INTERNATIONAL RESEARCH JOURNAL OF PHARMACY

www.irjponline.com

ISSN 2230 – 8407

Research Article

HEMATOLOGICAL ALTERATIONS AMONG QUARRY WORKERS EXPOSED TO GRANITE DUST IN THAILAND

Vijaya Bhaskara Reddy M *

Faculty of Public Health, ST Theresa International College, Rang Sit, Nakhonnayok Road, Klong 14, Bungsan, Ongkharak, Nakhonnayok, Thailand

*Corresponding Author Email: vijaya@stic.ac.th

Article Received on: 13/01/20 Approved for publication: 27/02/20

DOI: 10.7897/2230-8407.110326

ABSTRACT

The objective of this study is Granite quarry dust predominantly present at quarry sites. Exposure to quarry dusts is a major concern to the quarry workers and human beings. Lethal dust particles are detrimental to human's health. Hence, it may be accountable for hematological alterations in exposed community. This paper is a first to investigate the alterations of hematological parameters and to mimic silica dust implications in exposed and control workers. A total 200 subjects were selected randomly and grouped as experimental (N = 100) and control (N = 100). Blood samples were collected and used for hematological analysis. The study indicates that 84% of the quarry workers have 12-17 years of exposure to the quarry dust. Significant alteration was observed among selected hematological profile of exposed workers when compared with their control groups. The levels of total leukocytes (TLC) increased significantly ($P < 0.0001$) in both the groups exposed to quarry dust. Results of this study indicate that the quarry dust particles insist inflammation in humans. A significant depletion was observed in red blood cells (RBC) in both exposed groups. Declined levels of red blood cells could be due to inflammation. Increased levels of eosinophils were observed in exposed group. Analysis of hematopoietic system alterations in quarry dust exposed workers are crucial to mimic the health effects of quarry dust, as biomarkers of quarry dust exposure, inventing a better and easy diagnosis develop preventive standards and to measure epidemiological surveillance. Granite quarry management should take initiative steps to frequent monitoring of their quarry workers.

Keywords: Quarry dust, hematopoietic system, platelet, occupational exposure, and silica.

INTRODUCTION

The global burden of quarry industries is substantial; especially, occupational hazards and illnesses in developing countries. In most of the countries, the construction industry contributes significantly to gross domestic product. It plays a vital role in terms of socioeconomical development of the country. In the 19th century, crushed stone dust was reported as potential material of choice for building durable roads and constructions. In due course of time, trap rock, metamorphic rock and limestone, granite, sandstone and shale were used for laying roads and other construction sectors. These potential uses and high demand posed to grew the industry[1]. The majority of local communities depend on small scale quarrying to lead their livelihood[2]. Despite their significant contribution, it has negative impacts on the health and safety of the quarry workers are highly concerned[3]. Occupational injury fatality rates are rise approximately two to five times in the developing regions of the world, when compared to North America and Western regions of Europe[4]. Mixing, disaggregation and ambient nature, exposure to atmospheric surface and segregation due to reduced particle size may induce physical and chemical transformation with devastating environmental effects. Silica constitute with 12% of earth's crust, abundant in nature and causes silicosis. Silicosis is one of the dreaded occupational diseases[5]. Operators front end loaders, rotary drills, crusher and truck drivers are prone to expose to stone dust[6]. During these operations majority of the dust particles generated, visible, invasive and potential to cause irritation among the workers and neighbourhood[7].

The quarry dust is a powder like adhesive substance, which contains 76% of silicon oxide (SiO_2), 10.4% of aluminium trioxide (AL_2O_3), ferric oxide (Fe_2O_3), calcium oxide (CaO), magnesium oxide. As well as sodium oxide Na_2O, potassium oxide K_2O, titanium dioxide TiO_2, lithium oxide LOI, clay, other impurities[8], asbestos lead, and airborne solids[9]. Mineral composition as follows silicon oxide (SiO_2), 76%, 10.4% of aluminium trioxide (AL_2O_3), and the rest 13.3 % share the remaining minerals such as K_2O, Na_2O, FeO, CaO, Fe_2O_3, TiO_2, P_2O_5 and MnO respectively[10].

