which is probably due to chronic exposure to petrol and petroleum products (Table 1) [4]. Similar observation was reported after short term exposure with human and animal to cause significant increase of in the exposed [20]. This was attributed to the presence of highly toxic substances such as xylene, toluene and benzene [20]. Contrary to this, a similar exposure to petrol for both short and long term exposure did not show significant variation in the serum level of inflammatory markers and the control [21]. In this study, determination of the serum concentration of these parameters showed that 21.1%, 13.0% and 10.1% of the exposed population had already, exceeded the reference health value for IL-6, TNF- $\alpha$  and CRP respectively. These show toxic effect of exposure and the risk of inflammatory diseases in the future.



**Fig. 1. Mosaic plot showing age group of study participants treatment**

These changes are in agreement with earlier studies on petrol exposure [3][4][22]. In this study, Clark et al [23], the mean serum IL-6 and TNF- $\alpha$  level of the exposed individuals were significantly higher compared with the control individuals, ( $p < 0.05$ ). These changes were attributed to exposure to petrol and the petroleum products. This is in consistent with some earlier studies Brucker et al [4] and Kamal et al [24] and inconsistent with others Clark et al, [23] and Hajat et al [20]. There was significant increase in the mean blood Hs-CRP level between the exposed and the control subjects. This indicates adverse biological changes, due to chronic exposure to petrol. The significant increase in the inflammatory biomarker (Hs-CRP) and the pro-inflammatory cytokines agrees with the study of Brucker et al. [24], Kamal et al [8] and Tsai et al. [8]. There were other studies which reported no significant difference in the mean concentration of inflammatory markers for both short and long exposures to traffic related pollutants Clark et al, [23], Hajat et al [20]. In this study the mean Hs-CRP level of the exposed

group, was significantly higher compared to the control group, ( $p < 0.05$ ) indicating exposure to petrol and petroleum products. This is consistent with the earlier study on exposure to petrol Shaikh et al [3] and inconsistent with the study of Clark et al [23]. The route of exposure, concentration of the toxic substance, the time interval between exposure and excretion in an individual are strong factors which can influence the toxic effect of Xenobiotics Prati et al [7] and Caravedo et al [25].

There was significant difference in the mean inflammatory marker Hs-CRP and pro-inflammatory markers, IL-6 and TNF- $\alpha$  ( $p < 0.05$ ). The mean IL-6 concentration of the exposed group was significantly higher compared with the control group, ( $p < 0.05$ ) (Table 2).

With age considerations there was no significant difference in mean of IL-6 between the exposed and the control groups ( $p > 0.05$ ). In the individual treatment group there was no definite trend in the mean but in the exposed the age group < 25yrs a

**Table 1. Assessment of the Inflammation markers based on cut off points in the Exposed and in the Control subjects**

Parameter	Exposed (n=123)	Control (n = 81)
*IL-6 (pg/ml) [16]		
<48	26 (21.1%)	8(0.99%)
>48	97 (78.9%)	73(90.1%)
**TNF- $\alpha$ (pg/ml) [17]		
<80	13 (10.1%)	3(3.7%)
>80	110(89.5%)	78(96.3%)
***CRP (ng/l) [18]		
< 7.9	16(13.0%)	5(6.1%)
> 7.9	107(87%)	76(93%)

Key: IL-6= Interleukin 6, TNF- $\alpha$  = tumour necrotic factor alpha and CRP = C-reactive protein, (ng/l)

reduction in IL-6 value was recorded compared recorded for the age group 55yrs+. Earlier studies show increase in CRP with increase in age [26]. In another study, reports showed that no significant variation in the level of cytokines, were reported between the age groups of the exposed individual [25].

