Assessment of workers' lipid profiles in cement factories in South-South Nigeria

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Abstract

Cement dust exposure has also been associated with both increased oxidative stress and decreased anti-oxidant capacity, which in turn can promote lipid peroxidation and consequently dyslipidaemia. This study was carried out to determine the lipid profile of cement factory workers in Ekpoma and environs. A total of eighty (80) samples were used in this study comprising fifty (50) cement factory workers (subjects) and thirty (30) non-cement factory workers (control). Lipid profile was determined using standard laboratory procedures. The results were presented in tables as mean ± standard deviation. Statistical analysis was done using one-way analysis of variance (ANOVA) and the student's t-test. Significant difference was accepted at p<0.05. The results presented in mean ± standard deviation showed that the age of the subjects and control was 24.60±4.46 and 30.45±6.80, the Height (m) was 1.64±0.09 and 1.82±0.74, the Weight (kg) was 64.25±4.91 and 71.10±12.75, while the BMI (kg/m²) was 23.78±2.16 and 22.35±4.09 respectively. The concentration of Total cholesterol (mg/dl) in mean ± standard deviation of the subjects and control group was 157.65±25.07 and 152.40±22.67, Triglyceride (mg/dl) was 192.45±44.84 and 111.75±11.35, HDL (mg/dl) was 35.65±3.36 and 45.60±5.89, while LDL (mg/dl) was 84.30±20.16 and 77.35±15.70 respectively. There was no significant difference (p>0.05) in the lipid profile of cement factory workers with respect to duration of work. There was significant difference (p<0.05) in Height, Triglyceride and LDL of the subjects with respect to age, while others were not significant (p>0.05). The study concludes that total cholesterol, triglyceride and LDL were higher among cement workers compared with control, while HDL was lower among cement workers. Long term exposure to cement dust should be monitored, thus calling for collaboration between the health workers and cement factory management to ensure frequent monitoring of their workers' health.

Keywords: Cement; Dust; Lipid profile; Workers; Factory

1. Introduction

Cement is an important binding agent for construction industry and is produced world-wide in large amounts. Cement is a fine, grey or white powder largely made up of Cement Klin Dust (CKD), a by-product of the final cement product usually stored as waste in open pits and unlined landfills. Cement dusts are basically made of calcium oxide, silicon oxide, aluminum oxide with little quantity of iron oxide, magnesium oxide, potassium, sodium and sulphur (Neboh et al., 2015). Cement is an important key to economic growth because of its role in construction, housing and infrastructural development (Potgieter, 2012). Its demand is directly associated with economic growth and the need for rapid infrastructural development in many growing economies is the main motivation for the tremendous growth
in cement manufacture and use (Potgieter, 2012). In spite of its popularity and profitability, the cement industry faces many challenges due to sustainability issues, environmental and health concerns (Khana et al., 2014).

Lipids and lipoproteins are essential constituents of the body, and their activities assist in maintenance of the body homeostasis. The lipids can be classified as Total Cholesterol (TC) and its derivatives such as; Triglycerides (TAG), Low Density Lipoprotein (LDL), High Density Lipoprotein (HDL) and Very Low-Density Lipoprotein (VLDL) cholesterol. The cholesterol along with some other types of fats cannot dissolve in the blood. In order to be transported to and from cells, they have to be specially carried by certain molecules called lipoproteins, which consist of an outer layer of protein with an inner core of cholesterol and triglycerides (Forouzanfar et al., 2016).

Cholesterol is an organic molecule. It is a sterol or modified steroid defined as a type of lipid (Forouzanfar et al., 2016). Cholesterol is a fatty substance essential for normal body functioning. Higher levels of cholesterol in the blood are associated with an increased risk of coronary heart disease (CHD), stroke, and peripheral arterial disease. The Global Burden of Disease Project estimated in 2015, high total cholesterol (TC) which accounted for 4.3 million deaths globally and the loss of 88.7 million disability-adjusted life years (Forouzanfar et al., 2016). Globally, the burden attributable to high TC is increasing, probably because of aging populations and westernization of traditional diets. An observational epidemiologic study reported that decreasing levels of TC below those currently considered “normal” would further reduce the risk of CHD and stroke (Huxley et al., 2002).

