

***In vivo* Activity of Dust Inhalation at Ratcon Quarry, Sokuro Village, Oluyole, Ibadan Oyo State**

Adeniyi Abayomi Olusegun^{1*}

¹*Department of Technical Education, Osun State College of Education, Ila –Orangun, Nigeria.*

Author's contribution

This work was carried out by the author. Author AAO designed the study, performed the statistical analysis, wrote the protocol and wrote the first draft of the manuscript. Author AAO managed the analyses of the study, managed the literature searches, read and approved the final manuscript.

Article Information

DOI:10.9734/JALSI/2021/v24i130215

Editor(s):

(1) Dr. Palanisamy Arulselvan, Universiti Putra Malaysia, Malaysia.

Reviewers:

(1) Vitor Augusto Queiroz Mauad, ABC Medica School, Brazil.

(2) Karmegam Karupiah, Universiti Putra Malaysia, Malaysia.

Complete Peer review History: <http://www.sdiarticle4.com/review-history/64912>

Original Research Article

Received 25 November 2020

Accepted 30 January 2021

Published 17 February 2021

ABSTRACT

A total of twenty (20) experimental adult male rats, aged 60 days (7 to 8 weeks) with average body weight between 150-200 gm were grouped, restrained inside laboratory approved plastic holders and exposed to dust inhalation at the quarry site with exposure time of 7, 14 and 21 days to reflect short-term effects while 42days represent long-term effects of dust inhalation on human beings. Each specimen was collected and sacrificed at their grouped survival periods and subjected to laboratory analysis that include Hematology and Histopathology of the lungs. The Hematology results of the 7 and 14days specimens revealed no remarkable changes in the Erythrogram (PCV, HB and RBC), the Leucogram (WBC) and the Platelets but however, the results of the 21 and 42 days specimen revealed leukocytosis (increase in WBC), lymphocytosis (increase Lym) and neutrophilia (increase neutrophils) ($p < 0.05$). The Histopathology results of the first specimen (7 days exposure) showed no observable lesion, the second specimen (14 days) showed capillary congestion and mild interstitial pneumonia, while the third (21 days) and fourth (42 days) samples showed the rats graduating from mild to moderate interstitial pneumonia and oedema. The risk of these diseases depends on the amount of organic or inorganic dusts inhaled and deposited in the alveolar region, the air concentration of respirable dust as well as the exposure time and breathing pattern.

Keywords: *Specimen; hematology; histopathology; alveolar; pneumonia.*

*Corresponding author: E-mail: dnyolusegun@gmail.com;

1. INTRODUCTION

Air pollution caused by dust has been a major environmental and health problem in recent years. "Suspended particles" refers to solid or liquid particles in the air. These particles which are the primary air pollutants can have diverse sources and may come in variable sizes [1]. Dust storms are natural events that occur mainly in arid areas, reducing air quality and visibility. When air visibility declines to less than a kilometer, dust storms are created [2]. Due to lack of vegetation in the areas prone to dust, the air starts to warm and move up, when these currents reach winds with high transfer speed, the result may be winds circulating downward. These intense winds cause dust storms near the ground's surface. Most often, precipitation is below 50 mm per year in these areas [3]. However, some researchers have suggested 100 mm [4].

1.1 Sources of Dust

Dust particles, often referred to as particulate matter (PM), in the atmosphere arise from a wide variety of sources. Both the size and chemical composition vary widely in relation to the nature of the source and the history of the particles. Coarse particulates can be regarded as those with a diameter greater than 2.5 μm (e.g. PM_{10} – 10 μm), and fine particles less than 2.5 μm ($\text{PM}_{2.5}$). Under humid conditions many particles attract water vapour and grow to form small droplets. The term 'aerosol' is often used for both solid particles and droplets suspended in air [5]. Primary particles form from combustion, soil erosion and disintegration. Pollen and spores figure in this category. Secondary particles are generated by vehicular traffic, industrial activities and thermoelectrical implants. Atmospheric dust particles with a diameter of less than 10 μm and 2.5 μm draw special attention and are defined PM_{10} and $\text{PM}_{2.5}$ (PM= Particulate Matter), respectively. $\text{PM}_{2.5}$ particles are a subset of PM_{10} and count for 60% of its weight. PM_{10} is an inhalable particle as it can travel deep into the breathing apparatus to the larynx; and it's also breathable as it can settle in the pulmonary alveoli. Particles less than 10 μm may damage the lungs, cause dramatic intensification effects on atmospheric chemical reactions, increase the probability of precipitation, increase fogs and clouds, reduce visibility and solar radiation, cause track time changes in temperature and biological rhythms in plant growth, and profoundly change the materials in soil [6]. Most