Quarry workers are exposed to dust, due to their wide range of activities at quarrying sites. Quarry dust generates mainly during the quarrying activities, handling of raw materials, grinding the clinker, blending and transportation[11]. Dust particle sizes ranged from 1 – 100 micro-meters in diameter and they may be airborne depending on the size, origin and ambient conditions[12]. Respiratory system and gastrointestinal tracts are the possible ways to enter the quarry dust particles into body. Quarry dust particles are classified as inhalable or respirable based on the size of the particles. If the particle size is PM10, categorised as inhalable, which is deposited mainly in the airways and PM 2.5 then respirable, deposits in the respiratory tract especially in the gas exchange area. Whenever these particles accumulated into head and or lungs, they have the potential to cause detrimental hazards either locally; consequently elsewhere in the body[2]. The accumulated quarry dust particles could have an impact on the physical and chemical properties of dust particles, as well as on host. The physical properties of dust particles are such as particle size, shape, density, surface area, penetrability, hygroscopic

activity, electrostatic charge, and acidity or alkalinity of the inhaled agent. Quarry dust particles could cause adverse effects in host. The inflated concentration and duration of exposure to quarry dust initiates a potential root of occupational disease among quarry workers. According the environmental protection agency recommended safe levels for PM10 and PM2.5 are 180 mg/m^3 and 35 mg/m^3 respectively. Similarly, it has been found and reported that cement dust particles could be found in wide range of body organs such as blood, liver, spleen, and bone. Thus, cement particles could induce wide variety lesions[13,14].

In the recent past most frequently reported adverse symptoms are cough, impaired lung function, phlegm production, alterations in respiratory muscles[15], emphysema, bronchitis, bronchial asthma, restrictive lung disease, chest tightness, skin irritation, stomach-ache, inflammation and conjunctivitis[16]. Moreover, dust of quarry and related industries like cement also imposes atrophic alterations in nasal and pharyngeal mucosa, itching burning, tissue fibrosis, watery eyes, prostration, headache, lung cancer, and stomach and colon cancers in extreme cases[17-19].

However, few studies have been reported on devastating effects of urban dust on cellular functions, impaired proliferation, phagocytic activity and defence mechanisms[20], wooddust[21], asbestosis[22], dust storms[23]. Quarry based industries are one of the largest sectors and predominantly abundant with global development. Quarry industries providing millions of job opportunities globally. The point of worth to discuss is that millions of workers have been waged on daily basis in quarry industries. However, there is a lacuna in literature based on haematological alterations as biomarker indicators to elucidate the adverse effects of quarry dust exposure in human beings. As we know the haematological parameters play a vital role in the basic immune system. Therefore, this study was aimed to determine the detrimental effects of quarry dust on selected blood parameters to mimic the potential effects in quarry workers.

MATERIAL AND METHODS

Haematological and biochemical parameters were analysed by using commercially available kits. Measured variables included red blood cell (RBC) count, white blood cell (WBC) count, haemoglobin (Hb). All the procedures are approved by the St Theresa international college, institutional animal ethics committee (STIC/TH/PH/01/2019).

Primary data was collected by the author with the help of Thai-native speaker through personal interactions with the subjects. To acquire accurate and reliable data, author took all the precautionary measures, apart from that caution and care were taken while data collection. The author took all possible effort to establish a friendly relationship with the quarry respondents. So that the respondents do not feel hesitation to provide samples, personal information and data. Prior to interview, the author described the prime objectives of the study to each and every quarry worker. Consequently, they were convinced that the purpose of the study was purely a research and was not likely to have any adverse effects or diminish their livelihood.

The experimental data was tabulated and statistically evaluated by ANOVA with the help of SPSS version 22.0 package

RESULTS AND DISCUSSION

Table 1: Distribution of male quarry workers based on the years of exposure to dust

Male		Frequency	Percent
Valid	0-2	4	4
	3-5	5	5
	6-8	5	5
	9-11	10	10
	12-14	24	24
	15-17	40	40
	17 and above	12	12
	Total	100	100.0

Source: Field survey

In this study based on the primary data of quarry workers categorized into different groups based on their exposure to the quarry dust. From the Table 1 demonstrates the years of exposure of workers. The study indicates that 84% of the quarry workers have 12-17 years of exposure to the quarry dust.

