# Assessment Of Oxidative Stress, Haematological, Kidney And Liver Function Parameters Of Libyan Cement Factory Workers

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**Abstract:** Cement workers are exposed to different types of health hazards, which are risk factors in developing occupational diseases. The hypothesis of this study focuses on the opinion that cements worker impact on their health by pollution with cement dust and this affects the different tissues including liver and kidney. This study was carried out in exposed workers of the cement factory of Libya that has not yet been studied. This study measured the plasma oxidant, antioxidant status and haemopoietic, liver and kidney functions in workers occupationally exposed to cement dust in order to test the hypothesis that cement dust exposure may perturb these parameters. 21 volunteer cement plant workers and 30 volunteer office workers (control) with a mean age of 38.79 ± 4.68 and 39.40 ± 2.19 years respectively (Mean ± S.E) participated. The levels of P-MDA, P-ALT, P-AST, P-AlP, P-LDH activities, TLC count and total bilirubin level were significantly increased ($P<0.05$), while TEC, PLT count, Hb concentration, P-E, P-C, β-carotene levels, P-SOD, P-CAT, P-GST activities and the total protein, albumin and globulin were significantly decreased ($P<0.05$) compared with the unexposed group. The results presented in this study showed that cement workers are exposed to more oxidative stress compared to the control group. The present data showed that the exposure of humans to cement dust is capable of inducing free radicals, marked hazardous alterations in some enzymatic activities, liver functions and some biochemical parameters. To protect the health of their workers dust needs to be removed from the critical area of the factory and with the use of industrial masks.
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## 1. Introduction

The major pollution problems come from cement factories because of dust and particulate matter emitted at various steps of cement production (Ogunbileje et al., 2010). In an industrial setting, the most important routes of entry of cement dust chemicals into the body to produce adverse effects are inhalation and skin contact (Tewari et al., 1991; Oliver, 1980). Cement dust is a grey powder and is manufactured from contaminated clay and limestone and has a diameter ranging from 0.05 to 5.0 µm (Abrons et al., 1997; Kalacic, 1973). 60% - 70% calcium oxide (CaO), 17% - 25% silicon dioxide ($SiO_2$), 3% -5% aluminium oxide ($A1_2O_3$), chromium potassium, sodium sulphur and magnesium oxide with smaller amount of iron oxide are of primary importance in the cement industry (Scheidegger et al., 2001; Wang et al., 2001). These chemical compounds, especially aluminium and chromium are considered toxic. So, chronic exposure to these pollutants could increase per-oxidation of membrane lipids in different tissues resulting in neurotoxicity, renal failure and anaemia (Guo et al., 2004; Gupta et al., 2005; Ranjbar et al., 2008). Workers exposure to cement dust (silicates) has been reported to cause respiratory diseases, laryngeal cancer (Ghio et al., 1990), gastrointestinal tumours (Ghio et al., 1990; Ezeonu and Ezejiofo, 1999) and also augments free radical generation (Riley, 1994; Mengesha and Bekele, 1998; Froom et al., 2000). Occupational exposure to cement dust might cause toxic effects on vital organs such as respiratory, renal and liver (Bright et al., 1997; Costa and Klein, 2006; Goldoni et al., 2006) via generation of free radical especially reactive oxygen species (ROS) which lead to damaged membrane cells (Castranova, 2004). It is believed that the excess generation of free radicals or level index of antioxidant enzymes such as glutathione S-transferase (GST), catalase (CAT), superoxide dismutase (SOD) and non enzymatic antioxidant plasma vitamin C, E and β-carotene create a condition known as oxidative stress which plays an important role in various diseases (Krinsky and Denace, 1982; Frei et al., 1989; Grzelinska et al., 2007). A principal biological function of vitamin C is to scavenge oxygen free radicals and it is converted to dehydroascorbate. Vitamin E is a lipid-soluble, chain breaking antioxidant that protects cell membranes from peroxidative damage (Levine et al., 1999; Bendich et al., 1986). Vitamins C and E are synergistic antioxidants (Gitto et al., 2001; Wafers and Sies, 1988). The objective of this study was to measure the levels of plasma enzymatic and non-enzymatic antioxidants, and some biochemical

parameters in cement plant male workers, who had been exposed to cement dust for the last 10 years. Measurements of enzymatic and non-enzymatic antioxidants are key markers to study the extent of free radical damage in the vital organs (Rahman, 2007). The hypothesis of this study focuses on the opinion that cements worker impact on their health by pollution with cement dust and this affects the different tissues including liver and kidney. This study was carried out in exposed workers of the cement factory of Libya that has not yet been studied.