High density lipoprotein cholesterol (HDL-C) is the smallest of the lipoprotein particles. It is the densest because it contains the highest proportion of protein-to-lipid ratio (Sirtori, 2006). Increasing concentrations of HDL particles are strongly associated with decreasing the accumulation of atherosclerosis within the walls of arteries (Toth, 2005). This is important because atherosclerosis eventually results in sudden plaque ruptures, CVD, stroke, and other vascular diseases. HDL-C is sometimes called “good cholesterol” despite being the same as cholesterol in LDL particles. Those with higher levels of HDL-C tend to have reduced risk with CVDs, while those with low HDL-C levels (especially <40 mg/dL or about 1 mmol/L) have increased risks of heart disease (Toth, 2005). Higher native HDL-C levels are correlated with better cardiovascular health (Sirtori, 2006) but it does not appear that further increasing one’s HDL-C improves cardiovascular outcomes (National Institute of Health, 2011).

Low density lipoprotein cholesterol (LDL-C) is one of the five major groups of lipoproteins which transport fat molecules around the body in extracellular water (CDC, 2017). LDL-C delivers fat molecules to cells. It can contribute to atherosclerosis if oxidized within the walls of the arteries. It is important to note that the popular press calls LDL-C a “bad cholesterol”. However, much recent research has shown that it is not necessarily bad because LDL particle appears harmless until they are within the blood vessel walls and oxidized by free radical. It has been stipulated that ingesting antioxidant and minimizing free radical exposure may reduce LDL-C contribution to atherosclerosis, though results are inconclusive (Stocker & Keaney, 2004).

In humans, high levels of triglycerides (TGs) in the bloodstream have been linked to atherosclerosis and, by extension, the risk of heart disease and stroke (Drummond & Brefere, 2014). However, the relative negative impact of raised levels of TG compared to that of LDL-C:HDL-C ratio is yet unknown. The risk can be partly accounted for by a strong inverse relationship between TG level and HDL-C. Their levels remain temporarily high for a period after eating; hence, they are best tested after fasting 8–12 h. The American Heart Association recommends optimal TG level of 100 mg/dl (1.1 mmol/L) or lower to improve heart health (Nordestgaard et al., 2007). High level of TGs is a component of metabolic syndrome (a cluster of conditions that includes too much fat around the waist, high blood pressure, high blood sugar, obesity, and abnormal cholesterol levels). Extremely high TG of >1000 mg/dl (11.29 mmol/L) can cause acute pancreatitis. Certain medications such as beta-blockers, birth control pills, diuretics, or steroids could also increase TG levels. Any cause of malnutrition like cancer, memory loss, depression, and trauma can deplete the body of fat, thus contributing to low TGs levels (Mayo Clinic, 2015).

The majority of cardiovascular disease (CVD) is caused by risk factors that can be controlled, treated or modified. Cardiovascular risk is outcome of the contributions of the effects of many risk factors. The clustering together of individual factors in a significant pattern tends to increase an individual’s total cardiovascular risk (Mendis et al., 2011). These cardiovascular risk factors include high blood pressure, abnormal blood lipid, diabetes, overweight and obesity, smoking, alcohol intake, physical inactivity. Other cardiovascular risk factors are non-modifiable and include age, gender and family history (Mendis et al., 2011). The level of cholesterol in blood is related to the development of atherosclerosis and myocardial infarction. Abnormality of cholesterol metabolism may lead to cardiovascular accidents and heart attacks (Vasudevan et al., 2011).
Workers associated with cement are exposed to dust at various stages of manufacturing which include production and post-production processes. Main entries of cement dust particle into the body are by inhalation or swallowing and as such, its target of deposition is the respiratory and gastrointestinal tracts also affected are the skin and the eyes (Meo, 2004). Hazardous materials in cement such as calcium oxides are corrosive to human tissue; crystalline silica is abrasive to the skin and damages the lungs and chromium causes allergic reactions (Syed et al., 2013) resulting in various health problems such as chronic cough, phlegm production, impairment of lung function, chest tightness, bronchial asthma restrictive lung disease, skin irritation, conjunctivitis, stomach ache, headache as well as cancer of the lungs (Meo et al., 2008). A single and short-term exposure to cement dust presents with little or no hazard. However, prolonged or repeated exposure, depending on the duration, level of exposure and individual sensitivity has health implications on the skin, eyes, respiratory and haematological systems (Mojiminiyi et al., 2008; John & Olubayo, 2011; Zawilla et al., 2014).