Table 2: Distribution of female workers based on the years of exposure to dust

Female		Frequency	Percent
Valid	0-2	11	11
	3-5	15	15
	6-8	12	12
	9-11	8	8
	12-14	18	18
	15-17	30	30
	17 and above	6	6
	Total	100	100.0

Source: Field survey

Table 2 demonstrates the years of exposure of female workers. The study indicates that 48% of the female quarry workers have 12-17 years of exposure to the quarry dust.

Table 3: Hematological alterations due to silica dust exposure in both female and male groups

Parameter	Control	Exposed workers	ANOVA
Hemoglobin	12.76 ± 0.35	11.71 ± 0.54	$p < 0.0001^{**}$ t = 16.53
Hematocrit	37.54 ± 0.266	35.01 ± 0.75	$p < 0.0001^{**}$ t = 31.90
Total leukocytes	6907.86 ± 46.40	8257.10 ± 99.86	$p < 0.0001^{**}$ t = 123.03
Polymorphs	59.20 ± 0.51	62.97 ± 0.69	$p < 0.0001^{**}$ t = 44.19
Lymphocytes	35.68 ± 0.68	33.41 ± 1.37	$p < 0.0001^{**}$ t = 14.80
Eosinophils	2.02 ± 0.40	3.20 ± 0.18	$p < 0.0001^{**}$ t = 26.49
Monocytes	1.25 ± 0.17	1.65 ± 0.24	$p < 0.0001^{**}$ t = 13.43
Basophils	0.48 ± 2.98	0.13 ± 0.02	p = 0.24 t = 1.17
Platelets	2.05 ± 0.15	2.46 ± 0.12	$p < 0.0001^{**}$ t = 22.01
Red blood cells	4.21 ± 0.31	3.38 ± 0.16	$p < 0.0001^{**}$ t = 25.94

Table 4: Hematological alterations due to silica dust exposure in males

Parameter	Control	Exposed workers	ANOVA
Hemoglobin	12.75 ± 0.35	12.18 ± 0.25	$p < 0.0001^{**}$ t = 8.94
Hematocrit	37.54 ± 0.29	35.58 ± 0.75	p < 0.0001 t = 20.30
Total leukocytes	6907.86 ± 46.40	8167.81 ± 32.93	$p < 0.0001^{**}$ t = 165.94
Polymorphs	59.20 ± 0.51	63.40 ± 0.69	$p < 0.0001^{**}$ t = 44.06
Lymphocytes	35.94 ± 0.68	32.19 ± 0.29	$p < 0.0001^{**}$ t = 29.19
Eosinophils	2.23 ± 0.43	3.10 ± 0.08	$p < 0.0001^{**}$ t = 13.73
Monocytes	1.19 ± 0.14	1.43 ± 0.07	$p < 0.0001^{**}$ t = 10.56
Basophils	0.16 ± 0.12	0.14 ± 0.01	p = 0.34 t = 0.95
Platelets	2.02 ± 0.15	2.47 ± 0.12	$p < 0.0001^{**}$ t = 13.42
Red blood cells	4.18 ± 0.44	3.46 ± 0.08	$p < 0.0001^{**}$ t = 11.72

