



Research Journal of  
**Environmental  
Toxicology**

ISSN 1819-3420



Academic  
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## Variation in Exposure to Cement Dust in Relation to Distance from Cement Company

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### ABSTRACT

The variation in the degrees of exposure to elements in cement dust and health implications arising from working or living within the vicinity of a cement company was monitored using forty albino rats aged between four and five weeks old. The albino rats were exposed to cement dust for one hundred and eighty days at three selected different locations around the vicinity of a Portland cement company in Ewekoro, Ogun state, South-Western Nigeria. The results of the study revealed that the chemical elements associated with cement dust were detected in the lungs of the exposed rats and the amounts increased with closeness to the Cement Company and length of exposure. It was also observed that the exposed and un-exposed (Control) rats gained considerable weights during the period of the exposure and furthermore, there was a significant difference ( $p < 0.05$ ) between the mean weight of the rats placed at 250 m from the cement company and the control. There was a direct linear association between the mean weights of the rats and all the chemical elements after 180 days. Atomic absorption spectroscopy of the lung tissues of the exposed rats showed significant levels of calcium, silicon, aluminum, chromium and lead compared to the un-exposed rats ( $p < 0.05$ ). The histopathology study of the lung tissues of the exposed rats showed abnormal alveolar architecture, disrupted bronchus, damaged bronchioles, degenerated epithelium lining, weak respiratory connective tissues, inflammations and blue-black pigments. Some of the exposed rats died before the end of the exposure. The results of the investigation highlight the hazards of prolonged exposure to cement dust and underscore the need for urgent action for the protection of animals and plants.

**Key words:** Pollutants, cement dust, albino rats, histopathology, atomic absorption spectroscopy

### INTRODUCTION

Air Pollution is a significant factor in morbidity and mortality within industrial areas. Hazardous substances are distributed widely in ecosystem due to diverse human activities such as energy usage, agriculture, urbanization, mining, industrialization and on few cases, high demand of infrastructure due to population increase (Jude *et al.*, 2002; Lori, 1996). Industrialization which is the focus point of this research, large amount of waste substances or pollutants are produced from chemicals, toxic wastes, effluents and radioactive wastes (Paivoke, 2002). Hazardous wastes are generated by nearly every industry; even those industries that themselves generate few hazardous wastes nonetheless use products from hazardous waste generating industries. Cement production is one of the sources of air pollution and the main impacts of the cement activity to the environment are the broadcast of dusts and gases (Bilen, 2010).

Portland cement is the most common type of cement in general use around the world because it is a basic ingredient of concrete, mortar, stucco and most non-specialty grout (Davidovits, 1994). Portland cement is made up of four main compounds; calcium-silicate ( $3\text{CaO}\cdot\text{SiO}_2$ ), di-calcium silicate ( $2\text{CaO}\cdot\text{SiO}_2$ ), tri-calcium aluminate ( $3\text{CaO}\cdot\text{Al}_2\text{O}_3$ ) and tetra-calcium aluminoferrite ( $4\text{CaO}\cdot\text{Al}_2\text{O}_3\cdot\text{Fe}_2\text{O}_3$ ). Small amount of other elements are also present (Fatima *et al.*, 2001). However, the burning and calcination process of cement produces pollutants such as heavy metals, dioxins, particulate matters, iron, aluminum, silicon, copper, sulfur dioxide and nitrogen dioxide (Abimbola *et al.*, 2007; Akinola *et al.*, 2008). These pollutants have been implicated in a lot of diseases including respiratory tract diseases, genetic diseases, hematological problems, organs and systems damage, skin damage, sight problems and brain damage (Meo, 2004). Cement dust can cause ill health by skin contact, eye contact or inhalation and risk of injury depends on duration of exposure, level of exposure and individual sensitivity (Abdul-Wahab, 2006). A single or short-time exposure to cement dust may not cause serious harm but exposure to cement dust of sufficient duration may cause serious irreversible health conditions (Heather, 2003). The mean packed cell volume of the blood of Nile tilapia in water treated with cement dust had been observed to decrease significantly with increased concentrations of cement dust in the water (Mohammed and Sambo, 2008). But despite the abundant proofs of toxicity of cement dust to both plants and animals, its use remained sacrosanct because it is the basic ingredient of concrete upon which modern structures lie (Carey, 2005). Then, one of the options left for us in order to avoid the effects of cement dust is to avoid the dust itself by leaving outside the coverage areas of fugitive cement dust from cement companies. Although, much has been said on the coverage areas around a cement company in which the effects of cement dust can be felt (Yang *et al.*, 1996; CRRC, 2003), there is no information on emission coverage potentials of cement companies in Nigeria. Therefore, the objective of this research was to investigate the levels of exposure to elements in cement dust in relation to closeness to cement company and its health implications.

