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Evaluation of Lipid Profile and Oxidative Stress of Workers in Fast Food Restaurants and The Risk of Atherosclerosis

Muntaha A. Mohammed[1], Falah S. Al-Fartusie[1], Mohammed Z. Thani[1]*

[1]Department of Chemistry, College of Science, Mustansiriyah University, 10052 Baghdad, IRAQ.
*Correspondent contact: mountahaabd@gmail.com

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Abstract

Cooking fumes and biomass smoke pose significant threats to air degradation and human health, especially to people who work in this field on a daily basis, due to the large amounts of hazardous pollutants released from grilling activities. Chronic exposure to these fumes is associated with a risk of inflammation and oxidative stress, which can increase the risk of atherosclerosis. In this study, the lipid profile Glutathion (GSH) and Malondialdehyde (MDA) of workers in fast food restaurants and patients with atherosclerosis were evaluated. The study included 40 Atherosclerosis patients, 40 fast food workers, and 40 healthy individuals as a control group, the ages ranges of (20-40) years. The obtained results indicated a significant increase in cholesterol, triglycerides, LDL, and VLDL and a decrease in HDL ($p < 0.01$) in patients and fast-food workers compared to control group. The results also indicated a significant increase in the level of MDA, and a significant decrease in the level of GSH for patients and fast-food workers compared to the control. In conclusion, it appears that fast food workers experience oxidative stress and disruption in lipid metabolism that may increase the risk of atherosclerosis. Therefore, safety and quality control measures must be strengthened to reduce the occupational risks of fast-food restaurant workers.

Keywords: Atherosclerosis, lipid profile, Glutathion, Malondialdehyde, Fast Food Workers.

الخلاصة

تشكل أبخرة الطهي ودخان الكتلة الحيوية تهديدات كبيرة لتدهور الهواء وصحة الإنسان ، وخاصة للأشخاص الذين يعملون في هذا المجال وعلى أساس يومي ، بسبب الكميات الكبيرة من الملوثات الخطرة المنبعثة من أنشطة الشواء. يرتبط التعرض المزمن لهذه الأدخنة بخطر الالتهاب والإجهاد التأكسدي ، مما قد يزيد من خطر الإصابة بتصلب الشرايين. في هذه الدراسة ، تم تقييم ملف الدهون ، GSH الكلوتاثيون و MDA والمالون دي الديهايد للعاملين في مطاعم الوجبات السريعة والمرضى الذين يعانون من تصلب الشرايين. اشتملت الدراسة على ٤٠ مريضاً و ٤٠ عاملاً و ٤٠ من الأصحاء كمجموعة ضابطة تتراوح أعمارهم بين (٢٠-٤٠) سنة. شارت النتائج المتحصل عليها إلى زيادة معنوية ($p <0.01$) في الكوليسترول .Chol والدهون الثلاثية .T.G و البروتينات الدهنية منخفضة الكثافة LDL والبروتينات الدهنية المنخفضة جدا VLDL وانخفاض معنوي ($p <0.01$) في البروتينات الدهنية عالية الكثافة. HDL في المرضى والعاملين في الوجبات السريعة مقارنة بقيم التحكم. كما أشارت النتائج إلى زيادة معنوية في مستوى MDA ، وانخفاض معنوي في مستوى GSH للمرضى وعاملي الوجبات السريعة مقارنة بقيم التحكم. في الختام ، يبدو أن العاملين في الوجبات السريعة يعانون من الإجهاد التأكسدي واضطراب في التمثيل الغذائي للدهون مما قد يزيد من خطر الإصابة بتصلب الشرايين. لذا ، وجب تعزيز تدابير السلامة ومراقبة الجودة لتقليل المخاطر المهنية لعمال مطاعم الوجبات السريعة.