## 2. Material and Methods

In the present study, samples were collected from 21 volunteer male workers in a cement factory in Libya, who had been exposed to cement dust for 9.71 ± 1.72 years (Mean ± S.E). All subjects were given permission by the factory management and informed about the study, then asked to sign the permission form. Personal data such as disease status, and use of any medication, alcohol consumption and smoking habit were obtained for all subjects. Non-healthy workers were excluded. The population of the present study was distributed in five cement production units including: Mill, Furnaces, Packing, Precipitation, and Crusher, the workers who held jobs in these sections were selected to supply us with a real direct exposure to the cement dust (Alakija et al., 1990; Mwaiselage et al., 2005). Thirty healthy volunteers from the city near to the factory with similar age and health conditions were selected as the unexposed group (control group).

### 2.1. Reagents

The reagent kit for plasma parameters and enzymes was purchased from SENTINEL CH. (via principle Eugenio 5-20155 Milan, Italy). All other chemicals used in this experiment were of analytical grade.

### 2.2. Sampling and biochemical analyses

Blood samples from the study subjects were collected in appropriate sterile vials by venous arm after overnight fasting and placed immediately on ice. Heparin was used as an anticoagulant and plasma samples were obtained by centrifugation at 10000 Xg for 20 minutes at 4°C and stored at –20°C until measurements. QBC II machine (Becton Dickinson, Franklin Lakes, NJ, and USA) was used for analyses haematological parameters. The QBC II machine was calibrated before use. The plasma was used for determination of biochemical parameters, assay of antioxidant enzyme, enzymatic antioxidants and plasma malondialdehyde (P-MDA). P-MDA level is measured by the thiobarbituric acid reactive substances (TBARS) assay according to the colorimetrical method of Esterbauer and Cheeseman, 1990. Samples were read spectrophotometrically at 532 nm. The catalase enzyme (P-CAT; EC 1.11.1.6) activity in plasma was measured spectrophotometrically at 240 nm according to the method of Xu et al., 1997. Plasma superoxide dismutase (P-SOD; EC 1.15.1.1) was assayed according to Misra and Fridovich at 480 nm in a spectrophotometer (Misra and Fridovich, 1972). The activity of plasma glutathione S-transferase (P-GST; EC 2.5.1.18) was measured according to the method of Habig et al. 1974. Plasma Vitamin C (P-VC) was determined according to the procedure described by Erel et al. using dinitrophenylhydrazine (DNPH) (Erel et al., 1997). Plasma Vitamin E (P-VE) and β-Carotene were assayed according to the calorimetrical method of Baker & Frank and Oliver, respectively (Baker and Frank, 1968; Oliver, 1980). Liver function parameters were measured namely: plasma aspartate transaminase (P-AST; EC 2.6.1.1) and plasma alanine aspartate transaminase (P-ALT; EC 2.6.1.2) activities were determined with kits from SENTINEL CH. (via principle Eugenio 5–20155 MILAN, Italy). The activity of plasma lactate dehydrogenase (P-LDH; EC 1.1.1.27) was determined by the method of Martinek, 1972. Plasma alkaline phosphatase (P-AlP; EC 3.1.3.1) activity was measured at 405 nm by the formation of paranitrophenol from paranitrophenylphosphate as a substrate (Principato et al., 1985). Plasma creatinine and urea concentrations were analysed by the method of Patton & Crouch and Henry et al., respectively (Patton and Crouch, 1977; Henry et al., 1974). Plasma total bilirubin was measured using the method of Pearlman and Lee, 1974. The amount of protein in each sample was calculated by the bicinchoninic acid (BCA) based assay (Smith et al., 1985). Albumin concentration was analysed by the method of Doumas et al., 1971. Globulin concentration was determined as the difference between total protein and albumin.