## **MATERIALS AND METHODS**

**Source of animals:** A total of 40 albino rats aged between 4 and 5 weeks old were purchased from an animal farmhouse in Lagos in April 2010. The initial weights of the rats were taken and recorded.

**Experimental plan:** The albino rats were divided into four groups of ten animals per cage. The control rats were resided in Cage 1 and located in 'Ifo' town which is about 6 km from the Cement factory. 'Ifo' is identified to be free of cement dust and is within the same climatic zone as 'Ewekoro'. Group I and II of the test rats were housed in Cages 2 and 3 and placed at 1000 and 500 m away from the factory. Cage 4 housed group III of the test rats and was only 250 meters from the factory. The locations of the cages were determined according to the report of Salami *et al.* (2002). The rats had access to food and water *ad libitum*. The weights of the rats (test and control) were recorded before and after the experiment, at intervals of 30, 60, 90, 120, 150 and 180 days. Their lungs were thereafter removed for chemical analysis using Atomic Absorption Spectrophotometer UNICAM model 969 and histopathology examination according to the method of Taylor *et al.* (2003).

**Statistical analysis:** Data were expressed as Mean Standard Error (Mean $\pm$ SEM) and statistical analysis was carried out using one-tailed student's t-test.  $p < 0.05$  was considered significant.

## RESULTS

Table 1 showed that the exposed and control albino rats gained weights after 180 days of exposure. The minimum weight of the control rats at the start of the study was 182.5 g whereas the maximum weight was 315.3 g at the end of the exposure. The minimum and maximum weights of the exposed rats placed at 1000, 500 and 250 m at the start of the research were 189.8, 181.4 and 180.8 g, respectively and 301.3, 288.6 and 267.3 g, respectively at the end of the research. The mean weight gained by the control rats was 125.6 g, where as the exposed rats placed at 1000, 500 and 250 m from the cement factory exhibited mean weights of 106.0, 98.4 and 82.3 g, respectively. A significant difference ( $p < 0.05$ ) exists between the mean weight gained at the 250 m location and control but not so to other rats at 500 and 1000 m locations and control.

The atomic absorption spectroscopy of the lung tissues of the exposed rats revealed significant amount of calcium, aluminum, silicon, chromium and lead when compared with the control rats. The concentrations of these elements in the lungs of the exposed rats increased with closeness to the cement factory and length of exposure (Table 2-5). For example, from Table 2, the level of calcium and silicon had increased from 8.51 and 0.000 mg kg<sup>-1</sup> to 75.50 and 0.97 mg kg<sup>-1</sup>, respectively for rats placed at 250 m from the cement factory after 180 days. The mean change in the concentrations of calcium, silicon, aluminum, chromium and lead at the end of the study was 67.0, 0.97, 2.81, 0.53 and 0.61 mg kg<sup>-1</sup>, respectively for rats placed at 250 m away from the factory (Table 2). In Table 3, calcium and silicon increased from 8.60 and 0.000 mg kg<sup>-1</sup> to 75.33 and 0.53 mg kg<sup>-1</sup>, respectively for rats placed at 500 m while the mean change in the concentrations of calcium, silicon, aluminum, chromium and lead in the location was 66.7, 0.53, 1.56, 0.34 and 0.4 mg kg<sup>-1</sup>, respectively (Table 3). Calcium and silicon also increased from 8.20 and 0.000 mg kg<sup>-1</sup> to 69.70 and 0.50 mg kg<sup>-1</sup> for rats placed at 1000 m from the cement factory (Table 4) while in the location, the mean change in the concentrations of calcium, silicon, aluminum, chromium and lead are 61.5, 0.40, 0.20, 0.06 and 0.07 mg kg<sup>-1</sup>, respectively (Table 4). Finally, calcium and silicon increased from 7.9 and 0.000 mg kg<sup>-1</sup> to 8.3 and 0.0015 mg kg<sup>-1</sup> for rats in the control group placed in Ifo town (Table 5) while the mean change in the concentrations of calcium, silicon, aluminum, chromium and lead in the location are 0.40, 0.0015, 0.0013, 0.000 and 0.000 mg kg<sup>-1</sup>, respectively (Table 5). The mean concentrations of the elements in the lung tissues of the exposed rats at the different locations were significantly ( $p < 0.05$ ) higher than the mean concentrations of the elements in the lung tissues of the un-exposed rats at the end of 180 days. Calcium was about nine times higher in the lungs of the exposed rats placed at 250 m from the factory compared to the control after 180 days of exposure.