INTRODUCTION

Atherosclerosis is a multifactorial chronic and inflammatory disease of blood vessels (medium and large arteries), involves a number of risk factors and complex events including hypercholesterolemia, endothelial dysfunction [1] increased permeability to low density lipoproteins (LDL) and their sequestration on extracellular matrix in the intima of lesion-prone areas. These events promote LDL modifications, particularly by oxidation [2] which generates acute and chronic inflammatory responses implicated in atherogenesis and lesion progression. Reactive oxygen species (ROS) (which include both free radical and non-free radical oxygen intermediates), play a key-role at

each step of atherogenesis [2][3], in endothelial dysfunction, LDL oxidation, and inflammatory events involved in the initiation and development of atherosclerosis lesions [4]. Most advanced knowledge supporting the "oxidative theory of atherosclerosis" the nature and the cellular sources of ROS and antioxidant defense, as well as the mechanisms involved in the redox balance [5].

Kitchen and grill workers are exposed daily to various gaseous and particulate airborne pollutants that are formed during the preparation of foods using different cooking methods (such as frying, grilling, and roasting) [6]. Cooking fumes are thermal oxidative decomposition products that contain many hazardous pollutants such as respirable particulate matter (PM), heavy metals, black carbon, heterocyclic amines, volatile organic compounds including polycyclic aromatic hydrocarbons (PAHs), aldehydes, and carbonyls [7]. In 2010, the International Agency for Research on Cancer (IARC) included emissions from frying at high temperatures in the list of possible human carcinogens [8]. Emitted cooking fumes containing health-related contaminants (via inhalation and skin contact) will be absorbed into the human body of exposed workers and long-term exposure has been associated with potential health risks [9]. Some authors have reported that restaurant workers are at increased risk of developing myocardial infarction, especially in chefs and kitchen workers [10]. It is known that cooking oil fumes may induce lipid peroxidation and the expression of various cytokines, which cause oxidative DNA damage in epithelial cells of human lungs [11] Other authors have also reported a direct association of exposure to emissions from cooking activities with higher respiratory cancer risks for bakers and cooks among other food service workers [9][12].

Dyslipidemia is a prerequisite for atherosclerotic cardiovascular disease, and autopsy studies have shown a strong association between antemortem cholesterol levels and postmortem atherosclerosis in adolescents and young adults [13]. The prevention of cardiovascular disease in adulthood begins with the diagnosis and treatment of dyslipidemia in childhood and adolescence. Pathophysiology is transitioning from an older system based on phenotype to a newer system based on genetic or metabolic mechanism [14]. Identifiable familial forms account for less than 2% of all hyperlipidemia yet carry the highest cardiovascular risk.

Familial hypercholesterolemia (FH) [15] is the most common single-gene disorder of lipoprotein metabolism, with an autosomal co-dominant pattern of transmission and a population prevalence of 0.5-1.0%. The FCH is characterized by genetic and metabolic heterogeneity [16], and its usual manifestations are a modestly elevated LDL-C level, elevated very-low-density lipoprotein-cholesterol (VLDL-C) and triglyceride (TG) levels, and decrease of high-density lipoprotein cholesterol (HDL-C) [17].

Lipid peroxidation is defined as the uncontrolled oxidative degradation and dissociation of lipids by a non-enzymatic mechanism, which occurs when (ROS) interact with polyunsaturated fatty acids (PUFAs) [18] altering their physical and chemical properties and affecting the integrity of the cellular membrane. The Polyunsaturated fatty acids (PUFAs) and phospholipids are peroxidized in the membrane by free radical reactions [19]. As a result, lipoperoxides, various aldehydes (for example, malondialdehyde MDA, which is an unsaturated aldehyde have molecular formula $C_3H_4O_2$ or CH_2 $(CHO)_2$ [20]. The Malondialdehyde (MDA) is produced in the body by two processes: food consumption and lipid peroxidation in tissues. Variety of endogenous and exogenous factors influence the generation of lipid peroxidation products [21] especially MDA, have cytotoxic, mutagenic, and carcinogenic properties [21]. They can also prevent cells from defending themselves against oxidative stress by inhibiting enzymes involved in oxidative stress defense [22] showed high plasma concentrations of MDA in neonatal myocardial infarction [23].

