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

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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 range 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.

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 in...
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]. Exhaled 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 C3H4O2 or
CH2 (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
carbons 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 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 °C. The samples in the gel tubes were centrifuged at 3000 rpm for 10 minutes. The resulting serum was stored at -20 °C until the time of analysis.

**Sample analysis**

The body mass index (BMI) was calculated from the subjects studied by the following formula: BM

\[ \text{BMI} = \frac{\text{weight (kg)}}{\text{length (m)}} \]

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 \( \lambda=535 \) 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 \( \lambda=420 \) 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-C and 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>
<thead>
<tr>
<th>parameters</th>
<th>control</th>
<th>Fast Food Workers</th>
<th>Patient</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (year)</td>
<td>Mean</td>
<td>33.7</td>
<td>34</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>86.9</td>
<td>9.5</td>
<td>84</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>180</td>
<td>5.5</td>
<td>175</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>26.6</td>
<td>2.5</td>
<td>27</td>
</tr>
</tbody>
</table>

*NS: Non-Significant.*
Table 2. Lipid profiles parameters for patient with atherosclerosis, fast food workers and control groups.

<table>
<thead>
<tr>
<th>parameters</th>
<th>control Mean</th>
<th>control Std. Deviation</th>
<th>Fast Food Workers Mean</th>
<th>Fast Food Workers Std. Deviation</th>
<th>Patient Mean</th>
<th>Patient Std. Deviation</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholesterol</td>
<td>184.02</td>
<td>39.279</td>
<td>229.0</td>
<td>28.7</td>
<td>237.0</td>
<td>40.8</td>
<td>.001*</td>
</tr>
<tr>
<td>TG</td>
<td>107.20</td>
<td>47.608</td>
<td>243.8</td>
<td>70.19</td>
<td>249.8</td>
<td>78.15</td>
<td>.001*</td>
</tr>
<tr>
<td>HDL-C</td>
<td>45.92</td>
<td>9.806</td>
<td>54.5</td>
<td>11.5</td>
<td>149.3</td>
<td>32.9</td>
<td>.001*</td>
</tr>
<tr>
<td>VLDL-C</td>
<td>21.50</td>
<td>9.467</td>
<td>47.5</td>
<td>11.5</td>
<td>52.9</td>
<td>23.9</td>
<td>.001*</td>
</tr>
<tr>
<td>LDL-C</td>
<td>115.57</td>
<td>30.58</td>
<td>140.4</td>
<td>26.5</td>
<td>149.3</td>
<td>32.9</td>
<td>.001*</td>
</tr>
</tbody>
</table>

*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 HDL and 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>
<thead>
<tr>
<th>Parameters</th>
<th>Control</th>
<th>Fast Food Workers</th>
<th>Atherosclerosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>GSH (µmol/l)</td>
<td>948.8675</td>
<td>515.025</td>
<td>441.7750</td>
</tr>
<tr>
<td>MDA (µmol/l)</td>
<td>1.5370</td>
<td>3.0785</td>
<td>3.8623</td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Mean</th>
<th>Std. Deviation</th>
<th>Mean</th>
<th>Std. Deviation</th>
<th>Mean</th>
<th>Std. Deviation</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>GSH (µmol/l)</td>
<td>948.8675</td>
<td>82.66851</td>
<td>515.025</td>
<td>27.578</td>
<td>441.7750</td>
<td>62.92763</td>
<td>0.001*</td>
</tr>
<tr>
<td>MDA (µmol/l)</td>
<td>1.5370</td>
<td>.53579</td>
<td>3.0785</td>
<td>.57122</td>
<td>3.8623</td>
<td>.57946</td>
<td>0.001*</td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th></th>
<th>Chol</th>
<th>TG</th>
<th>HDL</th>
<th>LDL</th>
<th>VLDL</th>
<th>GSH</th>
<th>MDA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chol</td>
<td>1</td>
<td>.358*</td>
<td>.23</td>
<td>.829**</td>
<td>.368*</td>
<td>-.104</td>
<td>-.113</td>
</tr>
<tr>
<td>TG</td>
<td>.358*</td>
<td>1</td>
<td>.089</td>
<td>.044</td>
<td>.820**</td>
<td>.021</td>
<td>-.128</td>
</tr>
<tr>
<td>HDL</td>
<td>.023</td>
<td>.089</td>
<td>1</td>
<td>-.165</td>
<td>.003</td>
<td>-.192</td>
<td>.095</td>
</tr>
<tr>
<td>LDL</td>
<td>.829**</td>
<td>.044</td>
<td>-.165</td>
<td>1</td>
<td>.014</td>
<td>.090</td>
<td>-.074</td>
</tr>
<tr>
<td>VLDL</td>
<td>.368*</td>
<td>.820**</td>
<td>.003</td>
<td>.014</td>
<td>1</td>
<td>-.007</td>
<td>-.181</td>
</tr>
<tr>
<td>GSH</td>
<td>-.104</td>
<td>.021</td>
<td>-.192</td>
<td>.090</td>
<td>-.007</td>
<td>1</td>
<td>-.118*</td>
</tr>
<tr>
<td>MDA</td>
<td>-.113</td>
<td>-.128</td>
<td>.095</td>
<td>-.074</td>
<td>-.181</td>
<td>-.118*</td>
<td>1</td>
</tr>
</tbody>
</table>

* 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, VLDL and VLDL 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

<table>
<thead>
<tr>
<th></th>
<th>Chol</th>
<th>TG</th>
<th>HDL</th>
<th>LDL</th>
<th>VLDL</th>
<th>GSH</th>
<th>MDA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chol</td>
<td>1</td>
<td>-.099</td>
<td>.004</td>
<td>.511**</td>
<td>.044</td>
<td>-.075</td>
<td>-.059</td>
</tr>
<tr>
<td>TG</td>
<td>-.099</td>
<td>1</td>
<td>.026</td>
<td>-.322*</td>
<td>.717**</td>
<td>-.244</td>
<td>.116</td>
</tr>
<tr>
<td>HDL</td>
<td>.004</td>
<td>.206</td>
<td>1</td>
<td>-.006</td>
<td>.226</td>
<td>.087</td>
<td>-.139</td>
</tr>
<tr>
<td>LDL</td>
<td>.511**</td>
<td>-.322*</td>
<td>-.006</td>
<td>1</td>
<td>-.134</td>
<td>-.112</td>
<td>-.062</td>
</tr>
<tr>
<td>VLDL</td>
<td>.044</td>
<td>.717**</td>
<td>.226</td>
<td>-.134</td>
<td>1</td>
<td>-.283</td>
<td>-.111</td>
</tr>
<tr>
<td>GSH</td>
<td>-.075</td>
<td>.244</td>
<td>.087</td>
<td>-.112</td>
<td>-.283</td>
<td>1</td>
<td>-.063*</td>
</tr>
<tr>
<td>MDA</td>
<td>-.059</td>
<td>.116</td>
<td>-.139</td>
<td>-.062</td>
<td>-.111</td>
<td>-.063*</td>
<td>1</td>
</tr>
</tbody>
</table>

* 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.

References