Table 5: Hematological alterations due to silica dust exposure in female

Parameter	Control	Exposed workers	ANOVA
Hemoglobin	12.81 ± 0.35	11.39 ± 0.36	$p < 0.0001^{**}$ t = 19.80
Hematocrit	37.55 ± 0.266	34.44 ± 0.30	$p < 0.0001^{**}$ t = 58.80
Total leukocytes	6908.92 ± 46.40	8346.39 ± 52.87	$p < 0.0001^{**}$ t = 139.23
Polymorphs	59.20 ± 0.43	62.55 ± 0.67	$p < 0.0001^{**}$ t = 29.43
Lymphocytes	35.41 ± 0.22	34.64 ± 0.81	$p < 0.0001^{**}$ t = 6.51
Eosinophils	1.81 ± 0.22	3.30 ± 0.19	$p < 0.0001^{**}$ t = 35.30
Monocytes	1.31 ± 0.18	1.86 ± 0.11	$p < 0.0001^{**}$ t = 17.79
Basophils	0.21 ± 0.21	0.12 ± 0.01	$p < 0.004^{*}$ t = 2.97
Platelets	2.08 ± 0.05	2.47 ± 0.10	$p < 0.0001^{**}$ t = 22.22
Red blood cells	4.23 ± 0.04	3.16 ± 0.06	$p < 0.0001^{**}$ t = 91.91

Haematological parameters of this study were tabulated in Table 3, 4, and 5. Table 3 deals with pooled data of both male and female, Table 4 represents the data of males in exposed and control groups and Table 5 represents the data of female groups. Table one represents haematological parameters of both male and female groups. The levels of total leukocytes (TLC) increased significantly ($P < 0.0001$) in both the groups exposed to quarry dust. Results of this study indicate that the quarry dust particles insist inflammation in humans. A significant depletion was observed in red blood cells (RBC) in both exposed groups.

Declined levels of red blood cells could be due to inflammation and theses conditions found in anaemia. Quarry dust may cause anaemic conditions in the quarry workers. Haemoglobin, haematocrit, lymphocytes were depleted significantly among exposed quarry workers. This could be due to inhalation of quarry dust which has silica and evidenced that our body intensifies the immunological response against to silica dust; whereas total leukocytes were increased significantly among the occupationally exposed group when compared with their respective control groups. The elevated levels of WBC in this study suggest our immune system response against quarry dust allergy in our body.

Furthermore, reduced levels of RBCs were observed in occupationally exposed workers to quarry dust. This in aggregation with results of Mandal and Suva[24]; found and reported decreased Haemoglobin and elevated levels of eosinophils in construction workers exposed to cement dust. Decreased levels of haemoglobin could be due to stress response of immune system to keep the normal range of RBCs, it leads to significant increase in immature erythrocytes in blood. The increased WBCs count suggests enhanced allergic response after exposure to silica dust. Elevated levels of platelet count were observed in workers exposed to quarry dust. Findings of this study in agreement with Mojiminiyi[25] and similarly reported among the cement dust exposed workers in Nigeria. These significant depletions and elevated levels of haematological parameters clearly evidenced and revealed that the granite quarry dust could alter the cell walls of blood cells. Alterations in haematology could be due damaged RBC membranes, leads to stress in haemolysis production, RBC from the spleen and hypoxic conditions[26,27]. Haematocrit plays an important role in diagnosis of anaemia in mammals. The assessments of haematological parameters play an inevitable role in determination the devastating effects of quarry dust generated occupational implications in quarry workers. It helps in surveillance activities and establishes the consequences of the quarry dust induced hazardous alterations in humans. Continuous flow of exogenous and endogenous reactive oxygen species, leads oxidative damage to cellular compounds and consequently alters the cellular functions[28,29].

In this study the levels of haematocrit were suppressed significantly in the quarry workers. These could be due to lower levels of blood cells and evidenced that the haematocrit abundant to anaemia in quarry workers. Total leukocytes increased significantly in quarry workers of the all groups, it indicates that they have infections, inflammations, oxidative and emotional stress. Moreover, higher levels indicate especially our immune system combating against to the infections. Continuous flow of white blood cells may lead to abnormal cell production at last. As a result, the levels of polymorphs (neutrophils) fluctuate in the body. Prime role of the neutrophils is to defend the foreign body which enters in to our body. Similarly, in this study the levels of polymorphs increased significantly which means the quarry workers are suffering with inflammatory stress in their body. Raised levels of polymorphs could be due to bronchitis, tonsillitis, polymyalgia rheumatica, polyarthritis, stress due to extensive work and coughing due to percolation of dust particles, silicosis and lithium. In the light of the above observations it is noticed that due the inflammations, cellular damage and infections of silica containing quarry dust particle substances harming the hematopoietic system of the quarry workers. When our body senses the trouble in the form of harmful substance or infection in any part of our body it sends the signal to enhances the yield of white blood cells. Polymorphs are main elements of white blood cells, when white blood cells increase polymorphs increases. Significant decrease in lymphocytes indicates the presence of infections in the body (Table 3, 4 and 5).