epidemiological studies have considered suspended particles of 10 μm or lower in diameter as indicators of exposure, as this size includes coarse particles (diameter 2.5 to 10 μm) and fine particles (diameter less than 2.5 μm) [7,8]. These dust particles raise serious health concerns as they have been linked to a number of breathing and cardiovascular diseases. Sources of dust particles can be natural (volcanic eruption, sea aerosols, spores, pollen, soil erosion, or man-made (vehicular traffic, industrial emissions and combustion processes) [9].

1.2 The Mechanics of the Human Lungs

The lungs are protected by a series of defense mechanisms in different regions of the respiratory tract. When a person breathes in, particles suspended in the air enter the nose, but not all of them reach the lungs. The nose is an efficient filter. Most large particles are stopped in it, until they are removed mechanically by blowing the nose or sneezing. Some of the smaller particles succeed in passing through the nose to reach the windpipe and the dividing air tubes that lead to the lungs. These tubes are called bronchi and bronchioles. All of these airways are lined by cells. The mucus they produce catches most of the dust particles. Tiny hairs called cilia, covering the walls of the air tubes, move the mucus upward and out into the throat, where it is either coughed up and spat out, or swallowed. The air reaches the tiny air sacs (alveoli) in the inner part of the lungs with any dust particles that avoided the defenses in the nose and airways. The air sacs are very important because through them, the body receives oxygen and releases carbon dioxide. Dust that reaches the sacs and the lower part of the airways where there are no cilia is attacked by special cells called macrophages. These are extremely important for the defense of the lungs. They keep the air sacs clean. Macrophages virtually swallow the particles. Then the macrophages, in a way which is not well understood, reach the part of the airways that is covered by cilia. The wavelike motions of the cilia move the macrophages which contain dust to the throat, where they are spat out or swallowed. Besides macrophages, the lungs have another system for the removal of dust. The lungs can react to the presence of germ-bearing particles by producing certain proteins. These proteins attach to particles to neutralize them. The way the respiratory system responds to inhaled particles depends, to a great extent, on where the particle settles. For example, irritant dust that

settles in the nose may lead to rhinitis, an inflammation of the mucous membrane. If the particle attacks the larger air passages, inflammation of the trachea (tracheitis) or the bronchi (bronchitis) may be seen. The most significant reactions of the lung occur in the deepest parts of this organ. Particles that evade elimination in the nose or throat tend to settle in the sacs or close to the end of the airways. But if the amount of dust is large, the macrophage system may fail. Dust particles and dust-containing macrophages collect in the lung tissues, causing injury to the lungs. The amount of dust and the kinds of particles involved influence how serious the lung injury will be. For example, after the macrophages swallow silica particles, they die and give off toxic substances. These substances cause fibrous or scar tissue to form. This tissue is the body's normal way of repairing itself. However, in the case of crystalline silica so much fibrous tissue and scarring form that lung function can be impaired. The general name for this condition for fibrous tissue formation and scarring is fibrosis. The particles which cause fibrosis or scarring are called fibrogenic. When fibrosis is caused by crystalline silica, the condition is called silicosis [10].

1.3 Effects of Dust on the Lungs

The lungs are the organs of breathing and they are responsible for bringing oxygen from the atmosphere into the body through a series of branching air tubes Fig. 1 and exchanging it for

carbon dioxide that is released back into the atmosphere. The lungs are constantly exposed to danger from the dusts we breathe. Luckily, the lungs have another function - they have defense mechanisms that protects them by removing dust particles from the respiratory system. For example, during a lifetime, a coal miner may inhale 1,000 g of dust into his lungs. When doctors examine the lungs of a miner after death, they find no more than 40 g of dust. Such a relatively small residue illustrates the importance of the lungs' defenses, and certainly suggests that they are quite effective. On the other hand, even though the lungs can clear themselves, excessive inhalation of dust may result in disease [11].