There is a dearth however of information on the effect of exposure to cement dust on cardiovascular risk factors. Some studies have associated cement dust exposure with both increased oxidative stress and decreased anti-oxidant capacity, which in turn can promote lipid peroxidation and consequently dyslipidaemia (Aydin et al., 2010). A modest association between exposure to cement and type 2 diabetes mellitus has also been suggested (Haro-Garcia et al., 2010). These conditions are associated with increased cardiovascular disease risk. The few available studies were carried out on Caucasian and Hispanic populations, hence the need for this study to evaluate the effect of cement dust on lipid profile of cement factory workers in our study area.

2. Materials and methods

This study was carried out in Ekpoma. Ekpoma is the administrative headquarters of Esan West Local Government Area in Edo state which falls within the rain forest/savannah transitional zone of south western Nigeria. Ekpoma has a land area of 923 square kilometers with a population of 170,123 people as at the 2006 census. The town has an official post office and it is the home of Ambrose Alli University. The area lies between latitudes 6° 43’ and 6° 45’ North of the equator and longitudes 6° 5’ and 6° 8’ East of the Greenwich median. Majority of people in Ekpoma are students, Lecturers/Teachers, civil servants, farmers, traders, business men/women, doctors, lawyers and self-employed. The inhabitants are mainly Christian, few Muslims and pagans scattered within the area. Ekpoma is made up of many communities, including Eguare, Iruekpen, Emaudo, Ujolen, Ihumudumu, Illeh, Uke, Uhiele, Ujemen, Ukpenu, Egoro, Emuhli, Igor and Idumebo.

2.1. Population of study

A total of eighty (80) subjects between the ages of 20-60 years were recruited for this study which consist of fifty (50) cement factory workers and fifty (30) individuals who are not cement factory workers nor exposed to cement dust (controls).

Apprently healthy cement factory workers and those constantly exposed to cement dust who gave their consent were included in this study. Individuals with a history of cigarette smoking, tobacco sniffing or chewing, liver disease or pulmonary disorders, chronic organ or systemic illness, and long-term medication were excluded from the study.

2.2. Sample Collection

For each participant, about five (5) ml of blood was collected by veinpuncture under aseptic conditions into a labeled dry, clean plain sample container. They were allowed to clot and centrifuged at 3,500 revolutions per minute for 5 minutes. After centrifuging, the serum was separated with the aid of a Pasture pipette and dispensed into dry chemically clean serum container, after which the samples were analyzed immediately or stored at −20°C for subsequent analysis.

2.3. Sample Analysis

2.3.1. Determination of total cholesterol

Enzymatic Endpoint method (CHOD-PAP).

- Procedure

Ten microlitres of distilled water, standard and samples were dispensed into tubes labelled blank, standard and sample respectively. One millilitre of cholesterol reagent was added into the respective test tubes, and the contents were mixed
and incubated at 37°C for five minutes. The absorbance of standard and samples were measured against blank at a wavelength of 500nm using spectrophotometer.

2.3.2. Determination of triglyceride
Colorimetric method.

- **Procedure**

Ten microlitres of distilled water, standard and samples were dispensed into tubes labelled blank, standard and sample respectively. One millilitre of triglyceride reagent was added into the respective test tubes, and the contents were mixed and incubated at 37°C for five minutes. The absorbance of standard and samples were measured against blank at a wavelength of 500nm using spectrophotometer.

2.3.3. Estimation of high-density lipoprotein
Precipitation method.

- **Procedure**

**Stage I:** Two hundred microlitres of standard/sample was dispensed into test tube labelled standard/sample. Five hundred microlitres of precipitant was added into the test tubes and mixed well, the contents were allowed to stand for ten minutes at room temperature and then centrifuged for ten minutes at 4000rpm. The supernatant was separated and the cholesterol content was estimated using CHOD-PAP method.