Glutathione is a tripeptide consisting of amino acids L-glutamate, L-cysteine, and L- glycine, having a molecular weight of 307.4 g mol^{-1}. It is the most abundant non-protein thiol in cells; where it is present in intracellular concentration

from 1 to 15 mM [24-26]. In contrast, the extracellular concentrations of this thiol are usually lower [27]. It may be present in the form of reduced thiol (GSH), as well as oxidized disulfide (GSSG); although under normal conditions almost 99% of cellular GSH is in the form of reduced thiol [27-28]; about 1–2% GSH in cells is in oxidized form and increases only under conditions of oxidative stress [25]. In cells, GSH performs a variety of functions that contribute to maintain cellular homeostasis [29]; however, the most important is its intracellular antioxidant activity, where GSH is the most abundant cellular antioxidant [30] R•/RO•/ROO• + 2 GS–H(reduced) → GSSG (oxidised) + RH/ROH/ROOH.

In fact, it is important to highlight the occupational health risks that fast food restaurant workers may be exposed to, and to take the necessary steps to prevent associated adverse effects. Therefore, this study aims to assess the health status of fast-food restaurant workers as a result of occupational exposure to unhealthy work activities, and an attempt to predict the risk of developing atherosclerosis.

MATERIALS AND METHODS

Subjects and study design

This study includes 120 male subjects with an age range between (20-40) years. Subjects were collected from patient who attended the Consulting clinic \Baghdad teaching hospital \ medical city and the Iraqi Center for Cardiology at Ghazi Al-Hariri Hospital, Baghdad, Iraq between October 2022 and January 2023. The subjects include 40 healthy individuals as a control group, 40 fast food workers working outside the scope of health control within a specific area of Baghdad and 40 patients with atherosclerosis. The patients were diagnosed by a Joint Cardiology specialist. The patients and the fast-food workers were diagnosed for a long time, at least 1 year. Some of the patients and fast-food workers had a family history of the disease, and patients with diabetes were excluded.

Sample collection

From each individual, 10 ml of blood was drawn through a vein puncture using disposable syringes, 2 ml was collected in EDTA tube and 8 ml was collected in a gel tube. After collection, the whole blood samples were stored in a cooling fridge at 2-4 ^{o}C. The samples in the gel tubes were centrifuged at 3000 rpm for 10 minutes. The resulting serum was stored at -20 ^{o}C until the time of analysis.

Sample analysis

The body mass index (BMI) was calculated from the subjects studied by the following formula: BMI = weight (kg) / length (m^2). Levels of lipid profile (cholesterol, triglyceride, and HDL) were determined enzymatically using the colorimetric assay following the protocol of the available kits supplied by linear chemicals, Spain. Levels of LDL and VLDL were calculated according to the Friedewald equation [31].

The lipid peroxidation was determined in serum by measuring MDA concentration, depending on the reaction between MDA and thiobarbituric acid (TBA) in an acidic medium to give a pink color supernatant that is read at wavelength λ=535 nm, following the method described previously by Buege and Aust [32]. In addition, GSH concentration was measured according to the Ellman's assay by measuring the serum thiol concentration at λ=420 nm, following the procedure described by Sedlak and Lindsay [33].

Statistical analysis

Data was analyzed using SPSS statistical software, version 23. ANOVA was performed between patients, fast food workers and control groups, and the resulting values were expressed as mean and standard deviation (SD) were used to express values. The relationships between all study variables were also determined in this work using Pearson's correlation analysis. The statistical tests were significant at $p<0.05$ and highly significant at $p<0.01$ with a confidence interval of 95%.