The histopathology examinations of the lung tissues of the control rats showed normal Bronchus (B), normal Respiratory Connective Tissues (RCT) and normal Alveolar Architecture (AA) (Fig. 1). The lung tissues of the exposed rats indicated multi-organ injuries: The lung tissues of the rats placed at 250 m indicated abnormal Alveolar Architecture (AA), Damaged Bronchioles (DB), disrupted Bronchus (B), weak Respiratory Connective Tissues (RCT), Inflammations (I) and blue-black Pigments (P) (Fig. 2); the rats placed at 500m had normal open Alveolar Space (AS), Damaged Bronchioles (DB), disrupted Bronchus (B), abnormal Alveolar Architecture (AA), weak Respiratory Connective Tissues (RCT) and mild area of Inflammations (I) (Fig. 3) and finally the rats placed at 1000 m showed normal Alveolar Space (AS), slightly normal Alveolar Architecture (AA) Damaged Bronchioles (DB), mild area of Inflammations (I) and slight blue-black Pigments (P) (Fig. 4).

Table 1: Mean weights (g) gained by the exposed rats at the different locations

Rat location	0 day	30 day	60 day	90 day	120 day	150 days	180 days	Minimum weight	Maximum weight	Mean weight
Control (Ifo town) about 6 km	185.7±24.0 <sup>a</sup>	214.7±16.2 <sup>b</sup>	239.3±16.6 <sup>a</sup>	259.7±15.0 <sup>b</sup>	279.3±15.0 <sup>a</sup>	293.7±13.4 <sup>b</sup>	311.3±11.6 <sup>a</sup>	182.5	315.2	254.81
1000 m	192.0±24.1 <sup>a</sup>	216.7±25.6 <sup>b</sup>	235.7±23.4 <sup>a</sup>	254.0±22.0 <sup>b</sup>	272.3±22.4 <sup>a</sup>	286.3±20.3 <sup>b</sup>	298.0±20.3 <sup>a</sup>	189.81	301.32	250.7
500 m	186.3±16.4 <sup>a</sup>	202.0±16.7 <sup>b</sup>	221.3±16.2 <sup>a</sup>	241.3±15.8 <sup>b</sup>	261.0±15.3 <sup>a</sup>	277.3±13.5 <sup>b</sup>	284.7±12.8 <sup>a</sup>	181.43	288.64	239.1
250 m	183.0±18.2 <sup>a</sup>	193.7±16.31 <sup>a</sup>	200.3±12.5 <sup>a</sup>	212.0±15.8 <sup>b</sup>	230.0±17.3 <sup>a</sup>	247.7±18.2 <sup>b</sup>	265.3±16.40 <sup>a</sup>	180.82	267.33	218.9

Data are expressed as Mean±SEM. When (\*p<0.05); Significant from control and when (\*\*p>0.05); Not significant from control. Values with different superscript from the next value in the same row are significantly different at p<0.05

Table 2: Concentrations in (mg kg<sup>-1</sup>) of the elements in the lungs of the exposed rats placed at 250 m from the cement company

