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

ABSTRACT

Background: There has been a growing concern about the health of people exposed to cement dust.
Objective: This study was aimed at assessing the effect of cement dust on inflammatory markers on cement loaders with different duration of hourly exposure.
Methods: This is a cross-sectional study design conducted in Port Harcourt, Nigeria with 100 male cement workers grouped into 3 groups based on the hourly duration of cement dust exposure. Group 1 (27 subjects) was exposed to 1-5 daily hours; Group 2 (62 subjects), was exposed to 6-10 daily hours, and Group C (11 subjects), was exposed to more than 10 daily hours. The ELISA analysis of C-Reactive protein (CRP), IL-1β, and IL-10 was performed. The data generated were analyzed for mean and standard deviation by ANOVA.
Results: The CRP levels were 5.42 ± 4.72; 6.71 ± 4.96 and 9.04 ± 8.83 in groups 1, 2, and 3 respectively. The p-value was 0.1771 implying no significant difference. The IL-10 levels were 15.72 ± 12.58; 12.23 ± 10.12 and 11.83 ± 8.88 in groups 1, 2, and 3 respectively. The p-value was 0.3421 implying no significant difference. The IL-1β levels were 3.81 ± 1.02; 3.63 ± 1.36 and 3.91 ± 1.21 in groups 1, 2, and 3 respectively. The p-value was 0.7031 implying no significant difference.
Conclusion: The study has shown that hourly changes in exposure to cement dust don't have an impact on the studied inflammatory markers among cement workers in Port Harcourt.

Keywords: *C-reactive protein, interleukin 10, interleukin 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 an increase in environmental pollution results in increased toxicants in humans which may affect normal physiological processes [1,2]. Heavy metals are key toxicants of industrial activities [3,4,5]. Cement is an adhesive and cohesive substance that acts as glue for individual elements used in construction works [6]. It is a very fine particulate matter made from the blending and mixing of limestone with quartz, or other sources of silica, iron ore, and additives such as gypsum in a chemical process conducted at high temperature [7]. The end product of this mixture is a very fine particulate grayish substance called cement. Cement has applications in the construction of bridges, houses, and other types of buildings concrete structures. The main ingredients present in cement are calcium, silicon, aluminum, and iron which are sourced from limestone, sand, bauxite, and iron ore respectively [7]. The manufacturing process of cement has been found to generate and emit a large amount of dust 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 exposure to cement dust, 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 performed 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. If persistent may progress to fibrosis, granulomatous diseases, and even cancer according to a report by Aminian *et al*. [12]. John and Olubayo, [13] found a result suggestive of the nephrotoxic effects of cement in an exposed group in their study. This is consistent with a study that recorded that exposure to silica results in 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 exposure to aluminum has been reported to have the possibility to elevate lipid peroxidation in different tissues which could lead to anemia, neurotoxicity, and renal failure [19]. Hexavalent chromium Cr (VI), a derivative of Chromium, one of the ingredients in cement, is known to be a first-class human carcinogen according to International Agency for Research on Cancer [20]. Each ingredient in cement has been reported to cause one or more health challenges to those exposed to cement dust over time. This study evaluated the effect of working hours on some total antioxidant status, inflammatory, and cancer 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 is a cross-sectional study design conducted with 100 male cement workers grouped into 3 groups based on the hourly duration of cement dust exposure. Group 1 (27 subjects) was exposed to 1-5 daily hours; Group 2 (62 subjects), was exposed to 6-10 daily hours, and Group C (11 subjects), was exposed to more than 10 daily hours.

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 exposed 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 illnesses especially inflammatory diseases were not included. Subjects not working at cement sites were not included.

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

2.4 Data Collection

Data collection was done by way of an interviewer-administered self-structured questionnaire, to determine the period of exposure. Information on general health and history of the past disease(s) was obtained by a trained health professional. The technique for sampling was simple random where every subject was given the same chance for selection.