RESULTS AND DISCUSSION

The results obtained for the present study showed that the mean age of the patient with atherosclerosis group was 34.0, fast-food Workers group 34.0 and the control group was 33.7 years with a non-significant p-value ($p>0.01$). Weight, height and BMI mean values for patients, workers and control groups were found approximately similar with a non-significant p-value ($p>0.01$). These results give a valuable chance to do a case study between patients with atherosclerosis, fast food workers and control. The present study reported elevated levels of triglycerides, (TG), cholesterol (Chlo), LDL-C VLDL-Cand decreased HDL-C cholesterol in the group of fast-food workers. The levels were found close to the levels recorded for patients with atherosclerosis. According to the lipid hypothesis, elevated levels of cholesterol in the blood lead to atherosclerosis which may increase the risk of heart attack, stroke [38], and peripheral arterial disease. The first step in the development of atherosclerosis is the exposure of vascular cells to excess fat with concomitant activation/dysfunction in the endothelium, the assimilation and deposition of lipids in the inner lining [39], located near the points of branching and along the internal bends, are most susceptible [40]. The results of the lipid profile analysis are presented in Table 2 and Figures (1, 2, and 3). showed a significant increase in the blood serum of male fast-food workers compared with control group also the study showed a higher increase in the levels of the lipid profile of male patients with atherosclerosis compared with control group, as shown in Table 2 and Figures (1, 2, and 3). These results confirm the potential impact of occupational exposure to fast food workers outside health control and the absence of preventive measures to reduce the risks associated with long-term exposure to hazardous pollutants emitted from barbeque activities and their role in weakening the total capacity of antioxidants and escalating oxidative stress that damages endothelial cells of blood vessels and contributes to Increased oxidation of LDL, which contributes to the development of atherosclerosis. These results are consistent with the results of a previous study. [34-37]. Epidemiological studies have shown a positive relationship between total cholesterol concentrations and mortality from coronary heart disease (CHD). It has been found that small, dense LDL particles are more amenable to hardening than large, floating LDL particles, and LDL oxidation increases atherosclerosis [41]. Smoking plays a strong role not only in atherosclerotic cardiovascular disease (ASCVD) initiation but also significantly contributes to and causes disease progression and fatal cardiovascular outcomes. The key processes in smoking-induced atherogenesis initiation are endothelial dysfunction and damage [42] increase in and oxidation of proatherogenic lipids, as well as decrease of HDL-C induction of inflammation, and the shift toward a procoagulant state in the circulation) [42]. This explains why GHS level decreased, lipid peroxidation level increased, and HDL-C level decreased in the study sample of fast-food workers that were compared to sclerosis group and control group as shown in the current data clearly indicated that passive smoking also can lead to life-threatening conditions. Current data clearly show that also secondhand smoking can trigger life-threatening conditions [43].

Table 1. Measurement information for patients with atherosclerosis, fast food workers and control groups.

	control		Fast Food Workers		Patient		
parameters	**Mean**	**Std. Deviation**	**Mean**	**Std. Deviation**	**Mean**	**Std. Deviation**	**P-value**
Age (year)	33.7	3.6	34	4.6	34	5.1	0.554 NS
Weight (kg)	86.9	9.5	84	11.7	84.5	9.9	0.464 NS
Height (cm)	180	5.5	175	6.9	177	5.7	0.478 NS
BMI (kg/m^2)	26.6	2.5	27	3.19	26.4	2.3	0.582 NS

*NS: Non-Significant.

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Table 2. Lipid profiles parameters for patient with atherosclerosis, fast food workers and control groups.

	control		fast food Workers		Patient		
parameters	**Mean**	**Std. Deviation**	**Mean**	**Std. Deviation**	**Mean**	**Std. Deviation**	**P-value**
Cholestrol(Chol)	184.02	39.279	229.0	28.7	237.0	40.8	.001*
TG	107.20	47.608	243.8	70.19	249.8	78.15	.001*
HDL-C	45.92	9.806	43.8	5.62	36.6	7.78	.001*
VLDL-C	21.50	9.467	47.5	11.5	52.9	23.9	.001*
LDL-C	115.57	30.58	140.4	26.5	149.3	32.9	.001*

*Significant at P<0.01

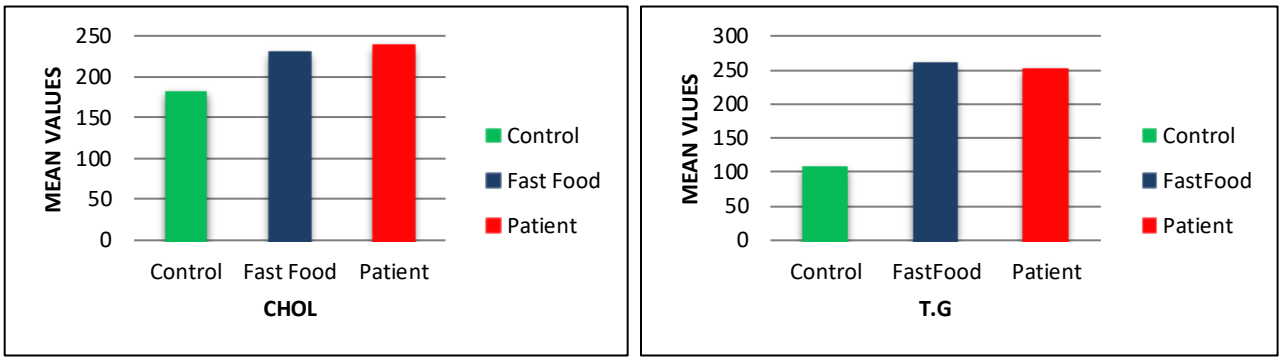

Figure 1. The Chol. and T.G levels in fast food workers, patients with atherosclerosis and control.