1.4 Silicosis

Silicosis is a fibronodular lung disease caused by inhalation of dust containing crystalline silica (alpha-quartz or silicon dioxide), which is distributed widely. They further postulated that small ($\leq 1 \mu\text{m}$) particles are more dangerous because they are more likely to be deposited distally in the respiratory bronchioles, alveolar ducts, and alveoli. The surface of these particles generates silicon-based radicals that lead to the production of hydroxyl, hydrogen peroxide, and other oxygen radicals that damage cell membranes by lipid peroxidation and inactivate essential cell proteins. Alveolar macrophages ingest the particles, become activated, and release cytokines, including tumor necrosis factor, interleukin-1, and leukotriene B-4, as well

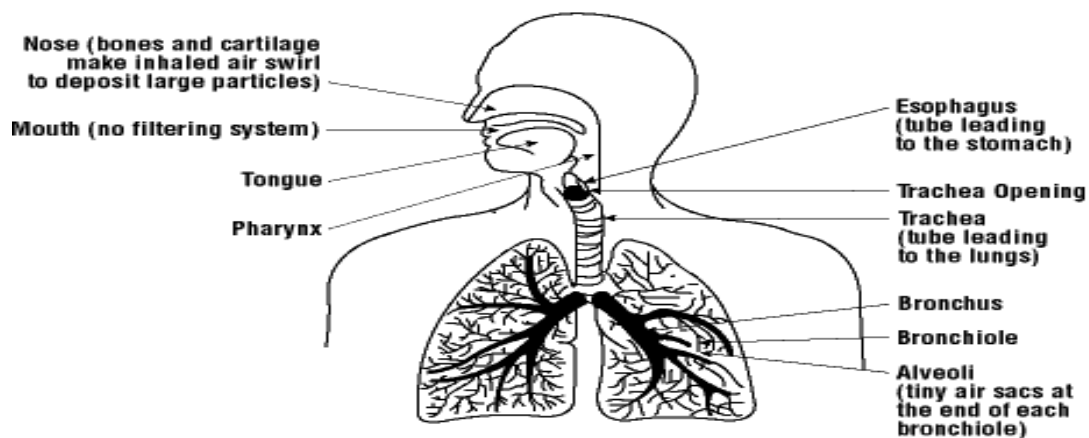


Fig. 1. Diagram showing air inhalation in a human being (culled from Canadian Centre for Occupational Health and Safety)

aschemotactic factors that recruit other inflammatory cells. The ensuing inflammation damages resident cells and the extracellular matrix. Transforming growth factor- α induces proliferation of type 2 pneumocytes, and other cytokines (e.g., platelet-derived growth factor, insulin - like growth factor) stimulate fibroblasts to proliferate and produce collagen; fibrosis results. Silica particles outlive the alveolar macrophages that ingested them, thereby continuing the cycle of injury and Cavitation caused by lung parenchymal necrosis in complicated silicosis may predispose individuals to *Aspergillus* colonization and to formation of an aspergilloma (mycetoma) [12].

This paper therefore intends to examine and establish the negative effects of dust inhalation on human health by using laboratory rats as the basis of investigation.

2. METHODOLOGY

2.1 Area of Study

Ratcon Quarry was chosen as the area of study due to thenature of the experiment and investigation that requires a location with concentration of both inorganic and organic dust. Ratcon Quarry is located at Sokuro Village, Oluyole L.G.A, along the right side of Lagos-Ibadan expressway, about 15km away from Ibadan City, Oyo State, Nigeria. Geographical location on the map is Longitude 7°28'14" N and 7°25'83" N and Latitude 3°84'36" E and 3°76'24" E.

2.2 Experimental Methods

Laboratory rats were used as the specimen material for investigation and experimental model since their respiratory systems and shapes are similar to the human respiratory system. A total of twenty (20) experimental adult male rats, aged 60 days (7 to 8 weeks) with average body weight between 150-200gm were grouped, restrained inside laboratory approved plastic holders and exposed to dust inhalation at the quarry site. These rats were randomly divided and placed into five (5) plastic holders, exposed to quarry dusts and withdrawn at the end of seven (7), fourteen (14), twenty-one (21) and forty-two (42) days. A separate plastic holder specimen of four (4) rats was set aside for control at the Laboratory. The duration of the exposure time for short-term effects was 7, 14 and 21 days while 42days was adopted as the exposure time for

long-term effects of dust inhalation on human beings.