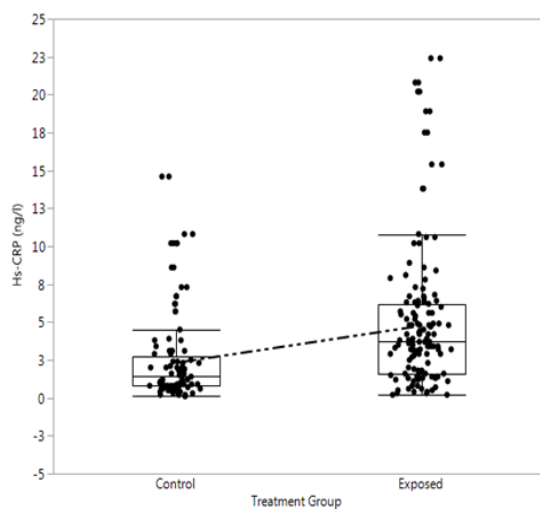
The toxic effect of exposure to chemicals have been reported to affect the response of the inflammation markers [8] With short-term exposure there was a strong positive association of IL-6 and TNF- $\alpha$  with toxic chemical exposure, which became stronger with longer period of exposure [8]. Also chronic exposure showed significant difference in the pro-inflammation markers between the first and day and the end of the week traffic-related exposure [26]. In this study there was no significant difference in the mean IL-6 and TNF- $\alpha$  between the groups of duration of exposure ( $p < 0.05$ ). Bivariate regression plot on individual serum CRP, IL-6 and TNF- $\alpha$  concentration; showed distribution of values on both sides of the fitted line in all groups based on duration of exposure. For example, the value for IL-6 at 30 years of duration of exposure, a low serum concentration, 10.6 pg/ml was recorded for an individual and also a very high value of 78.2 pg/ml was recorded for another individual. This shows that the duration of exposure did not affect the serum CRP, IL-6 and TNF- $\alpha$  concentration and there was no special trend in the value of these markers. Primarily, factors such as the concentration of the harmful substance, the immunity of the exposed

individual, nutritional status of an individual are known to significantly contribute to toxic effect of chemicals. The route of exposure, absorption, detoxification and the excretion ability have also been shown to play major roles in toxic effect of chemicals in an exposed individual. Earlier reports on similar exposure were in agreement with this work Krishnan et al [21] and Hajat et al [22]. In another study, significant variation in IL-6 concentration was reported but not in CRP concentration [27]. Initial exposure by inhalation of petrol fumes was said to have peripheral toxic effect (nasal passage and throat) and not systemic effect, which was associated with CRP concentration [26].

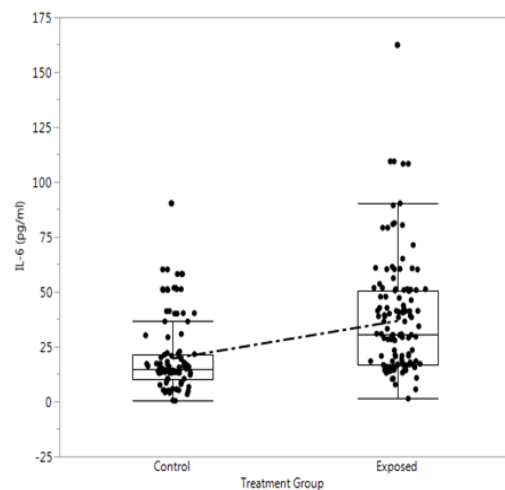
### 3.1 Age

Chronic inflammation has been described as an integral factor in aging process [26]. Although higher mean IL-6 and TNF- $\alpha$  level, was recorded for the exposed individuals in all age groups compared with the control group, there was no significant difference, (Table 3) ( $p > 0.05$ ).

This is in agreement with the study of Clark et al [23], but is contrary to the work of Singh and Newman [30]. This is probably due to physical exercise, efficient detoxification and excretion of toxic substances and strong immunity which is greatly influenced by nutrition and genetic factors [28][29]. Auto-mechanics in Aba metropolis are generally well nourished and are relatively very active [27].