Element	0 day	30 days	60 days	90 days	120 days	150 days	180 days	Minimum conc.	Maximum conc.	Mean conc.	Mean change	p-values	OSha regulatory limit
Ca	8.51±1.02 <sup>a</sup>	19.12±2.58 <sup>b</sup>	56.96±1.58 <sup>a</sup>	65.6±2.51	70.44±1.12 <sup>a</sup>	72.50±1.68 <sup>a</sup>	75.50±1.10 <sup>a</sup>	8.48	76.80	52.70	67.0	<0.001*	8-10.5
Si	ND	0.13±0.02 <sup>a</sup>	0.29±0.02 <sup>b</sup>	0.35±0.04 <sup>a</sup>	0.41±0.02 <sup>b</sup>	0.53±0.05 <sup>a</sup>	0.97±0.11 <sup>a</sup>	0.000	0.99	0.40	0.97	<0.001*	0.10
Al	ND	1.23±0.02 <sup>a</sup>	1.66±0.10 <sup>b</sup>	2.30±0.35 <sup>a</sup>	2.35±0.29 <sup>a</sup>	2.65±0.33 <sup>b</sup>	2.81±0.41 <sup>a</sup>	0.000	1.90	2.85	2.81	<0.001*	5.00
Cr	ND	0.04±0.02 <sup>a</sup>	0.13±0.01 <sup>b</sup>	0.21±0.02 <sup>a</sup>	0.32±0.02 <sup>b</sup>	0.42±0.02 <sup>a</sup>	0.53±0.03 <sup>b</sup>	0.000	0.56	0.20	0.53	<0.002*	0.05
Pb	ND	0.03±0.02 <sup>a</sup>	0.22±0.04 <sup>b</sup>	0.30±0.03 <sup>b</sup>	0.42±0.05 <sup>a</sup>	0.53±0.05 <sup>b</sup>	0.61±0.08 <sup>b</sup>	0.000	0.66	0.30	0.61	<0.001*	0.05

Data are expressed as Mean±SEM. When (\*p<0.05); Significant from control and when (\*\*p>0.05); Not significant from control. Values with different superscript from the next value in the same row are significantly different at p<0.05. OSHA: Occupational safety and health administration. ND: Not detected

Table 3: Concentrations in (mg kg<sup>-1</sup>) of the elements in the lungs of the exposed rats placed at 500 m from the cement company

Element	0 day	30 days	60 days	90 days	120 days	150 days	180 days	Minimum conc.	Maximum conc.	Mean conc.	Mean change	p-values	OSha regulatory limit
Ca	8.60±1.70 <sup>a</sup>	16.21±1.31 <sup>b</sup>	49.30±1.82 <sup>a</sup>	63.30±3.51 <sup>b</sup>	67.67±1.52 <sup>b</sup>	70.73±2.01 <sup>b</sup>	75.33±1.20 <sup>b</sup>	8.55	76.32	50.2	66.7	<0.0001*	8-10.5
Si	ND	0.073±0.02 <sup>a</sup>	0.13±0.02 <sup>b</sup>	0.27±0.06 <sup>a</sup>	0.33±0.04 <sup>a</sup>	0.42±0.02 <sup>b</sup>	0.53±0.04 <sup>a</sup>	0.000	0.56	0.30	0.53	0.00003*	0.10
Al	ND	0.12±0.01 <sup>a</sup>	1.34±0.03 <sup>b</sup>	1.40±0.01 <sup>b</sup>	1.50±0.02 <sup>a</sup>	1.53±0.03 <sup>a</sup>	1.56±0.02	0.000	1.58	1.10	1.56	<0.0001*	5.00
Cr	ND	0.002±0.0001 <sup>a</sup>	0.05±0.001 <sup>a</sup>	0.15±0.02 <sup>b</sup>	0.24±0.03 <sup>a</sup>	0.32±0.03 <sup>b</sup>	0.34±0.02 <sup>b</sup>	0.000	0.36	0.20	0.34	<0.0001*	0.05
Pb	ND	0.007±0.001 <sup>a</sup>	0.12±0.01 <sup>a</sup>	0.19±0.03 <sup>a</sup>	0.32±0.05 <sup>b</sup>	0.40±0.05 <sup>a</sup>	0.49±0.07 <sup>b</sup>	0.000	0.51	0.20	0.49	0.0001*	0.05