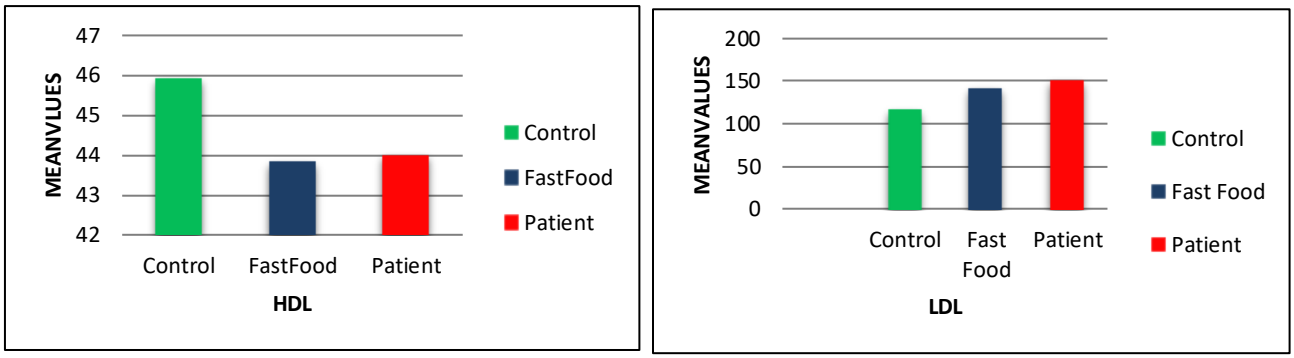

Figure 2. The HDLand LDL levels in fast food workers, patients with atherosclerosis and control.

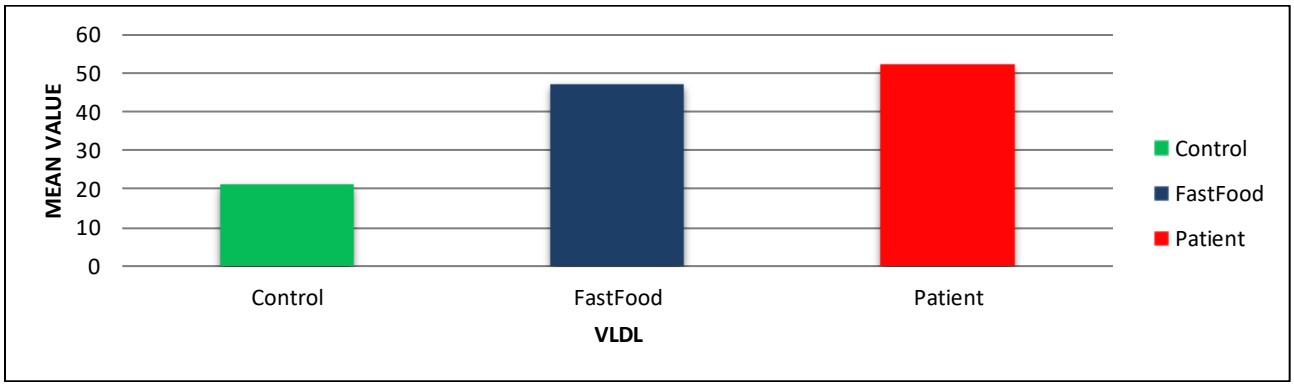

Figure 3. The VLDL level in fast food workers, patients with atherosclerosis and control.