2.5 Sample Collection, Transportation, Processing, and Preservation

In line with the procedure given by Cheesbrough, [23], blood samples were collected using the venipuncture technique. Venous blood (4mL) 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 before 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 Sample Analyses

The following parameters; C-Reactive protein (CRP), IL-1β, and IL-10 were assayed with the use of the Elabscience C-Reactive Protein (CRP) ELISA Kits manufactured by Elabscience Biotechnology Co Ltd, Inc., USA. 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 each biomarker assay. Samples (or standards) were added to the micro ELISA plate wells and combined with the specific antibody. Then a biotinylated detection antibody specific for the studied biomarker and avidin-Horseradish Peroxidase (HRP) conjugate were added successively to each micro plate well and incubated. Free components were washed away. The substrate solution was added to each well. Only those wells that contained the specific biomarker, biotinylated detection antibodies, and avidin-HRP conjugate appear in blue. The enzyme-substrate reaction was terminated by the addition of stop solution and the color turned yellow. The optical density (OD) was measured spectrophotometrically at a wavelength of 450 nm. The OD value was proportional to the concentration of the studied biomarker. The concentration of the biomarker in the samples was calculated by comparing the OD of the samples to the standard curve. The ELISA wells were determined for diluted standard, blank and sample into the appropriate wells (all samples and standards were assayed in duplicate). The plate was covered with the sealer provided in the kit. It was incubated for 90 minutes at 37 ^{0}C. The solutions were added to the bottom of the microplate well. Touching the inner wall was strictly avoided as that could result in foaming. From each well, the liquid was decanted. Washing was delayed for a while and 100 μL of biotinylated 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 ^{0}C. From each well, the solution was decanted, 350 μL 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 was 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, and the plate was covered with a brand new sealer and incubated for 30 minutes at 37 ^{0}C. The solution was decanted from each well; the washing process was repeated for 5 minutes and then 90 µL of the substrate reagent was added to each well. The plate was covered with a new sealer and incubated at 37 ^{0}C for 15 minutes. Protection of the plate was done to prevent the light to interfere with the reaction period. Before the OD measure, the microplate reader was preheated for 15 minutes. To each well, the addition of the stop solution was done in the same order that the substrate solution was. Instantly, with a microplate reader set to 450nm, the optical density (OD value) of each well was determined.

2.7 Statistical Analysis

Data generated from the study were analyzed for descriptive statistics (mean and standard deviation) by ANOVA. The 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

CRP = C-reactive protein, IL-10 = interleukin 10, and IL-1β = interleukin 1 beta, and NS = not significant at p-value > 0.05

The CRP, IL-10, and IL-1β, levels were compared among working hours classifications: 1-5hrs; 6-10hrs; >10hrs. The mean value for CRP levels among the classes were 5.42 ±4.72; 6.71 ±4.96 and 9.04 ±8.83 respectively but were statistically not significant (p-value = 0.1771). IL-10 level among the classes was not significantly different (p-value = 0.3421) but had a mean value of 15.72 ±12.58; 12.23 ±10.12 and 11.83 ±8.88 respectively. The mean value for IL-1β levels among the classes were 3.81 ±1.02; 3.63 ±1.36 and 3.91 ±1.21 but were not significantly different (p-value =0.7031). See table 1.

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 a result of communication with hydrogen ions 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. 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. CRP has been implicated in allergic skin diseases due to cement dust [27]. In this study, hourly exposure to cement dust on workers did not show any significant impact on PCR levels (p-value > 0.05).

The concentration of IL–6 gets elevated for smoking and non-smoking workers exposed to cement dust. According to their study, it was concluded that there was a constructive correlation between 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 in IL-1β levels among the groups of hourly exposure to 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 a predominantly anti-inflammatory cytokine [29]. A study showed significantly reduced levels of IL-10 which is an indication of the 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 different based on hourly exposure to cement dust (p-value > 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 an impact on the inflammatory markers of cement workers. Since most illnesses related to cement dust exposure occur over a long period of exposure, assessment of the effect of cement dust on higher timeframes may provide a clearer view of 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 the amount of time of exposure to cement dust did not result in significant differences in the inflammatory markers among cement workers.

Ethical Approval and Consent

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.

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