Data are expressed as Mean±SEM. When (\*p<0.05); Significant from control and when (\*\*p>0.05); Not significant from control. Values with different superscript from the next value in the same row are significantly different at p<0.05. OSHA: Occupational safety and health administration. ND: Not detected

Table 4: Concentrations in (mg kg<sup>-1</sup>) of the elements in the lungs of the exposed rats placed at 1000m (1 km) from the cement company

Element	Minimum										Maximum		Mean		Osha regulatory limit
	0 day	30 day	60 day	90 days	120 days	150 day	180 day	conc.	conc.	conc.	conc.	change	p-value		
Ca	8.20±1.00 <sup>a</sup>	10.6±1.41 <sup>a</sup>	42.9±2.51 <sup>b</sup>	58.3±2.12 <sup>a</sup>	59.9±1.21 <sup>a</sup>	62.1±1.68 <sup>a</sup>	69.7±0.32 <sup>a</sup>	7.96	70.23	44.5	61.5	<0.0001*	<0.0001*	8-10.5	
Si	ND	0.059±0.002 <sup>a</sup>	0.091±0.012 <sup>b</sup>	0.12±0.01 <sup>a</sup>	0.27±0.03 <sup>b</sup>	0.31±0.06 <sup>b</sup>	0.40±0.01 <sup>b</sup>	0.0	0.42	0.2	0.4	0.00001*	0.00001*	0.1	
Al	ND	0.003±0.0001 <sup>a</sup>	0.025±0.003 <sup>b</sup>	0.074±0.023 <sup>a</sup>	0.082±0.017 <sup>a</sup>	0.12±0.048 <sup>b</sup>	0.20±0.12 <sup>b</sup>	0.0	0.23	0.1	0.2	<0.0001*	<0.0001*	5.0	
Cr	ND	ND	ND	ND	0.01±0.01 <sup>a</sup>	0.04±0.01 <sup>a</sup>	0.06±0.02 <sup>a</sup>	0.0	0.08	0.02	0.06	<0.0009*	<0.0009*	0.05	
Pb	ND	ND	ND	ND	0.04±0.02 <sup>a</sup>	0.06±0.02 <sup>a</sup>	0.07±0.01 <sup>a</sup>	0.0	0.09	0.02	0.07	0.00054*	0.00054*	0.05	

Values are expressed as Mean±SEM. When (\*p<0.05); Significant from control and when (\*\*p>0.05); Not significant from control. Values with different superscript from the next value in the same row are significantly different at p<0.05. OSHA: Occupational safety and health administration. ND: Not detected

Table 5: Concentrations in (mg kg<sup>-1</sup>) of the elements in the lungs of the exposed rats placed in Ifo town, about 6km from the cement company

Element	Minimum										Maximum		Mean		Osha regulatory limit
	0 day	30 day	60 day	90 days	120 days	150 day	180 day	conc.	conc.	conc.	conc.	change	p-value		
Ca	7.91±0.18 <sup>a</sup>	8.05±0.42 <sup>a</sup>	8.24±0.61 <sup>a</sup>	8.24±0.54 <sup>a</sup>	8.28±0.48 <sup>a</sup>	8.29±0.50 <sup>a</sup>	8.30±0.51 <sup>a</sup>	7.88	8.35	8.20	0.40	0.6995**	0.6995**	8-10.5	
Si	ND	0.0012±0.0001 <sup>a</sup>	0.0013±0.0001 <sup>a</sup>	0.0013±0.0001 <sup>a</sup>	0.0014±0.0002 <sup>a</sup>	0.0015±0.0001 <sup>a</sup>	0.0015±0.0001 <sup>a</sup>	0.000	0.0016	0.0012	0.0015	0.3028**	0.3028**	0.1	
Al	ND	0.0012±0.0001 <sup>a</sup>	0.0012±0.0001 <sup>a</sup>	0.0012±0.0001 <sup>a</sup>	0.0013±0.0003 <sup>a</sup>	0.0013±0.0002 <sup>a</sup>	0.0013±0.0001 <sup>a</sup>	0.000	0.0014	0.0011	0.0013	0.3700**	0.3700**	5.00	
Cr	ND	ND	ND	ND	ND	ND	ND	0.000	0.000	0.000	0.000	NA	NA	0.05	
Pb	ND	ND	ND	ND	ND	ND	ND	0.000	0.000	0.000	0.000	NA	NA	0.05	

