Comparative study of changes in inflammatory markers on hours of exposure to cement dust

ABSTRACT

There has being some growing health concern on lives of people exposed to cement dust. This study was therefore aimed at assessing the effect of cement dust on inflammatory markers on cement loaders with different duration of hourly exposure. This work was a cross sectional study design conducted in Port Harcourt, Nigeria. 100 subjects were recruited using convenient sampling method. All 100 subjects were males cement workers and they were grouped into 3groups based on hourly duration of cement dust exposure; 1-5hours group, 6-10hours group and more than 10hours group. Group 1 (1-5hrs) had 27 subjects; Group 2 (6-10hrs) had 62 subjects while Group 3 (>10hrs) had 11 subjects. Sample was collected using venipuncture method into plain bottles for the analysis of C-Reactive protein, IL-1β and IL-10. ELISA machine was used for the assay of the study parameter. CRP levels were 5.42±4.72; 6.71±4.96 and 9.04±8.83 in group 1, 2 and 3 respectively and P-value=0.1771 which implies there was no significance difference. IL-10 levels were 15.72±12.58; 12.23±10.12 and 11.83±8.88 in group 1, 2 and 3 respectively and P-value=0.3421 which implies there was no significance difference. IL-1β levels were 3.81±1.02; 3.63±1.36 and 3.91±1.21 in group 1, 2 and 3 respectively and P-value=0.7031 which implies there was no significance difference. The study has shown that hourly changes in exposure to cement dust doesn't have a impact on inflammatory markers among cement workers in Port Harcourt.

Keywords: *C-reactive protein, interleukin 10, interleutin 1β, inflammation, cement dust*

1.0 INTRODUCTION

Urbanization is the quest of every under-developed area or community but this desire could become detrimental to the health of the people when the toxic products generated from industrial activities are not well managed. According to certain researchers, there is a strong relationship between environmental toxicity and human toxicity such that increase in environmental pollution results in increased toxicant in human which may affect normal physiological processes [1,2]. One key toxicant of industrial activities is heavy metal [3,4,5]. Cement is an adhesive and cohesive substance which acts as glue for individual elements used in construction works [6]. It is a very fine particulate matter made from the blending and mixing limestone with quartz, or other sources of silica, iron ore and other additives such as gypsum in a chemical process at high temperature [7]. The end product of this mixture is a very fine particulate grayish substance called cement. Cement has application in the construction of bridges, houses and other types of buildings, concrete materials and other types of structures. The main ingredients present in cements are Calcium, Silicon, Aluminum, and iron which is sourced from limestone, sand, Bauxite and Iron Ore respectively [7]. The manufacturing process of cement has been found to generate and emit large amount of dusts which studies have revealed to pose health hazards to those exposed to them often [8]. As a result, the cement industry has been listed as one of the major sources of air pollution because of dust and particulate matter emitted at various steps of cement production [9].

Studies around the world have linked certain diseases among cement workers, to exposures to cement dusts, ranging from respiratory symptoms to increased dynamic lung malfunction,

chronic bronchitis, emphysema, asthma and radiographic abnormalities of the lungs, although many of these studies have been laden with different limitations [10, 11]. Inflammatory response to occupational exposures to silica has been reported to be observed in specific organs, such as lungs, skin and the liver and if persistent may progress to fibrosis, granulomatous diseases and even cancer according to report of Aminian *et al*. [12]. John and Olubayo, [13] found result suggestive of nephrotoxic effects of cement in exposed group in their study. This is consistent with a study which recorded that exposure to silica results to silica nephrotoxicity [14]. Other authors reported remarkable nephrotoxic effects of silica exposure in separate studies [15]; [16] and [17]. Also, Colpan *et al.* [18] reported that silicon has a dose related harmful effect on the renal structure. Chronic exposures to aluminum has been reported to have the possibility to elevate lipid peroxidation in different tissues which could lead to anaemia, neurotoxicity and renal failure [19]. Hexavalent chromium Cr (VI), a derivative of Chromium, one of the ingredients in cement, is known to be first class human carcinogens according to International Agency for Research on Cancer [20]. Each ingredient in cement has been reported to cause one or more health challenge to those exposed to cement dust overtime. This study evaluated the effect of working hours on some total antioxidant status, inflammatory and cancers parameters among cement loaders in Port Harcourt. The connection that particle exposure has with inflammatory parameters signifies an elevated danger of cardiovascular disease in high dust exposed workers [21]. While the report of the health defects of exposure to cement dust is true when considered under different parameters, the duration of working hours per day has no reported significant impact on the levels of the parameters studied.