**Stage II:** One hundred microlitres of distilled water, standard supernatant and sample supernatant was added into test tubes labelled blank, standard and sample respectively. One millilitre of cholesterol reagent was added into the respective test tubes; the contents were mixed and incubated at 37°C for five minutes in the water bath. The absorbance of standard and sample were measured against blank at the wavelength of 500nm using spectrophotometer.

**Estimation of low-density lipoprotein:** Friedewald formula

\[
LDL\text{-cholesterol (mmol/l)} = \text{Total cholesterol} - (\text{TG}/2.2 - \text{HDL-cholesterol})
\]

2.4. Data Analysis
The results were presented using tables. Data was presented as mean ± S.D (standard deviation). Comparison was made between subjects and control groups using one-way analysis of variance (ANOVA) and the student’s t-test. Significant difference was accepted at p<0.05.

3. Result

3.1. Mean Age, BMI and Lipid profile of Cement factory workers and Control

Table 1 showed the Age, BMI and lipid profile of cement factory workers and control group. The results presented in mean ± standard deviation showed that the age of the subjects and control was 24.60±4.46 and 30.45±6.80, the Height (m) was 1.64±0.09 and 1.82±0.74, the Weight (kg) was 64.25±4.91 and 71.10±12.75, while the BMI (kg/m²) was 23.78±2.16 and 22.35±4.09 respectively. There was no significant difference (p>0.05) in the BMI of the subjects compared with control group, however, there was significant difference (p<0.05) in Age, Height and Weight respectively. The concentration of Total cholesterol (mg/dl) in mean ± standard deviation of the subjects and control group was 157.65±25.07 and 152.40±22.67, Triglyceride (mg/dl) was 192.45±44.84 and 111.75±11.35, HDL (mg/dl) was 35.65±3.36 and 45.60±5.89, while LDL (mg/dl) was 84.30±20.16 and 77.35±15.70 respectively. There was significant difference (p<0.05) in the mean triglycerides and HDL cholesterol of the subjects compared with control group.
Table 1 Mean Age, BMI and Lipid profile of Cement factory workers and Control

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Subjects Mean ± SD (n = 50)</th>
<th>Controls Mean ± SD (n = 30)</th>
<th>t-value</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>24.60±4.46</td>
<td>30.45±6.80</td>
<td>-3.267</td>
<td>0.004</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.64±0.09</td>
<td>1.82±0.74</td>
<td>-6.996</td>
<td>0.000</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>64.25±4.91</td>
<td>71.10±12.75</td>
<td>-2.495</td>
<td>0.022</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>23.78±2.16</td>
<td>22.35±4.09</td>
<td>1.709</td>
<td>0.104</td>
</tr>
<tr>
<td>T.Chol (mg/dl)</td>
<td>157.65±25.07</td>
<td>152.40±22.67</td>
<td>0.096</td>
<td>0.924</td>
</tr>
<tr>
<td>T.G (mg/dl)</td>
<td>192.45±44.84</td>
<td>111.75±11.35</td>
<td>7.616</td>
<td>0.000</td>
</tr>
<tr>
<td>HDL (mg/dl)</td>
<td>35.65±3.36</td>
<td>45.60±5.89</td>
<td>-6.357</td>
<td>0.000</td>
</tr>
<tr>
<td>LDL (mg/dl)</td>
<td>84.30±20.16</td>
<td>77.35±15.70</td>
<td>-1.257</td>
<td>0.224</td>
</tr>
</tbody>
</table>

Keys: BMI – Body mass index; T.Chol – Total cholesterol; T.G. – Total cholesterol; HDL – High density lipoprotein; LDL - Low density lipoprotein; n – Sample size