Levels of eosinophils suppressed significantly in the exposed groups; it could be due to eosinophilia. Elevated levels of the monocytes and basophils indicate in response to chronic infections due to quarry dust particles. Elevated levels of platelets (Table 3, 4 and 5) indicate the ongoing inflammation abnormal cells in bone marrow of the quarry workers, consequently it may cause thrombosis. Mammalian haematopoietic system is known to be susceptible to environmental toxicants because of extensive synthesis of cell and apoptosis, its immediate repercussions are increased metabolic demands[30]. So, mammalian haematopoietic system can be used as biomarker in toxicological studies. Similar findings have been reported on protective and preventive role of haematological parameters in diseases related to oxidative stress[31,32]. Especially, erythropoietin ameliorates oxygen radicals induced apoptosis[32].

Elevated levels of eosinophils in all the quarry dust exposed groups when compared with the control groups. The results of the in accordance with Conroy and Williams[33], who demonstrated that the increased levels of eosinophils are due to exotoxin induced oxidative stress. Hypobromous acid induced oxidative and DNA damages elevate the eosinophils[34]. Hence the results of this study suggest that quarry dust could induce inflammatory mediated DNA damage and may leads to lymphocytosis in quarry workers. Similar findings have been reported in cement factory workers[35,30,36]. Occupational exposures to dust containing silica induce inflammation in specific argons namely dermal, respiratory, and liver[37]. Persistently exposure could progress to granulomatous, fibrosis and even cancers in specific sites[37].

Health risks posed to quarry workers include particle size, composition and concentration of the dust, deposition location within the respiratory tract and the exposure duration[38]. Deposition of the heavy metals may cause kidney and brain damage and lung cancer[39]. Shortness of breath, chest pain, cough and wheezing are indicative of the onset of silicosis. Silicosis predisposes people to pneumonia, lung cancer and tuberculosis[40].

CONCLUSION

The observed blood parameter levels of quarry workers suggest that silica dust may imposes immense flow of exogenous and endogenous reactive oxygen species, which leads oxidative damage to cellular compounds and consequently alters the cellular functions. Elevated levels of eosinophils evidenced that long term granite dust particles induce exotoxins, consequently oxidative damage may have taken place in cellular functions. The findings of the study suggest that the long-term consequences of quarry dust must be kept on surveillance. Granite quarry management should take initiative steps to frequent monitoring of their quarry workers.

Therefore, analysis of hematopoietic system alterations in quarry dust exposed workers are crucial to mimic the health effects of quarry dust, as biomarkers of quarry dust exposure, inventing a better and easy diagnosis develop preventive standards and to

measure epidemiological surveillance. Further studies need to investigate the underlying phenomena of quarry dust induced toxicity.

ACKNOWLEDGMENT

Author is thankful to all the staff and crew of granite factories, volunteers who participated in the study. Special thanks to St. Theresa international college, partial funding, Faculty of Public Health, Division of Toxicology for helping in the collection of samples.