The results obtained for the present study of GSH and MDA of fast-food workers, atherosclerotic patients and control groups are shown in Table 3, and Figure 4. The estimated mean ± SD of GSH for the fast-food workers, patients with atherosclerosis and control groups

was 515.025 ± 27.5 µmol/L, 441.775± 62.9. µmol/L and 948.86 ± 82.66 µmol/L respectively. Statistically, it appears that the level of GSH in fast food workers and patients with atherosclerosis is significantly lower ($P < 0.001$) than it was in the control group, and between the two groups of fast food and patients with atherosclerosis, we find that the patients with atherosclerosis are slightly higher than those in fast food workers. In contrast, the mean ± SD values of MDA for fast food workers, patients with atherosclerosis and control groups were 3.078 ± 0.571 µ mol/L, 3.86 ± 0.579 µ mol/L and 1.53 ± 0.535 µ mol/L, respectively. The results showed that the MDA values were significantly higher ($p < 0.01$) in the fast-food workers and atherosclerosis groups compared to the control group, and in the fast-food workers group seemed slightly lower than the atherosclerosis patients, as shown in Table 3 and Figure 4.

Based on these results, it is found that the results are very similar and support the findings reported in previous study [44]. Endogenous GSH deficiency, characterized by decreased synthesis and/or increased depletion of GSH, has been linked to the pathophysiology of atherosclerosis via oxidative stress and inflammatory pathways [45]. Glutathione is an essential antioxidant that has been shown to have an important function in cellular resistance to oxidative damage [46]. It has been shown that excessive production of ROS accelerates the process of cell destruction.

Numerous studies have shown that an imbalance in redox balance can cause cardiovascular complications. Reactive oxygen species are produced by all layers of blood vessels, and they act as signaling molecules that regulate numerous functions such as vascular smooth muscle cell contraction, relaxation, and growth [47]. In contrast, an excessive or persistent increase in the generation of ROS plays an essential role in endothelial dysfunction (ED) and the development of CVD. Some cardiovascular risk factors are associated with either increased ROS production or decreased plasma GSH levels [48]. In both cases, cell damage is due to direct oxidative effects on proteins, lipids, and DNA [49].

The MDA findings of this research are consistent with those of other studies, which found significantly different and elevated levels of MDA in atherosclerotic patients compared to healthy subjects and in the serum of fast-food workers compared to healthy subjects.

The results also showed the closeness between a group of patients with atherosclerosis and workers in fast food. MDA is a product of peroxidation of polyunsaturated fatty acids that is used as a measure of oxidative damage[50]. Since free radicals play a major role in cellular immune response and inflammation in fast food workers and patients with atherosclerosis, they are indirectly involved in cell damage. Increased MDA and decreased markers of antioxidant status, such as GSH, support the idea that oxidative stress plays a role in the development of atherosclerosis [51]. Markers of lipid peroxidation can be used as surrogate markers of disease activity [51]. The MDA findings in this study could indicate increased lipid peroxidation and oxidative stress in fast food workers and patients with atherosclerosis, which resulted in a lower concentration of the antioxidant GSH [52].

The results in this study indicate that fast food workers may be at risk of developing atherosclerotic diseases due to their exposure to occupational risks due to lack of awareness of occupational health and safety requirements stipulated in the Occupational Health and Safety Organization [53].

Table 3. The GSH and MDA levels for patients with atherosclerosis, fast food workers and control groups.

	Control		Fast Food Workers		Atherosclerosis		
Parameters	**Mean**	**Std. Deviation**	**Mean**	**Std. Deviation**	**Mean**	**Std. Deviation**	***p*-value**
GSH (µmol/l)	948.8675	82.66851	515.025	27.578	441.7750	62.92763	0.001*
MDA (µmol/l)	1.5370	.53579	3.0785	.57122	3.8623	.57946	0.001*