2.0 MATERIALS AND METHOD

2.1 Study Design

This study employed a cross sectional study design. 100 subjects were recruited using convenient sampling method. All 100 subjects were males cement workers and they were grouped into 3groups based on hourly duration of cement dust exposure; 1-5hours group, 6-10hours group and more than 10hours group. Group 1 (1-5hrs) had 27 subjects; Group 2 (6-10hrs) had 62 subjects while Group 3 (>10hrs) had 11 subjects.

2.2 Study Area

The study was conducted in Port Harcourt metropolis, Rivers State, Nigeria. Port Harcourt is the capital and biggest city of Rivers State and it is one of the states that make up the south-south geopolitical region in Nigeria.

2.3 Eligibility

Inclusion Criteria

Subjects must be exposure to cement dust for at least 3months. Healthy subjects between 20 to 60 years of age were included. If criteria for inclusion were met, subjects were only included if they gave their consent.

Exclusion Criteria

Subjects having previous exposure or concurrent exposure to other occupational toxicants were excluded. Subjects with underlining medical illness especially inflammatory diseases were not included. Subjects not working at cement sites were not included.

With the aid of questionnaire and interview, all participating cement loaders were interviewed by trained interviewers. All participants went through medical assessment to rule out the presence of diseases like asthma, diabetes, hypertension, anemia, cancer, infections or those who have recently had blood transfusion, thyroid and heart problems. Participants with diseases, drug therapy and alcohol, antioxidants, exposure to deadly substances or radiation therapy were not included in the study.

2.4 Informed Consent and Ethical Clearance

Ethical clearance to conduct the research was obtained from Rivers State Health Research Ethics Committee. Informed consent was given by individuals before recruitment into the study.

2.5 Sample Collection, Transportation, Processing and Preservation

In line with the procedure given by Cheesbrough, [23], blood samples were collected using venipuncture technique. 4ml of the venous blood was lastly drawn into plain vacutainer bottles for the evaluation of C-Reactive protein, IL-1β, and IL-10. The blood samples were allowed to clot and then centrifuged. The serum was obtained and transferred into a new sterile plain sample bottle and stored at freezing temperature prior to the analysis of C-Reactive protein, IL-1β, and IL-10. To the point of analysis, all drawn samples were conveyed via cold chain (ice packs/crushed ice in air tight and sealed thermo-container).

2.6 Data Collection

Data collection was done by way of an interviewer-administered self-structured questionnaire, to determine period of exposure. Information on general health and history of past disease(s) was obtained by trained health professional.

2.7 Sampling Technique

Technique for sampling was simple random where every subject was given same chance for selection.

2.8 Sample Analyses

The following parameters; C-Reactive protein, IL-1β, and IL-10 were assayed with the use of ELISA machine

Determination of Human C - reactive protein (CRP)

Assay for C-Reactive Protein (CRP) was performed with the use of Elabscience C-Reactive Protein (CRP) ELISA Kits manufactured by Elabscience Biotechnology Co Ltd, Inc., , USA.

Principle:

The ELISA kits used the sandwich-ELISA principle. The micro ELISA plate provided in this kit had been pre-coated with an antibody specific to Human. Samples (or Standards) are added to the micro ELISA plate wells and combined with the specific antibody. Then a biotinylated detection antibody specific for Human CRP and avidin-Horseradish Peroxidase (HRP) conjugate are added successively to each micro plate well and incubated. Free components are washed away. The substrate solution is added to each well. Onlt those wells that contain Human CRP, biotinylated detection antibody and Avidin-HRP conjugate will appear blue in color. The enzyme- substrate reaction is terminated by the addition of stop solution and the color turns yellow. The optical density (OD) is measured spectrophotometrically at a wavelength of

450±nm. The OD value is proportional to the concentration of Human CRP. You can calculate the concentration of Human NSE in the samples by comparing the OD of the samples to the standard curve.