3.2. BMI and Lipid profile of cement factory workers with respect to duration of work

Table 2 showed the BMI and lipid profile of the subjects with respect to duration of work. The results obtained showed that the Height (m) of the cement workers with work duration of less than 1 year, 1–2 years and 3–5 years was 1.64±0.08, 1.61±0.08 and 1.58±0.11, Weight (kg) was 63.67±4.71, 64.67±3.40 and 65.33±5.02, while BMI (kg/m²) was 23.97±2.89, 24.53±2.51 and 26.19±3.03 respectively. Furthermore, the mean of Total cholesterol (mg/dl) of the cement workers with work duration of less than 1 year, 1–2 years and 3–5 years was 152.78±21.81, 150.44±18.97 and 155.67±26.41, Triglyceride (mg/dl) was 227.67±53.86, 210.33±42.94 and 212.89±72.28, HDL (mg/dl) was 35.56±4.56, 34.33±1.73 and 34.44±4.03, while LDL (mg/dl) was 73.33±20.39, 73.44±11.49 and 78.89±16.57 respectively. There was no significant difference (p>0.05) in the lipid profile of cement factory workers with respect to duration of work.

Table 2 BMI and Lipid profile of cement factory workers with respect to duration of work

<table>
<thead>
<tr>
<th>Parameters</th>
<th>&lt;1 year Mean ± SD</th>
<th>1–2 years Mean ± SD</th>
<th>3–5 years Mean ± SD</th>
<th>F-value</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Height (m)</td>
<td>1.64±0.08</td>
<td>1.61±0.08</td>
<td>1.58±0.11</td>
<td>0.526</td>
<td>0.613</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>63.67±6.02</td>
<td>64.67±3.40</td>
<td>65.33±5.02</td>
<td>1.440</td>
<td>0.188</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>23.97±2.89</td>
<td>24.53±2.51</td>
<td>26.19±3.03</td>
<td>0.521</td>
<td>0.616</td>
</tr>
<tr>
<td>T.Chol (mg/dl)</td>
<td>152.78±21.81</td>
<td>150.44±18.97</td>
<td>155.67±26.41</td>
<td>0.218</td>
<td>0.833</td>
</tr>
<tr>
<td>T.G (mg/dl)</td>
<td>227.67±53.86</td>
<td>210.33±42.94</td>
<td>212.89±72.28</td>
<td>0.893</td>
<td>0.398</td>
</tr>
<tr>
<td>HDL (mg/dl)</td>
<td>35.56±4.56</td>
<td>34.33±1.73</td>
<td>34.44±4.03</td>
<td>0.816</td>
<td>0.438</td>
</tr>
<tr>
<td>LDL (mg/dl)</td>
<td>73.33±20.39</td>
<td>73.44±11.49</td>
<td>78.89±16.57</td>
<td>1.237</td>
<td>0.247</td>
</tr>
</tbody>
</table>

NB: Values in a row with different superscript are significant at p<0.05. Keys: BMI – Body mass index; T.Chol – Total cholesterol; T.G. – Total cholesterol; HDL – High density lipoprotein; LDL - Low density lipoprotein; n – Sample size

3.3. BMI and Lipid profile of cement factory workers with respect to age

Table 3 showed the BMI and lipid profile of cement factory workers with respect to age. The results obtained showed that the Height (m) of the subjects in age group 19–25 years, 26–30 years and 31–35 years was 1.64±0.07, 1.62±0.12 and 1.65±0.09, Weight (kg) was 63.64±4.71, 65.72±4.40 and 60.00±2.10, BMI (kg/m²) was 23.86±1.77, 25.14±3.53 and 23.72±1.80 respectively. Furthermore, the mean Total cholesterol (mg/dl) in age group 19–25 years, 26–30 years and 31–35 years was 155.09±21.40, 156.82±28.51 and 148.00±37.95, Triglyceride (mg/dl) was 195.18±13.49, 227.64±47.35 and 248.36±44.68, HDL (mg/dl) was 36.00±2.65, 33.82±3.19 and 36.00±3.16, while LDL (mg/dl) was 80.18±13.49, 75.91±24.06 and 92.00±1.05 respectively. There was significant difference (p<0.05) in Height, Triglyceride and LDL of the subjects with respect to age, while others were not significant (p>0.05).
**Table 3** BMI and Lipid profile of the subjects with respect to age