**Fig. 2. Box plot of high sensitivity C reactive protein ng/l by treatment group**



**Fig. 3. Box plot of interleukins-6 (pg/ml) by treatment group**

**Table 2. Inflammation markers (Hs-CRP, IL-6 and TNF- $\alpha$ ) by treatment group and duration of exposure**

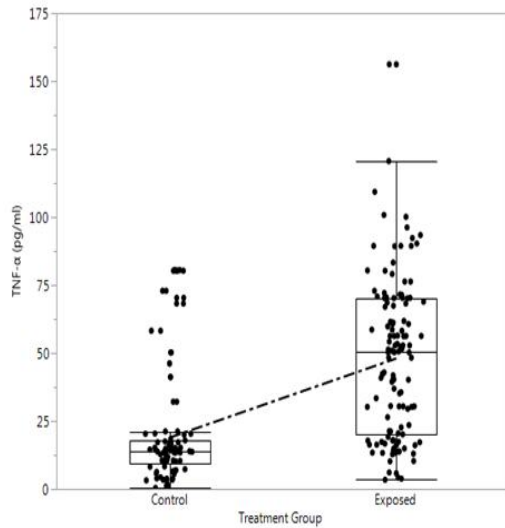
Characteristic	N	Hs-CRP (ng/l)		IL-6 (pg/ml)		TNF- $\alpha$ (pg/ml)	
		Mean $\pm$ SEM	P-value	Mean $\pm$ SEM	P-value	Mean $\pm$ SEM	P-value
Treatment Group							
Auto mechanic (Exposed)	123	4.8 $\pm$ 0.38 <sup>a</sup>		36.8 $\pm$ 2.21 <sup>a</sup>		48.0 $\pm$ 2.61 <sup>a</sup>	
Non-auto mechanic (Control)	81	2.3 $\pm$ 0.30 <sup>b</sup>	<0.0001****	19.6 $\pm$ 1.70 <sup>b</sup>	<0.0001****	18.9 $\pm$ 2.11 <sup>b</sup>	<0.0001****
Duration of Exposure (Years) <sup><math>\beta</math></sup>							
<=5							
6-10	31	4.9 $\pm$ 0.83		32.4 $\pm$ 4.21		43.5 $\pm$ 5.29	
11-20	14	4.8 $\pm$ 0.76		32.8 $\pm$ 5.87		49.5 $\pm$ 8.57	
21+	34	4.7 $\pm$ 0.80		40.4 $\pm$ 5.09		48.9 $\pm$ 5.68	
	44	4.7 $\pm$ 0.62	0.9982 <sup>ns</sup>	38.3 $\pm$ 3.27	0.5197 <sup>ns</sup>	50.1 $\pm$ 3.70	0.7955 <sup>ns</sup>

Abbreviations: SEM: Standard error of mean, Hs-CRP: High Sensivity C-Reactive Protein, IL-6: Interleukins-6, NTF- $\alpha$ : Tumor Necrotic Factor Alpha.  <sup>$\beta$</sup>  Applies only to participants in the exposure group (Auto mechanics); Within each Characteristic, means  $\pm$  SEM with different superscripts are significantly different at  $p < 0.05$ . Significance Level: \*= $p < 0.05$ ; \*\*= $p < 0.01$ ; \*\*\*\*= $p < 0.0001$ ; ns=Not Significant ( $p > 0.05$ ).

**Table 3. Inflammation markers (Hs-CRP,IL-6 and TNF- $\alpha$ ) of Treatment Group by Age Classification**

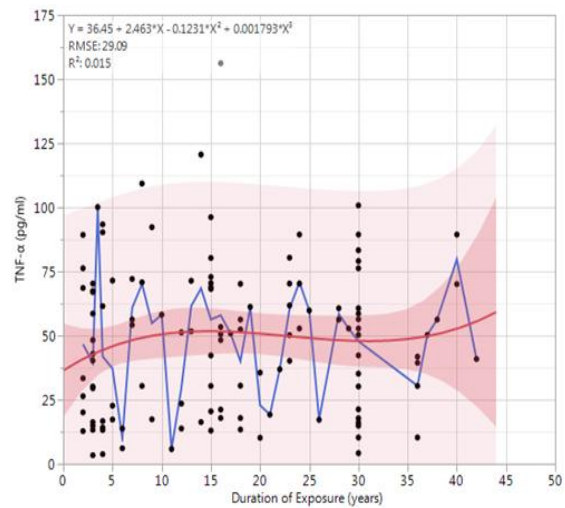
Age group (Years)	Treatment Group	N	Hs-CRP (ng/l)	IL-6 (pg/ml)	TNF- $\alpha$ (pg/ml)
			Mean $\pm$ SEM	Mean $\pm$ SEM	Mean $\pm$ SEM
< 25	Auto mechanic (Exp)	27	5.22 $\pm$ 0.73	34.82 $\pm$ 4.20	43.15 $\pm$ 4.92
	Non-auto mechanic (Ctr)	41	2.40 $\pm$ 0.59	22.68 $\pm$ 3.41	24.94 $\pm$ 3.99
25 – 34	Auto mechanic (Exp)	26	4.67 $\pm$ 0.74	36.02 $\pm$ 4.28	48.65 $\pm$ 5.01
	Non-auto mechanic (Ctr)	21	2.76 $\pm$ 0.82	15.14 $\pm$ 4.76	12.02 $\pm$ 5.58
35 – 44	Auto mechanic (Exp)	30	4.91 $\pm$ 0.69	35.76 $\pm$ 3.98	48.78 $\pm$ 4.67
	Non-auto mechanic (Ctr)	11	1.50 $\pm$ 1.14	21.39 $\pm$ 6.58	11.52 $\pm$ 7.71
45 -54	Auto mechanic (Exp)	27	4.66 $\pm$ 0.73	40.57 $\pm$ 4.20	51.37 $\pm$ 4.92
	Non-auto mechanic (Ctr)	6	1.77 $\pm$ 1.54	10.77 $\pm$ 8.91	18.33 $\pm$ 10.44
55+	Auto mechanic (Exp)	13	3.81 $\pm$ 1.05	36.60 $\pm$ 6.05	48.32 $\pm$ 7.09
	Non-auto mechanic (Ctr)	2	1.70 $\pm$ 2.67	19.30 $\pm$ 15.43	10.35 $\pm$ 18.08
Test Statistic, P-Value			0.9302 <sup>ns</sup>	0.5630 <sup>ns</sup>	0.2806 <sup>ns</sup>