Assay Procedure for C - reactive protein Test

The ELISA wells were determined for diluted standard, blank and sample into the appropriate wells (It was recommended that all samples and standards be assayed in duplicate). The plate was covered with the sealer provided in the kit. It was incubated for 90 minuites at 37^0C. The solutions were addedto the bottom of the micro plate well. Touching the inner wall was strictly avoided as that could result to foaming. From each well, the liquid was decanted. Washing was delayed for awhile and 100µL of Biotynylated detection Antibody working solution was immediately added to each well. The plate was then covered with a new sealer and incubated for 1 hour at 37^0C. From each well, the solution was decanted, 350µL of wash buffer was added to each well, soaked for an hour and the solution was aspirated or decanted from each well and was patted dry on an absorbent paper. These wash steps were repeated 3 times. To achieve these steps, a microplate washer could be used. Instantly, the test strips were used after the wash step. The wells were not allowed to get dry. 100µL of HRP Conjugate working solution was added to each well, the plate was covered with a brand new sealer and incubated for 30 minuites at 37^0C. The solution was decanted from each well; the washing process was repeated for 5 minuites as it was done in step 3 then 90µL of the substrate reagent was added to each well. The plate was covered with a new sealer and incubated at 37^0C for 15 minuites. Protecting the plate from light was very important as the reaction period could be reduced or extended based on color change. This should not have been for more than 30 minuites. Prior to the OD measure, the Microplate reader was preheated for 15 minuites. To each well, the addition of stop solution was done in the same order that the substrate solution was. Instantly, with a micro-plate reader set to 450nm, the optical density (OD value) of each well was determined.

Determination of Human IL-1β (Interleukin 1 Beta) Test
Principle

The ELISA kits use the sandwich-ELISA principle. The provided micro ELISA plate in this kit had already been pre-coated with Human IL-1β (Interleukin 1 Beta) specific antibody. Samples (or Standards) are then added to the micro ELISA plate wells and combined with the specific antibody. Then a biotinylated detection antibody specific for Human IL-1β (Interleukin 1 Beta) and avidin-Horseradish Peroxidase (HRP) conjugate are added successively to each micro plate well and incubated. Free components are washed away. The substrate solution is added to each well. Onlt those wells that contain Human IL-1β (Interleukin 1 Beta), biotinylated detection antibody and Avidin-HRP conjugate will appear blue in color. The enzyme- substrate reaction is terminated by the addition of stop solution and the color turns yellow. The optical density (OD) is measured spectrophotometrically at a wavelength of 450nm. The OD value is proportional to the concentration of Human NSE. You can calculate the concentration of Human IL-1β (Interleukin 1 Beta) in the samples by comparing the OD of the samples to the standard curve.

Procedure for Human IL-1β (Interleukin 1 Beta) Test

The ELISA wells were determined for diluted standard, blank and sample into the appropriate wells (It was recommended that all samples and standards be assayed in duplicate). The plate was covered with the sealer provided in the kit. It was incubated for 90 minutes at 37^0C. The solutions were then added to the bottom of the micro plate well. Contact with the inner wall was strictly avoided as that could result to foaming. From each well, the liquid was decanted. Washing was delayed for a while and 100µL of Biotynylated detection Ab working solution was immediately added to each well. The plate was the covered with a new sealer and incubated for 1 hour at 37^0C. From each well, the solution was decanted, 350µL of wash buffer was added to each well, soaked for an hour and the solution was aspirated or decanted from each well and was patted dry on an absorbent paper. These wash steps were repeated 3 times. To achieve these steps, a microplate washer could be used. Immediately, the test strips were used after the wash step. The wells were not allowed to dry up and 100µL of HRP Conjugate working solution was added to each well, the plate was covered with a brand new sealer and incubated for 30 minuites at 37^0C. The solution was decanted from each well; the washing process was repeated for 5 minuites as it was done in step 3 and after that, 90µL of the substrate reagent was added to each well. The plate was covered with a new sealer and incubated at 37^0C for 15 minuites. Protecting the plate from light was vital as the reaction period could be reduced or extended based on color change. This should not have been for more than 30 minuites. Prior to the OD measure, the Microplate reader was then preheated for 15 minuites. To each well, the addition of stop solution was done in the same order that the substrate solution was and straight away, with a micro-plate reader set to 450nm, the optical density (OD value) of each well was determined.