<table>
<thead>
<tr>
<th>Parameters</th>
<th>19–25 years Mean ± SD</th>
<th>26–30 years Mean ± SD</th>
<th>31–35 years Mean ± SD</th>
<th>F-value</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Height (m)</td>
<td>1.64±0.07&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.62±0.12&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.65±0.09&lt;sup&gt;b&lt;/sup&gt;</td>
<td>4.200</td>
<td>0.003</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>63.64±4.71&lt;sup&gt;a&lt;/sup&gt;</td>
<td>65.72±4.40&lt;sup&gt;a&lt;/sup&gt;</td>
<td>60.00±2.10&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.561</td>
<td>0.587</td>
</tr>
<tr>
<td>BMI (kg/m&lt;sup&gt;2&lt;/sup&gt;)</td>
<td>23.86±1.77&lt;sup&gt;b&lt;/sup&gt;</td>
<td>25.14±3.53&lt;sup&gt;b&lt;/sup&gt;</td>
<td>23.72±1.80&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1.802</td>
<td>0.105</td>
</tr>
<tr>
<td>T.Chol (mg/dl)</td>
<td>155.09±21.40&lt;sup&gt;c&lt;/sup&gt;</td>
<td>156.82±28.51&lt;sup&gt;c&lt;/sup&gt;</td>
<td>148.00±37.95&lt;sup&gt;c&lt;/sup&gt;</td>
<td>0.611</td>
<td>0.557</td>
</tr>
<tr>
<td>T.G (mg/dl)</td>
<td>195.18±50.73&lt;sup&gt;a&lt;/sup&gt;</td>
<td>227.64±47.35&lt;sup&gt;b&lt;/sup&gt;</td>
<td>248.36±44.68&lt;sup&gt;c&lt;/sup&gt;</td>
<td>8.258</td>
<td>0.000</td>
</tr>
<tr>
<td>HDL (mg/dl)</td>
<td>36.00±2.65&lt;sup&gt;d&lt;/sup&gt;</td>
<td>33.82±3.19&lt;sup&gt;d&lt;/sup&gt;</td>
<td>36.00±3.16&lt;sup&gt;d&lt;/sup&gt;</td>
<td>1.404</td>
<td>0.191</td>
</tr>
<tr>
<td>LDL (mg/dl)</td>
<td>80.18±13.49&lt;sup&gt;a&lt;/sup&gt;</td>
<td>75.91±24.06&lt;sup&gt;a&lt;/sup&gt;</td>
<td>92.00±1.05&lt;sup&gt;b&lt;/sup&gt;</td>
<td>2.940</td>
<td>0.016</td>
</tr>
</tbody>
</table>

**NB:** Values in a row with different superscript are significant at p<0.05. **Keys:** BMI – Body mass index; T.Chol – Total cholesterol; T.G – Total cholesterol; HDL – High density lipoprotein; LDL – Low density lipoprotein; n – Sample size

**Figure 1** Scattered plot showing Pearson Correlation between BMI and Total cholesterol of cement factory workers.

There was a positive correlation between BMI and Total cholesterol concentration in cement factory workers (Pearson correlation = 0.155) which was insignificant (p = 0.413).
Figure 2 Scattered plot showing Pearson Correlation between BMI and Triglyceride of cement factory workers.

There was a positive correlation between BMI and Triglyceride concentration in cement factory workers (Pearson correlation = 0.363) which was insignificant (p = 0.048).

Figure 3 Scattered plot showing Pearson Correlation between BMI and HDL of cement factory workers.

There was a negative correlation between BMI and HDL concentration in cement factory workers (Pearson correlation = -0.332) which was insignificant (p = 0.073).
There was a negative correlation between BMI and LDL concentration in cement factory workers (Pearson correlation = -0.141) which was insignificant (p = 0.458).