## 3. Results

Demographic information and years employed of healthy subjects and exposed cement workers are presented in table 1. There was no statistically significant difference between the two groups in the mean of age, weight and years of employment (Table 1). The results indicated that cement dust caused a significant increase ($P<0.05$) in total leukocyte count (TLC) in exposed cement dust workers in comparison to the healthy control group. Further more there was a significant decrease ($P<0.05$) in total erythrocyte count (TEC), haemoglobin (Hb), packed cell volume (PCV) and platelet count (PLT) of exposed workers compared to control subjects (Table 2).

Table 1. Demographic characteristics of exposed cementust workers and healthy unexposed control

| Parameter | Healthy control (n = 30) | Exposed worker (n = 21) |
|---|---|---|
| Age (Year) | 39.40 ± 2.19 | 38.79 ± 4.68 |
| Weight (Kg) | 68.74 ± 7.28 | 67.62 ± 5.37 |
| Employee (Year) | 0.0 | 9.71 ± 1.72 |

Data are Mean ± S.E.; (n) the number of subjects.

Table 2. Haematological parameters of healthy unexposed control and exposed cement dust workers

| Parameter | Healthy control (n = 30) | Exposed worker (n = 21) |
|---|---|---|
| Hb (g/dl) | 13.8 ± 0.98[a] | 11.78 ± 0.81[b] |
| PCV (%) | 45.3 ± 1.80[a] | 33.1 ± 1.12[b] |
| TEC ($x10^{12}l^{-1}$) | 5.01 ± 0.36[a] | 3.96 ± 0.51[b] |
| TLC ($x10^{9}l^{-1}$) | 6.29 ± 0.72[b] | 9.89 ± 1.01[a] |
| PLT ($x10^{9}l^{-1}$) | 297 ±11.92[a] | 196 ±14.92[b] |

Data are Mean ± S.E; (n) the number of subjects. Mean values within a row not sharing a common superscript letters (a, b) were significantly different, $P<0.05$. Hb: hemoglobin; PCV: packed cells volume; TEC: erythrocyte counts; TLC: total leukocyte counts; PLT: platelets.

Liver function parameters of the study groups are shown in table 3. The liver functions of exposed cement dust workers resulted a significant increase (P<0.05) in plasma aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (AlP) and lactate dehydrogenase (LDH) when compared to the unexposed control (Table 3).

The effects of exposure to cement dust on enzymatic antioxidants are shown in table 3. Exposure to cement dust caused a significant increase (P<0.05) in the overall means of plasma malondialdehyde (p-MDA) concentration than those of the control group (Table 3). On the other hand plasma catalase (CAT), glutathione S-transferase (GST), superoxide dismutase (SOD) levels of cement workers were found to be decreased significantly (P<0.05) when compared to the control group (Table 3). Table 3 also represents the effect of exposure to cement dust on non-enzymatic antioxidants. Plasma vitamin E, vitamin C and β-carotene were found to be significantly lower (P<0.05) in cement plant workers compared with the unexposed control group. On the other hand, kidney tests were to be increased in cement plant workers but no statistically significant difference (P>0.05) was observed (Table 4). Data listed in table 5 showed that cement dust caused a significant decrease (P<0.05) in plasma concentration of total protein, albumin and globulin. Moreover, cement dust caused a significant increase (P<0.05) in plasma total bilirubin of cement plant workers compared with the unexposed control.