Determination of Human IL-10 (Interleukin 10) Test

Principle of Human IL-10 (Interleukin 10) Test

The ELISA kits use the sandwich-ELISA principle. The provided micro ELISA plate in this kit had already been pre-coated with Human IL-10 (Interleukin 10) specific antibody. Samples (or Standards) are then added to the micro ELISA plate wells and combined with the specific antibody. Then a biotinylated detection antibody specific for Human IL-10 (Interleukin 10) and avidin-Horseradish Peroxidase (HRP) conjugate are added successively to each micro plate well and incubated. Free components are washed away. The substrate solution is added to each well. Onlt those wells that contain Human IL-10 (Interleukin 10), biotinylated detection antibody and Avidin-HRP conjugate will appear blue in color. The enzyme- substrate reaction is terminated by the addition of stop solution and the color turns yellow. The optical density (OD) is measured spectrophotometrically at a wavelength of 450nm. The OD value is proportional to the concentration of Human NSE. You can calculate the concentration of Human IL-10 (Interleukin 10) in the samples by comparing the OD of the samples to the standard curve.

Procedure for Human IL-10 (Interleukin 10) Test

The ELISA wells for human IL-10 test were determined for diluted standard, blank and sample into the appropriate wells (It was recommended that all samples and standards be assayed in duplicate). The plate was covered with the sealer provided in the kit. It was incubated for 90 minuites at 37^0C. The solutions were rightly added to the bottom of the micro plate well. Tampering with the inner wall was strictly avoided as that could result to foaming. From each well, the liquid was decanted. Washing was delayed for awhile and 100µL of Biotynylated detection antibody working solution was immediately added to each well. The plate was then covered with a new sealer and incubated for 1 hour at 37^0C. From each well, the solution was

decanted, 350µL of wash buffer was added to each well, soaked for an hour and the solution was aspirated or decanted from each well and was patted dry on an absorbent paper. These wash steps were repeated 3 times. To achieve these steps, a microplate washer could be used.At once, the test strips were used after the wash step. The wells were not allowed to get dry and then 100µL of HRP Conjugate working solution was added to each well, the plate was covered with a brand new sealer and incubated for 30 minuites at 37^0C. The solution was decanted from each well; the washing process was repeated for 5 minuites as it was done in step 3. At this point, 90µL of the substrate reagent was added to each well. The plate was covered with a new sealer and incubated at 37^0C for 15 minuites. Guarding the plate from light was very imperative as the reaction period could be reduced or extended based on color change. This should not have been for more than 30 minuites. Prior to the OD measure, the Microplate reader was preheated for 15 minuites.

To each well, the adding up of stop solution was done in the same order that the substrate solution was. Instantaneously, with a micro-plate reader set to 450nm, the optical density (OD value) of each well was determined.

2.9 Statistical Analysis

Data generated from the study was analyzed for descriptive statistics (mean and standard deviation) and ANOVA. P-values ≤0.05 were considered significant.