### 3.4. Relationship between BMI and Lipid profile of Cement Factory Workers using Pearson correlation

#### Table 4 Relationship between BMI and Lipid profile of Cement Factory Workers using Pearson correlation

<table>
<thead>
<tr>
<th></th>
<th>BMI</th>
<th>T. Chol</th>
<th>Triglyceride</th>
<th>HDL</th>
<th>LDL</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI</td>
<td>Pearson Correlation</td>
<td>0.155</td>
<td>0.363*</td>
<td>-0.332</td>
<td>-0.141</td>
</tr>
<tr>
<td>Sig. (2-tailed)</td>
<td>0.413</td>
<td>0.048</td>
<td>0.073</td>
<td>0.458</td>
<td></td>
</tr>
<tr>
<td>N</td>
<td>30</td>
<td>30</td>
<td>30</td>
<td>30</td>
<td></td>
</tr>
<tr>
<td>T. Chol</td>
<td>Pearson Correlation</td>
<td>0.155</td>
<td>0.244</td>
<td>0.140</td>
<td>0.792**</td>
</tr>
<tr>
<td>Sig. (2-tailed)</td>
<td>0.413</td>
<td>0.195</td>
<td>0.460</td>
<td>0.000</td>
<td></td>
</tr>
<tr>
<td>N</td>
<td>30</td>
<td>30</td>
<td>30</td>
<td>30</td>
<td></td>
</tr>
<tr>
<td>Triglyceride</td>
<td>Pearson Correlation</td>
<td>0.363*</td>
<td>0.244</td>
<td>-0.393*</td>
<td>0.024</td>
</tr>
<tr>
<td>Sig. (2-tailed)</td>
<td>0.048</td>
<td>0.195</td>
<td>0.032</td>
<td>0.899</td>
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<tr>
<td>HDL</td>
<td>Pearson Correlation</td>
<td>-0.332</td>
<td>0.140</td>
<td>-0.393*</td>
<td>0.398*</td>
</tr>
<tr>
<td>Sig. (2-tailed)</td>
<td>0.073</td>
<td>0.460</td>
<td>0.032</td>
<td>0.029</td>
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<tr>
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<tr>
<td>LDL</td>
<td>Pearson Correlation</td>
<td>-0.141</td>
<td>0.792**</td>
<td>0.024</td>
<td>0.398*</td>
</tr>
<tr>
<td>Sig. (2-tailed)</td>
<td>0.458</td>
<td>0.000</td>
<td>0.899</td>
<td>0.029</td>
<td></td>
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</table>

* Correlation is significant at the 0.05 level (2-tailed); ** Correlation is significant at the 0.01 level (2-tailed).
There was a negative correlation between BMI and LDL concentration in cement factory workers (Pearson correlation = -0.141) which was insignificant (p = 0.458). There was a negative correlation between BMI and HDL concentration in cement factory workers (Pearson correlation = -0.332) which was insignificant (p = 0.073). There was a positive correlation between BMI and Triglyceride concentration in cement factory workers (Pearson correlation = 0.363) which was insignificant (p = 0.048). There was a positive correlation between BMI and Total cholesterol concentration in cement factory workers (Pearson correlation = 0.555) which was significant (p<0.05) positive correlation between total cholesterol and LDL, and non-significant (p>0.05) positive correlation between total cholesterol and triglyceride, cholesterol and HDL. Triglyceride had significant (p<0.05) negative correlation with HDL and positive non-significant (p>0.05) positive correlation with LDL. Finally, HDL had positive significant (p<0.05) correlation with LDL.

4. Discussion

Cement dusts are basically made of calcium oxide, silicon oxide, aluminum oxide with little quantity of iron oxide, magnesium oxide, chromium, potassium, sodium and sulphur (Neboh et al., 2015). Exposure to cement dust has been implicated as a likely causative agent in various health problems (Meo et al., 2008). Cement dust exposure has also been associated with both increased oxidative stress and decreased anti-oxidant capacity, which in turn can promote lipid peroxidation and consequently dyslipidaemia (Aydin et al., 2010). This study was carried out to determine the lipid profile of cement factory workers in Ekpoma and environs.