Table 3. Effects of cement dust on liver functions, enzymatic, non-enzymatic antioxidants and plasma malondialdehyde of cement factory workers and unexposed control

| Parameter | Healthy control (n = 30) | Exposed worker (n = 21) |
|---|---|---|
| MDA (nmol/ml) | 1.97 ± 0.04[b] | 4.61 ± 0.8[a] |
| AST (U/l) | 21.11 ± 3.21[b] | 44.20 ± 5.48[a] |
| ALT (U/l) | 23.91 ± 4.18[b] | 46.14 ± 7.02[a] |
| AlP (U/l) | 75.84 ± 5.14[b] | 99.12 ± 9.06[a] |
| LDH (U/l) | 351 ± 37.21[b] | 533 ± 51.03[a] |
| CAT (nmol/min/ml) | 3.23 ± 1.50[a] | 2.25 ± 1.01[b] |
| GST (nmol/min/ml) | 0.03 ± 0.005[a] | 0.01 ± 0.002[b] |
| SOD (U/ml) | 1.19 ± 0. 04[a] | 0.61 ± 0.03[b] |
| Vitamin C (mg/dl) | 1.57 ± 0.08[a] | 0.71 ± 0.04[b] |
| Vitamin E (mg/dl) | 2.09 ± 0.41[a] | 0.99 ± 0.08[b] |
| β-Carotene (µg/100ml) | 9.75 ± 1.14[a] | 6.83 ± 0.69[b] |

Data are Mean ± S.E; (n) the number of subjects. Mean values within a row not sharing a common superscript letters (a, b) were significantly different, $P<0.05$. MDA: malondialdehyde, AST: aspartate transaminase; ALT: alanin transaminase; AlP: alkaline phosphatise; LDH: lactate dehydrogenase; CAT: catalase; GST: Glutathione S-transferase; SOD: Superoxide dismutase.

Table 4. Effects of cement dust on kidney function of cement factory Workers and unexposed control

| Parameter | Healthy control (n = 30) | Exposed worker (n = 21) |
|---|---|---|
| Urea (mmol/l) | 4.89 ± 0.31 | 5.12 ± 0.98 |
| Creatinine (µmol/l) | 49.79 ± 7.02 | 50.11 ± 13.16 |

Data are Mean ± S.E; (n) the number of subjects.

Table 5. Effects of cement dust on plasma biochemistry of cement factory workers and unexposed control

| Parameter | Healthy control (n = 30) | Exposed worker (n = 21) |
|---|---|---|
| Total protein (g/dl) | 7.86 ± 0.61[a] | 6.10 ± 1.12[b] |
| Albumin (g/dl) | 4.93 ± 0.09[a] | 3.51 ± 0.14[b] |
| Globulin (g/dl) | 2.93 ± 0.06[a] | 2.59 ± 0.19[b] |
| Total bilirubin (mg/dl) | 0.78 ± 0.04[b] | 1.17 ± 0.16[a] |

Data are Mean ± S.E; (n) the number of subjects. Mean values within a row not sharing a common superscript letters (a, b) were significantly different, $P<0.05$.

## 4. Discussion

The present finding in this study shows that chronic occupational exposure to cement dust for