At the end of each exposure time, each specimen was collected and sacrificed at their grouped survival periods and subjected to laboratory analysis that include Hematology and Histopathology of the lungs.

Hematology is the science or study of blood, blood-forming organs and blood diseases. Hematology is a branch of internal medicine that deals with the physiology, pathology, etiology, diagnosis, treatment, prognosis and prevention of blood-related disorders by focusing largely on lymphatic organs and bone marrow and may diagnose blood count irregularities or platelet irregularities and treating organs that are fed by blood cells, including the lymph nodes, spleen, thymus and lymphoid tissue [13]. Histopathology is the diagnosis and study of diseases of the tissues, and involves examining tissues and/or cells under a microscope [14].

3. RESULTS AND DISCUSSION

3.1 Hematology Test Results

The laboratory results of the first and second specimens (7 and 14days), revealed no remarkable changes in the Erythrogram (PCV, HB and RBC), the Leucogram (WBC) and the Platelets. This is because the lungs are protected by a series of defense mechanisms in different regions of the respiratory tract and these defence mechanism are still working to prevent any injury, infection or disease to the lungs.

However, the laboratory results of the third and fourth specimen (21 and 42 days) revealedleukocytosis (increase in WBC), with lymphocytosis (increased Lym), neutrophilia (increased neutrophils) ($p < 0.05$). The leukocytosis is due to stress or inflammation as a result of tissue damage from the inhalation of the dust. The observation of leukocytosis at 3rd and 4th sampling post exposure suggests that the animals have been stressed from discomfort in the environment and exposure to toxic dust substances which make the animal susceptible to opportunistic infection. The lymphocytosis is a result of the specimen trying to fight infections and the neutrophilia is an evidence of a bacterial infection or acute inflammation of the lungs. In comparison, the control specimen test results showed No Change in the Erythrogram (PCV,

HB and RBC), the Leucogram (WBC) and the Platelets.

3.2 Histopathology Test Results

Histopathology provided a good quality sectioning of the tissues from the lungs of the experimental rats after it was stained with Hematoxylin and Eosin (H&E) and then viewed under a light microscopic evaluation. The second rats specimen (14 days) showed capillary congestion and mild interstitial pneumonia, while the third (21 days) and fourth (42 days) samples showed the rats graduating from mild to moderate interstitial pneumonia and oedema. The twenty-one (21) days and forty-two (42) days

rat specimens started showing traces of mild to moderate pneumonia, due to bacteria, viruses, or fungi infections as a result of the exposure to dusts. Pneumonia causes inflammation in the air sacs in the bronchioles sacs, which are called alveoli. When the alveoli are filled (oedema) with fluid or pus, breathing becomes difficult. In comparison, the control specimens revealed Normal lungs (No observable lesion) for the 7, 14, 21 and 42 days.

3.3 Histopathology Photomicrographs

The histopathological microphotographs have been depicted as follows.