REFERENCES

1. Langer WH, Bon RL, Riordan RF, Tripp BT and Krukowski ST. Environmental impacts of mining natural aggregates. Proceedings of the 35th Forum on the Geology of Industrial Minerals. The International West Forum: Utah Geological Survey Miscellaneous Publication 2001; 0102: 127-138.
2. Asante F, Abass K, and Afriyie, K. Stone quarrying and livelihood transformation in Per-Urban Kumasi. Research in humanities and social sciences 2014; 4(13): 93-107.
3. (USGS, 2007, 2013). http://minerals.usgs.gov/minerals/ pubs/commodity/stone_crushed/stat/index.html. Accessed on 20-11-2019.
4. Concha-Barrientos M, Nelson DI, Driscoll T, Steen land NK, Punnett L, Fingerhut MA, Prüss-Üstün A, Leigh J, Tak SW, Corvalan C. Selected occupational risk factors. In: Comparative quantification of health risks: global and regional burden of diseases attributable to selected major risk factors. Geneva: World Health Organization; 2004. p. 1651-801.
5. Chattopadhyay BP, Gangopadhyay PK, Bandopadhyay TS, Alam, J. Comparison of Pulmonary Function Test Abnormalities between Stone Crushing Dust Exposed and Non-Exposed Agricultural Workers. Env Health and Prev. Med 2006; 11: 191-198.
6. Chekan GJ and Colinet JF. Silica dust sources in underground limestone mines. In: Proceedings of the 33rd Annual Institute on Mining Health, Safety and Research, Roanoke, Virginia; 2002. p. 11-13.
7. MIRO. Good practice guide: control and measurement of nuisance dust and PM10 from extractive industries. Report to the Mineral Industry Research Organization (MIRO). AEAT/ENV/R3 2011; 140: 141.
8. Short S, Petsonk EL. Non-fibrous inorganic dusts. In: Harber P, Schenker MB, and Balmes JR. Occupational and environmental respiratory disease. London: Mosby 1996; 356.
9. Levin, SM. Asbestos related Abnormalities among Demolition workers. Electronic library of construction and occupational safety and health. Georgia: Centre for Construction Research and Training; 1994.
10. Chapel BW and White AJR. Two granite types: 25 years later. Australian Journal of Earth Sciences 2001; 48(4): 489-499.
11. Abudhaise BA, Rabi AZ, Zwairy MAA, Hader AFE and Qaderi SE. Pulmonary manifestation in cement workers in Jordan. Int J Occup Med Environ Health 1997; 10: 417–428.
12. WHO. Prevention and control exchange: Hazard prevention and control in the work environment 219 Geneva, WHO; 1999.
13. Halwenge, Jennifer Atieno. Dust Pollution and Its Health Risks among Rock Quarry Workers in Kajiado County, Kenya. Reg. No. N50/Cty/Pt/24874/2011, A Thesis Submitted in Partial Fulfilment of the Requirements for The Degree of Master of Environmental Science in The School of Environmental Studies of Kenyatta University; 2011. (Accessed on 11-10-2019)
14. Pimentel JC, Menezes AP. Pulmonary and hepatic granulomatous disorders due to the inhalation of cement and mica dusts. Thorax 1978; 33: 219–227.
15. Sultan Ayoub Meo, Azeem MA, Ghori MG, Subhan MMF. Lung function and surface electromyography of inter costal muscles in cement mill workers. Int J Occup Med Environ Health 2002; 15(3): 269–77.
16. Oleru UG. Pulmonary function and symptoms of Nigerian workers exposed to cement dust. Environ Res 1984; 33: 379–385.
17. Johansson L, Albin M, Jakobsson K, Mikoczy Z. Histological type of lung carcinoma in asbestos Cement workers and matched controls. Br J Ind Med 1992; 49(9): 626–30.
18. Rafnsson V, Gunnarsdottir H, Kiilunen M. Risk of lung cancer among masons in Iceland. Occup Environ Med 1997; 54: 184–188.
19. Mc Dowall ME. A mortality study of cement workers. Br J Ind Med 1984; 41: 179–82.
20. Moller W, Hofer T, Ziesenis A, Karg E, Heyder J. Ultrafine particles cause cytoskeletal dysfunctions in macrophages. Toxcol Appl Pharmacol 2002; 182(3): 197–207.
21. Naarala J, Kasanen JP, Pasanen P, Pasanen AL, Liimatainen A, Pennanen S, *et al*. The effects of wood dusts on the redox status and cell death in mouse macrophages (RAW 264.7) and human leukocytes *in vitro*. J Toxicol Environ Health 2003; 66(13): 1221–1235.
22. Hurbankova M. One-year follow-up of the phagocytic activity of leukocytes after exposure of rats to asbestos and basalt fibres. Environ Health Perspect 1994; 102(5): 201–203.
23. Huang X, Jin Y, Guo X, Wang Y. Impact of dust storm PM2.5 and PM10 on the phagocytic function of alveolar macrophages of rat. Wei Sheng Yan Jiu 2004; 33(2): 154–157.
24. Mandal (Majee) A, Suva P. Haematological changes among construction workers exposed to cement dust in West Bengal, India. Prog. Health. Sci 2014; 4(1): 88-94.
25. Mojiminiyi FBO, Merenu IA, Ibrahim MTO, Nioku CH, and Ibrahim MTO. Regression Formulae for predicting Hematologic and liver functions from years of exposure to cement dust in cement Factory Workers in Sokoto, Nigeria. Afr. J. Biomed. Res 2007; 10: 235 - 240.
26. Shah SL. Toxicology of cement dust. J. of Appl. Toxic 2006; 26(3): 223 - 228.
27. Dier MS, Sirwan MM, Logman OHS. Some biochemical and haematological parameters among petroleum and cement factory workers in Sulaimaniyah city/ Kurdistan/Iraq. Chem and Mat Res 2014; 6(8): 29-33.
28. Kohen R, Nyska A. Oxidation of biological system: Oxidative stress phenomena, anticnåaclants, redox reactions and methods for their quantification. Toxicol. Pathol 2002; 30: 620-650.
29. Adly AAM. Oxidative stress and disease: An updated review. Res. J. Immunol 2010; 3: 129-145.
30. Jude ALC, Sasikala K, Kumar RA, Sudha S, Raichel J. Haematological and cytogenetic studies in workers occupationally exposed to cement (lust).J. Hum. Gen 2002; 2: 95-99.
31. Feng L, Zhou S, Gu L, GellDA, Mackay JP. Structure of oxidized a-haemoglobin bound to AHSP reveals a protective mechanism for haem. Nature 2005; 435: 697-701.
32. Gawad AE, Schlichting L, Strauss O, and Zeitz O. Anti apoptotic properties of erythropoietin: Novel strategies for protection of retinal pigment epithelial cells. Eye Lond 2009; 23: 2245-2250.
33. Conroy DM, Williams TJ. Eotaxin and the attraction of eosinophils to the asthlnatic lung. Respir Res 2001; 2: 150-156.