Values are expressed as Mean±SEM. When (p<0.05); Significant and when (p>0.05); Not significant. Values with different superscript from the next value in the same row are significantly different at p<0.05. OSHA: Occupational safety and health administration. ND: Not Detected. NA: Not applicable

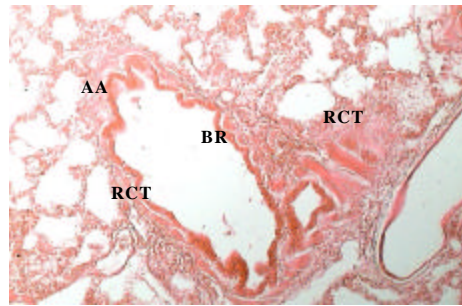


Fig. 1: Histological section through the lung of the control rat showing normal Bronchus (B), normal Respiratory Connective Tissues (RCT) normal bronchiole and normal Alveolar Architecture (AA)

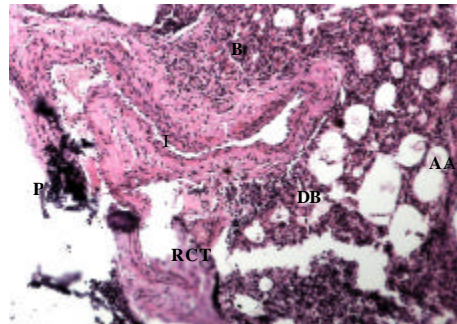


Fig. 2: Histological section through the lung tissues of the rats placed at 250 m showing Abnormal Alveolar (AA), Damaged Bronchiole (DB), Disrupted Bronchus (B), weak Respiratory Connective Tissues (RCT), Inflammation (I) and blue-black Pigment (P)

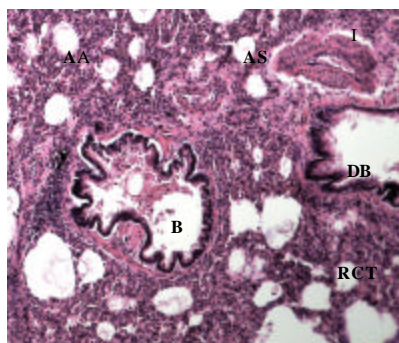


Fig. 3: Histological section through the lung tissues of the rats placed at 500 m showing normal open Alveolar Space (AS), Damaged Bronchiole (DB), disrupted Bronchus (B), Abnormal Alveolar (AA), weak Respiratory Connective Tissues (RCT) and mild area of Inflammation (I)

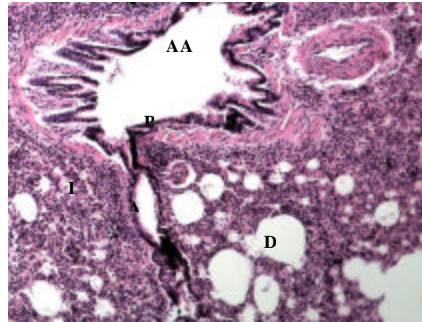


Fig. 4: Histological section through the lung tissues of the rat placed at 1000 m showing normal Alveolar Space (AS) (AA), Damaged Bronchiole (DB), mild area of Inflammation (I) and slight blue-black Pigment (P)

## DISCUSSION

The discovery of the toxic elements-aluminum, chromium, silicon and lead in the lungs of the exposed rats in this research supports the findings of Davidovits (1994), Fatima *et al.* (2001) Abimbola *et al.* (2007) and Akinola *et al.* (2008). The result is also in line with Gbadebo and Bankole (2007) and Ade-Ademilua and Obalola (2008) who discovered aluminum, copper, zinc, silicon, magnesium, iron, calcium, cadmium and lead in air-borne particles around West African Portland Cement Factory, Sagamu, Ogun State, Nigeria. The weights gained by the exposed rats in this study contradicts the earlier report by Akinola *et al.* (2008), who observed a reduction in the weights of the albino rats exposed to cement dust. The weights gained by the exposed rats might be as a result of calcium deposits in the lungs of the exposed rats; calcium is a major component of animal feeds and is very important in the formation of bones and blood. Thus, the weight gained by the exposed rats is not a surprise.