Abbreviations: SEM: Standard error of mean, Hs-CRP: High Sensivity C-Reactive Protein, IL-6: Interleukins-6, NTF- $\alpha$ : Tumor Necrotic Factor Alpha. Within each parameter, means  $\pm$  SEM are not significantly different from each other. Significance level: ns=not significant ( $p > 0.05$ ).



**Fig. 4. box plot of tumor necrotic factor alpha (pg/ml) by treatment group**

In this study, there was no significant difference in the mean Hs-CRP between the age groups of the exposed and the control groups ( $p>0.05$ ). This was supported by the fact that low and high CRP concentrations were recorded for individuals of same age. This suggests that the toxic effect on exposure to petrol CRP concentration did not depend on age of an individual but on other factors. It has been reported that, the level of contamination with petrol and petroleum products, the route of exposure and nutritional status may influence the toxic effect [9][28][30]. Earlier studies, on individuals, chronically exposed to biomass fuel smoke showed that proximity contributed primarily to toxic effect of chemicals [24]. This result is consistent with a similar study by Clark et al [23] and Caravedo et al [25], but inconsistent with earlier studies of individuals, chronically exposed to biomass fuel smoke [21]. Dietary intake is known as a strong determinant to inflammation and disease risk [30][31]. Proper intake of fruits, vegetables processed grains, saturated fat, fish and fiber in preference to increased intake of red meat and unsaturated fat has been shown to reduce the level of inflammatory mediators thereby promoting good health and increase in life expectancy [32]. Physical activity and good nutrition status maintains good health by reduction of inflammation mediators [9][28]. There is availability of fruits and vegetables with low cost of living in Aba and its metropolis. Generally, automechanics in Aba eat well and are very active.



**Fig. 5. Bivariate regression analysis of tumor necrotic factor alpha**

#### 4. CONCLUSION

This study shows that the exposure of automechanics may significantly increase the serum IL-6 TNF- $\alpha$  and Hs-CRP levels. Increase in the serum levels of the inflammation markers is predictive of the danger of future pathology in automechanics compared with non automechanics in Aba metropolis. Age and duration did not influence significant variation in the automechanics.

#### CONSENT

All authors declare that 'written informed consent was obtained from the patient (or other approved parties) for publication of this case report and accompanying images.

#### ETHICAL APPROVAL

Ethical and legal standards were according to Helsinki considerations [33] (WMDH, 2002). Ethical approval was given by the Ethical Committee in Abia State Ministry of Health, Umuahia, Abia State.

#### DISCLAIMER

The products used for this research are commonly and predominantly use products in our area of research and country. There is absolutely no conflict of interest between the authors and producers of the products because we do not intend to use these products as an avenue for

any litigation but for the advancement of knowledge. Also, the research was not funded by the producing company rather it was funded by personal efforts of the authors.

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## COMPETING INTERESTS

Authors have declared that no competing interests exist.

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