34. Kang JI, Sowers LC. Examination of hypochlorous acid-induced damage to cytosine residues in a CpG dinucleotide in DNA. Chem. Res. Toxicol 2008; 21: 1211-1218.
35. Ogunbileje JO, Akinosun ONI, Akinduti PA, Nwobi LA, E. iilude OA. Serum levels of some trace metals and leucocyte differential counts in Nigeria cement factory workers: Possible toxicity implications. Florida Int. J 2010b; 2: 55-58.
36. Tulinska, J, Jahnova E, Dusinska D, Kuricova M,Liskova A, *et al*. Immunomodulatory effects of mineral fibres in occupationally exposed workers. Mut. Res 2004; 553: 111-124.
37. Aminian O, Sharifian SA, Mehrdad R, Haghighi KS, Mazaheri M. Antinuclear antibody and rheumatoid factor in silica- exposed workers. Arh. Hig. Rada. Toksikol 2009; 60: 185-190.
38. National Institute of Occupational Safety and Health Hazard (NIOSH) Review. Health effects of occupational exposure to respirable crystalline silica. DHHS 2002; 129: 23.
39. Martin S and Griswold W. Human health effects of heavy metals. Environmental Science and Technology Briefs for Citizens: Cent. for Haz. Sub. Res (CHSR) 2009; 15: March 2009. Accessed on 11-12-2019.
40. Larson A. Silicosis and silica dust exposure. Expert Law Publication; 2004. www.expertlaw.com/library/ environmental_law/silicosis.html. Accessed on 10-12-2019.

Cite this article as:

Vijaya Bhaskara Reddy M. Hematological alterations among Quarry Workers exposed to Granite Dust in Thailand. Int. Res. J. Pharm. 2020;11(3):29-34 http://dx.doi.org/10.7897/2230-8407.110326

Source of support: Nil, Conflict of interest: None Declared