CONTROL SAMPLES - 42 DAYS EXPOSURE

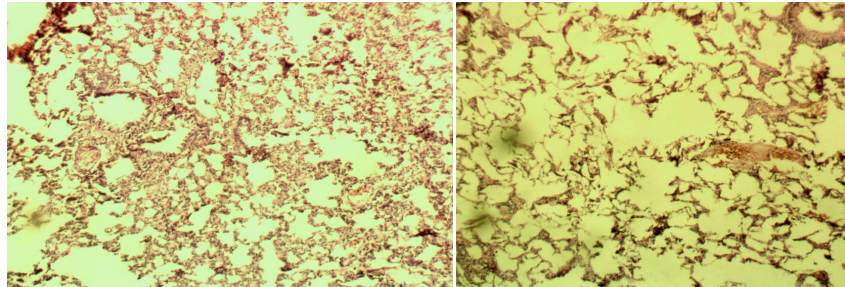


Fig. 2. Normal lungs- no observable lesion

FIRST SAMPLE -7 DAYS EXPOSURE

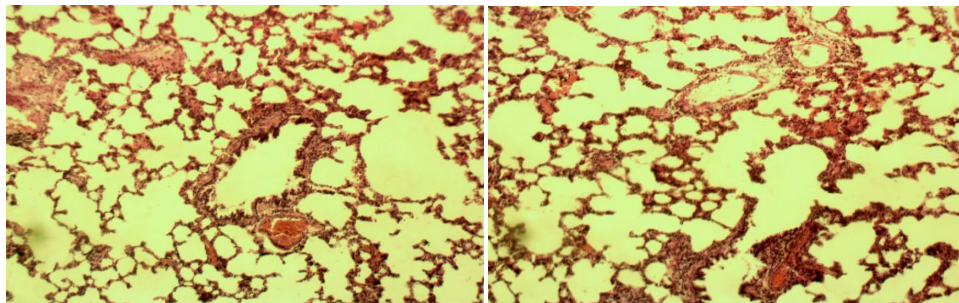


Fig. 3.No observable lesion

SECOND SAMPLE -14 DAYS EXPOSURE

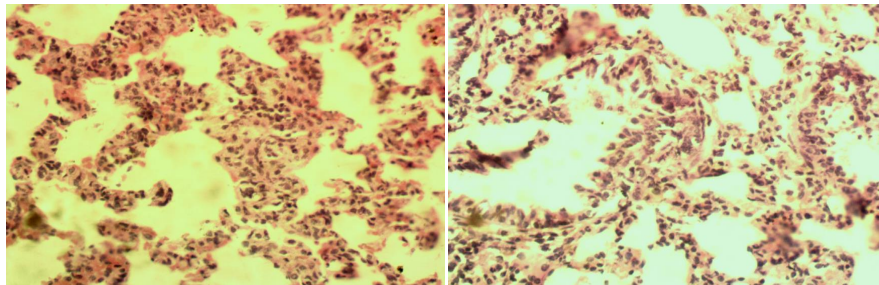


Fig. 4. Capillary congestion
THIRD SAMPLE -21 DAYS EXPOSURE

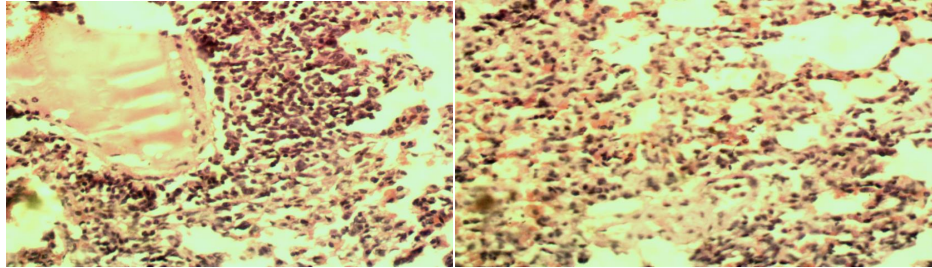


Fig. 5. Moderate interstitial pneumonia and oedema

FOURTH SAMPLE - 42 DAYS EXPOSURE

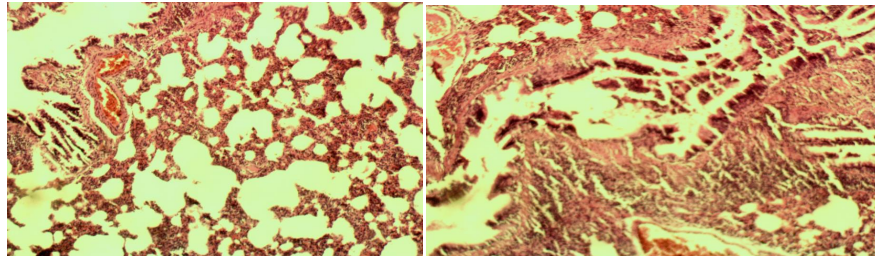


Fig. 6. Moderate interstitial pneumonia

4. MILD INTERSTITIAL PNEUMONIA

Mild interstitial pneumonias are a heterogeneous group of diffuse parenchymal lung diseases characterized by specific clinical, radiologic and pathologic features.