9.71 ± 1.72 years may have hematotoxicity and injurious effects on liver functions but not kidney functions. Thus haematological parameters may be sensitive biomarkers in assessing and monitoring the health of cement plant workers. The haemoglobin (Hb) concentration, erythrocyte counts (TEC), platelets (PLT) and packed cell volume (PCV) of the exposed workers were significantly decreased compared to those of the unexposed (Table 2). The reduced concentration of these parameters may not be due to nutritional deficiency as both groups were matched by socio-economic status. Besides, the exposed workers and unexposed control were not significantly different in weight, further arguing against nutritional deficiency as a basis for this observation (Table 1). The reduced Hb concentration, TEC, PLT and PCV counts and raised TLC or white cell counts probably suggests a reaction to irritant cement dust in body organs. The observed decreased Hb concentration could be due to either an increase in the rate at which Hb is destroyed or a decrease in the rate of Hb synthesis. I also reported a decrease in TEC count and Hb level (Table 1) in cement workers. This decreased level of TEC and Hb may be due to chronic exposure to cement dust. Cement bears calcium hydroxide as its important constituent (Ghio et al., 1990). It has been reported that chronic exposure to calcium hydroxide causes a decrease in TEC count and Hb (Proctor et al., 1988). The present study observations are in accordance with the above-reported results. The decrease in the level of Hb may also be related to the decrease in the level of plasma vitamin C, which was noted in our study. Vitamin C was reported as essential for the increased iron absorption that has been shown in many studies (Davidsson et al., 1998; Hunt et al., 1990). Moreover, reduction in the hemogram parameters brought about by exposure to cement dust could be due to decreased synthesis of red blood cells in bone marrow, the source of these cells, or biosynthesis of heme in bone marrow. Indeed, severe bone lesions have been seen in weaning pigs fed cement kiln dust as a way of boosting dietary calcium (Pond et al., 1982). According to Barger who found that hemolysis or increased activity of bone marrow could lead to impaired Hb synthesis, which resulted in macrocytic hypochromic anaemia (Barger, 2003). The present results on blood parameters agree with previous studies of Jude et al., which reported the first report of its type. They reported a reduction in TEC count, PCV and Hb concentration in exposed subjects although there was only a significant decrease in the PLT and TEC compared to the unexposed group (Jude et al., 2002). Although they reported no significant increase in TLC counts in the exposed workers, differential counts revealed an increase in lymphocyte count and a decrease in monocyte count, both of which were significant effects (Jude et al., 2002). Occupational exposure to silica containing cement has been reported to cause lung diseases (Chio et al., 1990) and also augments free radical production (Mengesha and Bekele, 1998; Froom et al., 2000; Halliwell and Gutteridge, 1986). In addition, the cement dust is a common air contaminant, though the toxicity associated with inhalation exposure to cement dust (Mwaiselage et al., 2005) may be causing excessive damage to erythrocytes which might reduce erythropoeisis, resulting in reduced TEC population and a similar effect to many pollutants (Mandal et al., 1996). The increase in TLC might be indicative of the activation of defence and immune system of the body (Yousef et al., 2003).

The findings of the present study suggest that the liver function parameters of cement plant workers may be adversely affected by cement dust exposure compared with those of the healthy control group (Table 3). An increase in the liver enzymes generally suggests an injury in the liver cell membrane (Pagana and Pagana, 2009). Several soluble enzymes of blood serum have been considered as indicators of the hepatic dysfunction and damage. Transaminases (AST and ALT) are important and critical enzymes in the biological processes. The increase in the activities of AST and ALT in plasma of exposed cement dust workers (Table 3) is mainly due to the leakage of these enzymes from the liver cytosol into the blood stream (Navarro et al., 1993). The activity of AST is significantly increased in such cases and escapes to the plasma from the injured hepatic cells. In addition, ALT level is of value also indicating the existence of liver diseases, as this enzyme is present in large quantities in the liver. It increases in serum when cellular degeneration or destruction occurs in this organ (Hassoun and Stohs, 1995). The present results, suggest that chronic exposure to cement dust has a deleterious effect on the haemopoetic system and cell membrane of liver. The reported liver cell membranes harmful effects seen in people occupationally exposed to cement dust further strengthens this idea (Jude et al., 2002). This finding is in agreement with other groups who also found increased ALT and AST activities in plasma cement workers (Aydin et al., 2004; Ezeonu and Ezejiofo, 1999).

Phosphatases (AlP) and dehydrogenases (LDH) are important and critical enzymes in biological processes, they are responsible for detoxification, metabolism and biosynthesis of energetic macromolecules for different essential functions. Any interference in these enzymes leads to biochemical impairment and lesions of the tissue and cellular

function (Khan et al., 2001). The increases in alkaline phosphatases (AlP) in plasma caused by exposure to cement dust (Table 3) are in accordance with the finding of Orman et al. in cement workers (Orman et al., 2005). In addition, Rahman et al. suggested that the increase in the activity of AlP in plasma might be due to the increased permeability of plasma membrane or cellular necrosis (Rahman et al., 2000). Also, the increase in plasma LDH activity of cement workers in the present study (Table 3) may be due to the hepatocellular necrosis leading to leakage of the enzymes into the blood stream (Wang and Zhai, 1988).