Higher levels of total cholesterol, LDL cholesterol and triglycerides were observed in the cement workers compared to the controls in this study. Our findings are consistent with those of other studies (Aydin et al., 2010; Modhir et al., 2014; Bassey et al., 2017) who reported increases in total cholesterol, LDL-cholesterol and triglycerides in cement workers elsewhere. On the other hand, this study showed that there was a decrease in HDL cholesterol in cement factory workers compared with control group. This finding is in agreement with the earlier report (Aydin et al., 2010), but contrary to the report of Bassey et al. (2017) who reported higher HDL in cement factory workers compared with control group. The reason for the higher HDL-cholesterol in the cement workers is unclear but might be attributed to physically active lives of the cement workers arising from the demands of their occupations (Ademuyiwa et al., 2005).

Cement dust contains a mixture of metals and other toxic substances such as dioxins that interfere with lipid metabolism (Haro-Garcia et al., 2010). 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) which is a type of dioxin present in cement inhibits lipoprotein lipase (Haro-Garcia et al., 2010) which results in decreased clearance of VLDL and chylomicrons resulting in increased levels of triglycerides. This may be responsible for the increased levels of triglycerides observed in the cement workers in this study. Cement dust from Nigeria contains higher concentrations of cadmium, lead and Hg compared to those from the USA (Ogunbileje et al., 2013). Exposure to these heavy metals has been implicated in distinct pathological changes including dyslipidemia.

Chronic exposure to both high and low dose cadmium concentrations adversely affect lipid and lipoprotein profile via lipid peroxidation and increase in the activity of hydroxy-3-methylglutaryl-coenzyme A (HMG CoA) reductase, which is the rate-limiting enzyme in cholesterol synthesis (Samarghandian et al., 2015). Lead increases the activity of HMG CoA reductase and reduces the number/affinity of LDL receptors for cholesterol. Exposure to mercury causes lipid peroxidation (Kobal et al., 2004). These might account for the higher total and LDL-cholesterol values seen in the cement workers in this study. Environmental factors are being increasingly implicated in the pathogenesis of dyslipidemia. Elevated concentrations of total or LDL cholesterol in the blood are powerful risk factors for coronary disease. One of the causes of increased total lipid concentration appears to be disturbance of carbohydrate metabolism, due to probable cytotoxic effect on cells of the pancreas leading to relative deficiency of insulin (Pothu et al., 2019). In insulin deficiency, carbohydrates are not used as energy source and most of the energy is derived from fats. To meet the energy demands lipolysis occurs and the amount of free fatty acids in blood is increased, which we have not estimated in our study, resulting in increased serum total lipid concentration (Reaz et al., 2004). There are many studies which state that elevated levels of persistent organic pollutants such as pesticides are associated with increased levels of serum lipids which are a major risk factor for cardiovascular disease (Pothu et al., 2019). If this association appears to be causal, it may have significant effects on human health.

5. Conclusion

The study concludes that total cholesterol, triglyceride and LDL was higher among cement workers compared with control, while HDL was lower among cement workers. There was significant difference (p<0.05) in Height, Triglyceride and LDL of the subjects with respect to age, while others were not significant (p>0.05). There was no significant
difference (p>0.05) in the lipid profile of cement factory workers with respect to duration of work. There was a significant (p<0.05) positive correlation between total cholesterol and LDL, and non-significant (p>0.05) positive correlation between total cholesterol and triglyceride, cholesterol and HDL. Triglyceride had significant (p<0.05) negative correlation with HDL and positive non-significant (p>0.05) positive correlation with LDL. Finally, HDL had positive significant (p<0.05) correlation with LDL.

We recommend that long term exposure to cement dust be discouraged in other not to prevent possible cardiovascular diseases. Long term exposure to cement dust should be monitored, thus calling for collaboration between the health workers and cement factory management to ensure frequent monitoring of their workers' health. Further study is necessary to study and understand the underline mechanism of cement dust toxicity on the cardiovascular system.

Compliance with ethical standards

Acknowledgments

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Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of ethical approval

Ethical approval for the collection of samples was obtained from the Ethics and Review Committee, Ambrose Alli University, Ekpoma. Informed consent was sought from each subject who participated in the study before the collection of samples.

Statement of informed consent

The authors declare no conflicts of interest. The authors alone are responsible for the content and the writing of the paper.

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Authors' Contributions

The entire study procedure was conducted with the involvement of all authors.

References