The abnormal high concentrations of the toxic elements in the lung tissues of the exposed rats is an indication of high level of pollution from the cement factory which supports the assertion of Bilen (2010), that cement production is one of the great polluters of the environment. The heavy pollution observed might be as a result of the use of old machinery and left-over cement kiln dust which were not properly disposed. Also, like what is being practiced in some countries, cement manufacturers in Nigeria, especially the Ewekoro cement, might just be using hazardous wastes like tires as alternative source of fuel. This supports the observations of IPC (1996) who found that the levels of heavy metals and dioxins in cement kiln dust from Ribblesdale, United States of America, were higher when cement fuel was burned. Cement fuel is an alternative fuel made of hazardous wastes which are burnt as a source of energy to reduce energy cost while achieving cement production. The concentrations of these toxic elements in the lungs of the exposed rats increased with duration of exposure, degree of exposure and closeness to the cement factory which is in line with what Abdul-Wahab (2006) and Heather (2003) reported. The result also supports the findings of Yang *et al.* (1996) who discovered that an area within 0-2 km of a cement factory is a high danger zone. It also supports the findings of a medical doctor, Crawford, who stated that an area within five miles of a cement plant will be the high danger zone and within twenty miles of a cement plant will be the danger zone (CRRC, 2003).



The diseases observed in the exposed rats are in line with the findings of Meo (2004) and Mohammed and Sambo (2008) who have implicated cement dust and its constituents in many health problems. Although calcium is important in metabolism in moderate quantities, excess amount could cause toxicity. Excess amounts of calcium have been implicated in brain injury (Fan *et al.*, 2007). Aluminum and silicon have also been implicated in some diseases. Thus, aluminum has been reported to lead to dementia in dialysis patients (CDCP, 2008), metabolic bone disease (Kausz *et al.*, 1999; Klein, 1998) while its inhalation has been associated with asthma (Sorgdrager *et al.*, 1998). It also caused induced degeneration of astrocytes (Suarez-Fernandez *et al.*, 1999). Silicon is believed to cause immune system changes in breast implant patients. Lapin *et al.* (1991) observed that the lungs of rats when exposed to silicon carbide whiskers, had inflammatory lesions, focal pleural fibrosis and lymphoid hyperplasia while Hubbs *et al.* (2001) showed that inhalation of cement dust is associated with pulmonary fibrosis.

The marked histological changes observed in the lungs of the exposed rats showed that there is interaction between the toxic elements in cement dust and the various organs of animals and plants living within the vicinity of cement plants. The results of the histopathology is in line with what Akinola *et al.* (2008) observed in the various organs of albino rats exposed to cement dust. Lead and chromium which were not detected in the control rats were present in the exposed rats after 30 days, increasing in concentrations with closeness to the factory and duration of exposure. These two elements, lead and chromium, can increase the risk of cancer (Laj *et al.*, 1984) and negatively affect the homeopathic, nervous and reproductive systems (Papanikolaou *et al.*, 2005; Lanphear, 1998).

## CONCLUSION

We report that rats exposed to cement dust exhibit histopathology changes in lung tissues as a result of cytotoxic agents emanating from the cement factory. The studies confirm that the amounts of the elements lodged in rats organs increased with closeness to the factory and length of exposure. Predictably, it is suggested that humans living or working within the vicinity of cement plants may be victims of the same health hazards reported for the experimental rats. We therefore, recommend that there is an urgent need for government agencies charged with environmental and health protection, to inspect cement factories from time to time and enforce the use of more efficient, low emission modern machines to reduce the amount of cement dust emitted into the environment. In addition, there should be legislation on the minimum distance between air polluting factories and residential areas. It is also important that residents of cement dust pollution prone zones, who may not be aware of the hazards of exposure to cement dust, need to be enlightened and protected as most of them are peasant farmers with limited or no education. Finally, the regulatory Agencies should be more committed to monitoring industrial activities generally to minimize all forms of environmental pollution including air pollution from cement dust emission.

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