Oxidative stress is believed to occur when there is an imbalance in the biological oxidant to antioxidant ratio, and can result in oxidative damage to lipids, proteins, carbohydrates, and nucleic acids. In most cases, the abnormal generation of ROS, which can result in significant damage to cell structure, is considered an important signal of oxidative damage (Barzilai and Yamamoto, 2004). Organisms have unique systems for protecting themselves against the damaging effects of activated reactive oxygen species (ROS). For example, $O_2^-$, the parental form of intracellular ROS, is a very reactive molecule, but it can be converted to $H_2O_2$ by SOD, and then to oxygen and water by several enzymes including CAT and peroxiredoxin (Pi et al., 2010). Therefore, examining the change in activity of antioxidant enzymes such as SOD, CAT, and GST is considered as an effective method of denoting oxidative stress. More recently, the differential expression of the genes encoding these enzymes has been used to detect biological toxicity and/or to monitor the impact of chemical pollutants (Xu et al., 1997). The present study showed an increase in the level of MDA in the plasma of cement workers (Table 3), and this suggests that exposure to cement dust for long periods might cause membrane lipid peroxidation. The data indicates a decline in SOD levels of cement workers plasma might be due to the fact that exposure to air polluted by cement dust generates an increase in the production of reactive oxygen species especially superoxide anion. The superoxide radical produced is neutralised by SOD and the decrease in the activity of this antioxidant reveals that exposure to cement dust leads to increased oxidative stress (Aydin et al., 2004). The present study showed an increase in the level of MDA in the plasma of cement workers (Table 3), and this suggests that exposure to cement dust for long periods might cause membrane lipid peroxidation. Aydin et al. found that plasma MDA levels were determined to be much higher in cement-exposed workers (Aydin et al., 2004). Kamal and El-Khafif showed that plasma MDA levels in asbestos-exposed workers were significantly higher than in the control group (Kamal and El-khafif, 1992). The data indicates a decline in SOD levels of cement workers plasma (Table 3) might be due to the fact that exposure to air polluted by cement dust generates an increase in the production of reactive oxygen species especially superoxide anion. The superoxide radical produced is neutralised by SOD and the decrease in the activity of this antioxidant reveals that exposure to cement dust leads to increased oxidative stress (Khan et al., 2001).

The result further indicated that exposure to cement dust decreased CAT activity by 30% (Table 3). It is generally believed that hydrogen peroxide can be detoxified by CAT, which removes it when CAT is present at a high concentration (Casado et al., 1995). The reduction in the level of this enzyme may render the liver more susceptible to hydrogen peroxide induced oxidative stress. Moreover, single oxygen and peroxyl radicals can inhibit SOD and CAT activities. Enzymes that scavenge oxygen free radicals like CAT and SOD decreased by 50% upon pollutant exposure (Khan et al., 2005).

The exposure to cement dust caused a decrease in the activity of GST in the plasma of cement workers (Table 3). Cervello et al. suggested that GST enzymes catalyse the reaction via the thiol (-SH) group of glutathione (GST), thereby neutralising and rendering the products more water-soluble (Cervello et al., 1992). Also, glutathione and glutathione-related enzymes are involved in the metabolism and detoxification of cytotoxic and carcinogenic compounds as well as reactive oxygen species (Knapen et al., 1999). In this study, cement dust is associated with the increased plasma MDA levels (Table 3) and the reduced GST activity providing an oxidative link. A functional disturbance in the cell membrane structure is implicated in view of the ROS specific attacks on unsaturated lipids as suggested by the increased lipid peroxidation in cement workers. Evelo and Bos found significantly decreased GSH activity of rats, which are exposed to quartz after 8 days (Evelo and Bos, 1993). Afaq and Abidi found that chrysotile is an important commercial variety of asbestos caused oxidative stress by enhancing the production of hydrogen peroxide ($H_2O_2$) and thiobarbituric acid reactive substances (TBARS), depleting GSH and altering levels of GSH redox system enzymes (Afaq and Abidi, 1996). Glutathione acts as a cofactor with the enzyme GST to detoxify $H_2O_2$ and lipid peroxide in cell and tissue. Therefore, glutathione plays an important role in cellular protection against oxidative damage (Sastre et al., 1996; Lang et al., 1992). The decreased activity of SOD, CAT and GST activities found in cement workers in the present study supports the hypothesis