3.0 RESULTS

Table 1.0: Effect of Working Hours on Inflammatory Markers on Exposed Subjects

Subjects	Parameters		
Working Hours (hr)	CRP (ng/mL)	IL-10 (pg/mL)	IL-1β (pg/mL)
1-5 (n=27)	5.42 ±4.72	15.72 ±12.58	3.81 ±1.02
6-10 (n=62)	6.71 ±4.96	12.23 ±10.12	3.63 ±1.36
>10 (n=11)	9.04 ±8.83	11.83 ±8.88	3.91 ±1.21
F-value	1.762	1.085	0.3535
P-value	0.1771	0.3421	0.7031
Remark	NS	NS	NS

Key: CRP- C-reactive protein, IL-10- intrleukin 10, and IL-1β- interleukin 1 beta, and ns= not significant at p>0.05

In Table 1, CRP, IL-10, IL-1β, levels were compared among working hours classifications: 1-5hrs; 6-10hrs; >10hrs. TAS level among the classes was not significantly different (p=0.3304) but has the mean value of 2.02 ±0.40; 2.16 ±0.41 and 2.11 ±0.41 respectively. Mean value for CRP level among the classes were 5.42 ±4.72; 6.71 ±4.96 and 9.04 ±8.83 respectively but were

statistically not significant (p=0.1771). IL-10 level among the classes was not significantly different (p=0.3421) but had the mean value of 15.72 ±12.58; 12.23 ±10.12 and 11.83 ±8.88 respectively. Mean value for IL-1β level among the classes were 3.81 ±1.02; 3.63 ±1.36 and 3.91 ±1.21 but were not significantly different (p=0.7031).

4.0 DISCUSSION

Constituents of cement such as silica-alumina have been reported to cause inflammatory reactions [24]. This finding agrees with the work of Rehabor *et al.* [25]. Free radical development by silica activities provokes oxidative stress. The silica gets to an alveolar macrophage and phagocytosis then takes place by the macrophage. This can bring about the destruction of the membrane of lysosomes. It is all as a result of communication with hydrogen ion and crystalline silica right in the cell membranes [22].

Inflammation is indicated by a high level of CRP in the blood. It can be influenced by a series of things, ranging from infection to malignancy. C-reactive protein (CRP) is a kind of acute-phase protein that is produced by the liver in response to inflammatory cytokines. CRP levels that are exceptionally high have been linked to a variety of diseases, inflammatory illnesses, certain malignancies, and problems affecting the lungs or pancreas [26]. High CRP levels can however signal inflammation in the heart's arteries, which can lead to an increased risk. C-reactive protein has been implicated in allergic skin diseases due to cement dust [27]. In this study, hourly exposure of cement dust on workers did not show any significant impact on C-reactive protein levels, P>0.05.

The concentration of IL–6 get elevated for smoking and non-smoking workers exposed to cement dust (Hilt *et al.,* 2002). According to their study, it was concluded that there was a constructive correlation in IL-6 values and exposure. In another study, the exposed subjects had significantly higher levels of IL-1β. IL-1β is a cytokine that mediates inflammatory response [28]. The finding from this study showed that there was no significant difference of IL-1β level among the groups of hourly exposure of cement dust. P>0.05.

IL-10 is a cytokine that exerts immunoregulatory effects. These immunoregulatory effects are broad and lead to attenuation of the expression of pro-inflammatory cytokine. In other words, IL-10 is predominantly anti-inflammatory cytokine [29]. A study showed significantly reduced levels of IL-10 which is an indication of reduced anti-inflammatory condition of the exposed subjects. The finding agreed with the work of Fell *et al.* [8] who reported a similar finding in Norwegian cement production workers. From this study, IL-10 values were not significantly difference based on hourly exposure to cement dust. P>0.05.

Generally, markers of inflammation studied in this work did not show any significant change following differences in hours of exposure to cement dust. Therefore, hourly exposure to cement dust may not have impact on inflammatory markers of cement workers. Since most illnesses related to cement dust exposure occur over a long period of exposure, assessment of the effect cement dust on higher timeframes may provide clearer view on the impact of the dust on human health.

CONCLUSION

This study evaluated some inflammatory markers among cement loaders in Port Harcourt, Nigeria. The study revealed that hourly exposure to cement dust does not have any impact on inflammatory markers among cement workers.

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