4.1 Discussion

The results from the hematology and histopathology of the lungs revealed there was inflammation of the lungs and lymphocytic activities i.e increase in lymphocytic cells. This is related to Slaoui et al.,[15] findings in which they reported cases of inflammatory lesions in the lungs of Wistar rats. The inflammation of the lungs and lymphocytic activities can be linked to increase in the white blood cells (cells responsible for fighting against external bodies) due to infection of the lungs by gradual deposition of the dust in the alveoli of their lungs. This infection showed pockets of fluids in the lungs and inflammation of the tissues, hence pneumonia was diagnosed which graduated from mild case to moderate case and which if not properly treated will move to severe case of the infection. A long term exposure to this condition may lead to chronic respiratory diseases. Continuous exposure to these conditions will lead to silicosis, pneumoconiosis and fibrotic lung diseases caused by overexposure to dusts.

5. CONCLUSION AND RECOMMENDATIONS

It has been established that human lungs will be infected if exposed to dust inhalation. Short term infections range from mild to severe pneumonia but long term infection will result in diseases like silicosis and other chronic respiratory diseases which may be irreversible, progressive, incurable, at later stages disabling and eventually fatal.

5.1 Recommendations

The following recommendations are hereby made in order to reduce the risk involved in the inhalation of dusts:

- use of wet processes for Industrial exhaust streams
- enclosure of dust-producing processes under negative air pressure (slight vacuum compared to the air pressure outside the enclosure)
- exhausting air containing dust through a collection system before emission to the atmosphere
- use of vacuums instead of brooms
- good housekeeping
- efficient storage and transport
- controlled disposal of dangerous waste

- Use of personal protective equipment may be vital, but it should nevertheless be the last resort of protection. Personal protective equipment should not be a substitute for proper dust control and should be used only where dust control methods are not yet effective or are inadequate.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Hinds WC. Aerosol technology: Properties, behavior, and measurement of airborne particles. New York, Wiley-Interscience. 1999;78-89.
2. Wang S, Yuan W, Shang K. The impacts of different kinds of dust events on PM10 pollution in northern China. Atmospheric Environment. 2006;40(40):7975-82.
3. Xuan J, Sokolik IN, Hao J, Guo F, Mao H, Yang G. Identification and characterization of sources of atmospheric mineral dust in East Asia. Atmospheric Environment. 2004;38(36):6239-52.
4. Goudie AS, Middleton NJ. Saharan dust storms: Nature and consequences. Earth Science Reviews. 2001;56(1):179-204.
5. APIS; 2016. Available: http://www.apis.ac.uk/overview/pollutants/overview_particles.htm
6. Eniscuola; 2013. Available: <http://www.eniscuola.net/en/argomento/air-pollution/pollutants-and-their-effects/atmospheric-dust/>
7. Bennion P, Hubbard R, O'Hara S, Wiggs G, Wegerdt J, Lewis S, et al. The impact of airborne dust on respiratory health in children living in the Aral Sea region. International Journal of Epidemiology. 2007;36(5):1103-10.
8. Gerivani H, Lashkaripour GR, Ghafoori M. The source of dust storm in Iran: A case study based on geological information and rainfall data. Carpathian Journal of Earth and Environmental Sciences. 2011;6(1):297-308.
9. World Health Organization. Occupational and environmental health team. WHO Air quality guidelines for particulate matter, nitrogen dioxide and sulfur dioxide: Summary of 2005 global update risk assessment; World Health Organization; 2006. Available: <http://www.who.int/iris/handle/10665/69477>
10. Kalair E, Abas N, Khan N. Kalair energy networks; 2019. Available: https://www.ccohs.ca/oshanswers/chemicals/lungs_dust.html
11. Wark K, Warner CF. Air pollution: Its origin and control. Harper and Row Publishers, New York, NY; 1981.
12. Basil and Zab Silicosis, Pathophysiology and Epidemiology. Medscape Journal; 2015. Available: <https://emedicine.medscape.com/article/302027-overview>
13. Healio; 2017. Available: <https://www.healio.com/hematology-oncology/news/online/%7B2dd178d0-7f92-46a8-add9-2c7d634d2cea%7D/what-is-hematology>
14. RCPATH; 2019. Available: <https://www.rcpath.org/discover-pathology/news/factsheets/histopathology.html>
15. Slaoui Mohamed, Dreef H, van Esch, Eric. Inflammatory Lesions in the Lungs of Wistar Rats. Toxicologic pathology. 1998;26:712-3; discussion 714.

© 2021 Olusegun; This is an Open Access article distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Peer-review history:

The peer review history for this paper can be accessed here:
<http://www.sdiarticle4.com/review-history/64912>