that cement dust leads to a greater oxidative burden and depletion of the antioxidant defence system. Generally, cells are equipped with endogenous defence comprising of both enzymatic and non-enzymatic antioxidants (vitamin E, C and β-carotene), tripeptides and others to safeguard the cells from probable oxidative injury. Still then, the cells suffer from oxidative assault when the antioxidant capabilities of the cells are inhibited by the heavy generation of ROS and its products resulting in the cells capacity to protect or to repair itself (Heffner and Repine, 1989). The result shows that vitamin E, C and β-carotene was decreased in cement workers (Table 3). The results showed significantly decreased plasma vitamin E, C and β-Carotene in cement workers than in the unexposed control population (Table 3). Deficiency of vitamin E is known to cause oxidant-induced injuries to the erythrocyte membrane (Bendich et al., 1986; Prakasam and Sethupathy, 2001). Decreased concentration of plasma vitamin E may be due to its increased utilisation in scavenging the oxygen radical or decreased plasma vitamin C concentration because vitamins C and E are synergistic antioxidants (Bendich et al., 1986; Mccay, 1985). β-Carotene, a precursor of vitamin A has been found to be an effective quencher of reactive oxygen species (Burton and Ingold, 1984). The apparent decrease in this non-enzymatic antioxidant observed in this study for the group exposed to cement dust reflects the fact that the groups exposed to cement dust suffer from oxidative injury. The present finding on depletion of antioxidant vitamins are in agreement with Oluwayernis, who reported that the level of vitamin C and β-Carotene were significantly decreased in albino rats exposed to cement dust for 14 days and 28 days respectively (Oluwayernis, 2012).

There was no significant difference in plasma urea and creatinine level between exposed workers and the unexposed control group (Table 4). The results suggest that the kidney may not be adversely affected by cement dust exposure.

Plasma total bilirubin concentrations were increased in cement workers (Table 5). Rana et al. reported that the increase in plasma bilirubin (hyper-bilirubenimia) may be a result of decreased liver uptake, conjugation or increased bilirubin production from hemolysis (Rana et al., 1996). Also, the elevation in plasma bilirubin concentration could be due to the onset of periportal necrosis. The present results also showed that the activities of AST, ALT and LDH were significantly increased in the plasma (Table 3) of the exposed cement dust group and this is an indication of damage of liver cell membrane.

The decline in plasma total protein due to exposed to cement dust was primarily due to a reduction in albumin and globulin fraction (Table 5). The reduction in plasma protein in workers exposed to environmental pollutants (cement dust) could be attributed to changes in protein and free amino acid metabolism and their synthesis in the liver (Oluwayernis, 2012). Additionally, the protein depression in the blood was also reported to be mainly due to excessive loss through nephrosis. It may also be due to reduced protein synthesis or increased proteolytic activity or degradation (Rana et al., 1996). Also, the observed decrease in plasma proteins (Table 5) could be attributed in part to the damaging effect of cement dust on liver cells as confirmed by the increase in the activities of plasma AST, ALT, AlP and LDH (Table 3).

## Conclusion

In conclusion, the present data showed that the exposure of humans to cement dust is capable of inducing free radicals, marked hazardous alterations in some enzymatic activities, liver functions and some biochemical parameters. To protect the health of their workers they should remove the dust from the critical area of the factory and use industrial masks. Furthermore, support for this conclusion comes from the study that there should be supplementation of cement workers with antioxidant vitamins such as ascorbic and μ-tocopherol, which can improve plasma antioxidant enzymes (Aydin et al., 2004).

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