*Significant at P<0.01, NS: Non-Significant

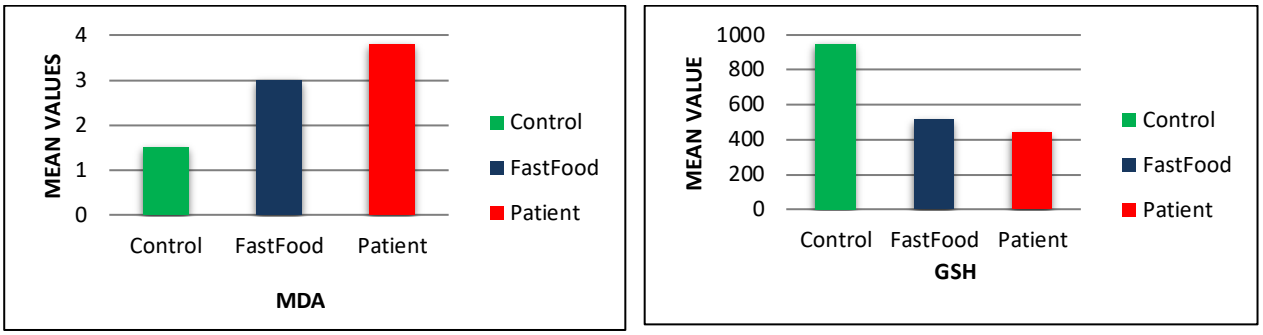

Figure 4. The GHS and MDA levels in fast food workers, patients with atherosclerosis and control.

The association between all the variables included in this study of fast-food workers and patients with atherosclerosis was examined using Pearson correlation analysis, and the results are represented in the Tables 4 and 5. For fast food workers group, the results revealed a positive correlation between the levels of TG, LDL and VLDL, with the level of Chol. The analysis also revealed a positive correlation between TG levels, with VLDL level in Fast Food group. In addition, a negative correlation observed between GSH and MDA of fast-food workers group.

Table 4. Correlations between variables in the fast food workers group

	Chol	TG	HDL	LDL	VLDL	GSH	MDA
Chol	1	.358*	0.23	.829**	.368*	-.104	-.113
TG	.358*	1	.089	.044	.820**	.021	-.128
HDL	.023	.089	1	-.165	.003	-.192	.095
LDL	.829**	.044	-.165	1	.014	.090	-.074
VLDL	.368*	.820**	.003	.014	1	-.007	-.181
GHS	-.104	.021	-.192	.090	-.007	1	-.118*
MDA	-.113	-.128	.095	-.074	-.181	-.118*	1

*. Correlation is significant at the 0.05 level (2-tailed). **. Correlation is significant at the 0.01 level (2-tailed).

In the patients group, the results revealed a positive correlation between the levels of, LDL, with the level of Chol. The analysis also revealed a positive correlation between TG level with V LDL level, and negative correlation between LDL and TG. The results revealed a negative correlation between GSH and MDA.

Table 5. Correlations between variables in the patient Atherosclerosis group

	Chol	TG	HDL	LDL	VLDL	GSH	MDA
Chol	1	-.099	.004	.511**	.044	-.075	-.059
TG	-.099	1	.026	-.322*	.717**	-.244	.116
HDL	.004	.206	1	-.006	.226	.087	-.139
LDL	.511**	-.322*	-.006	1	-134	-112	-.062
VLDL	.044	.717**	.226	-.134	1	-.283	-.111
GSH	-.075	-.244	.087	-.112	-.283	1	-.063*
MDA	-.059	.116	-.139	-.062	-.111	-.063*	1

*. Correlation is significant at the 0.05 level (2-tailed). **. Correlation is significant at the 0.01 level (2-tailed).

CONCLUSIONS

This study evaluates some important biomarkers for fast food workers outside health control that enhance their risk factors for atherosclerosis when compared to their peers with the disease. The lipid profile was observed and characterized by an increase in cholesterol, TG , LDL-C, VLDL-C, and a decrease in HDL-C The increase in oxidative activity was also observed by measuring the level of GSH, which showed a

decrease, and measuring lipid peroxide MDA, which showed rise in fast food workers as indicators of occupational exposure to unhealthy work activities, as a result of the toxic substances secreted by these restaurants such as fumes and gases resulting from grilling, frying and cooking operations that have unhealthy and environmental impact.

Further studies, including larger numbers of participants being monitored over a longer period of time and addressing different determinants of exposure, are needed to explore the results achieved and to enhance control safety measures to reduce occupational risks for fast food workers. Furthermore, monitoring of workers breathing air should also be considered to explore the relationship between PM, airborne and/or skin concentrations and their potential sources with the biomarkers level consider in this study.

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Disclosure and Conflict of Interest: The authors declare that they have no conflicts of interest.

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